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Research Article

Performance of contrast-associated acute kidney injury predictive risk models in Thai cardiac angiography or angioplasty patients

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ABSTRACT

Several risk prediction models of Contrast-associated acute kidney injury (CA-AKI) in patients undergoing cardiac angiography or angioplasty are available. However, the lack of extensive external validations limits generalizability and clinical acceptance. This study conducted the external validation of three CA-AKI predictive risk models (Chen's, Inohara's, and Tziakas' risk models) and determined the incidence of CA-AKI in Thai patients undergoing cardiac angiography or angioplasty. A total of 647 medical records of patients who underwent elective cardiac angiography (n=446) and angioplasty (n=201) were reviewed. Fifty-five percent were male, mean age 62.6±10.2 years, and mean estimated glomerular filtration rate (eGFR) 69.93±24.30 ml/min/1.73 m2). Incidents of CA-AKI, defined as an absolute increase of serum creatinine of at least 0.3 mg/dL within 48 hours or a relative increase of at least 50% within seven days after the procedure, were collected. The results showed that 78 patients (12.1%) had developed CA-AKI. Chen's, Inohara's, and Tziakas' predictive risk models exhibited low discriminative ability with c-statistic of 0.571, 0.551, and 0.530, respectively. Due to low discriminative capability, these risk models may have low sensitivity to predict CA-AKI in Thai patients undergoing elective cardiac angiography or angioplasty.

Keywords:
Contrast media, Contrast-associated acute kidney injury, Nephropathy, Angiography, Angioplasty, Percutaneous coronary intervention

1. INTRODUCTION

Contrast-associated acute kidney injury (CA-AKI) associated with short- and long-term adverse outcomes in patients undergoing cardiac angiography or angioplasty is common[1-2]. Identifying high-risk patients is of utmost criticality because no specific treatment is available for CA-AKI[3]. Preventive measures: reciving adequate hydration and minimizing the volume of radiocontrast in the high-risk CA-AKI patients have become crucial[3-6]. Therefore, several CA-AKI risk prediction models to identify patients at high risk for developing CA-AKI after angiography or angioplasty have been developed. However, the lack of extensive external validations limits generalizability and clinical acceptance[7-9]. Since 2012 the Kidney Disease Improving Global Outcomes (KDIGO) has updated the definition for CA-AKI. CA-AKI defines as an increase in serum creatinine at least 0.3 mg/dL within 48 hours or 1.5 times baseline within seven days, or urine output less than 0.5 mL/kg/hour for at least 6 hours after receiving intravascular radiocontrast[1]. As a result, validating these previous predicting risk models with the updated standard characterization of CA-AKI is essential.

The risk prediction models of CA-AKI depend on either pre-procedural or procedural characteristics. Procedural risk models include variables such as the volume of contrast media and the use of intra-aortic balloon pumps, which are unknown until the completion of the procedure. On the contrary, pre-procedural risk models require only factors available before performing angiography or angioplasty[10-11]. Therefore, they facilitate a clinical routine to target high-risk patients of CA-AKI

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before initiating the procedures[4,10,12]. Only Mehran's model has been validated in Thai cardiac angiography or angioplasty patients and shown a good discrimination [13]. However, this procedural risk model is only marginally beneficial in detecting high-risk individuals before the procedure. Moreover, three validated pre-procedural risk prediction models, Chen (2014), Iohara (2015), and Tziakas (2013) have been reported to have good discrimination[10,12,14]. The objectives of this study were to validate the CA-AKI pre-procedural predictive risk model's discrimination and determine the occurrence of CA-AKI with KDIGO's CA-AKI definition in Thai patients undergoing cardiac angiography or angioplasty.

2. PATIENTS AND METHODS

This retrospective study collected the data from patients aged ≥18 years who underwent elective cardiac angiography or angioplasty at Maharaj Nakorn Chiang Mai hospital between January 1, 2011, and December 31, 2016. We excluded medical records whose serum creatinine values were within 30 days before and seven days after the procedure was unavailable, or who had an unstable renal function, end-stage renal disease, or planned for renal replacement therapy after the procedures. The patients who underwent coronary artery bypass graft (CABG) or received radiocontrast within 14 days were excluded. Demographic data, including age, sex, body weight, height, underlying diseases, and medication use, were collected. Procedural data contained procedure type, fluid administration, radiocontrast dose, and laboratory data: SCr, hematocrit, and low-density lipoprotein (LDL), were collected. The analysis included only datafrom the first cardiac angiography or angioplasty.

The primary outcomes were the ability to discriminate between the three risk prediction models for CA-AKI established by Chen, Inohara, and Tziakas[10,12,14-15]. The secondary outcome was the occurrence of CA-AKI (defined as an increase in SCr ≥0.3 mg/dL within two days or an increase in SCr ≥50% within seven days after radiocontrast administration)[1,16].

In brief, Chen's risk model contained nine variables: age, history of myocardial infarction, diabetes mellitus (DM), hypotension, left ventricular ejection fraction, anemia, estimated glomerular filtration rate (eGFR), high-density lipoprotein and, urgent angioplasty[10]. Iohara's model included seven variables: age, hypertension, DM, previous angioplasty, heart failure with New York Heart Association class III/IV, angioplasty for the acute coronary syndrome, and SCr[12]. Tziakas's model contained four variables: preexisting renal disease, metformin, previous angioplasty, and peripheral arterial disease[14].

We used $N = [Z2_{\alpha/2}\ V(AUC)]/d2$ whereas $\alpha=0.05$, d=±10%, AUC=80% and estimated CA-AKI occur at 5% and determine sample size with N(1+R) which required at least 600 patients in our study[17-18]. Descriptive data were reported as numbers and percentages. Quantitative data were present as mean±standard deviation (SD) or median and interquartile range (Q1-Q3) as appropriate. To evaluate the ability of the model discrimination, the area under the receiver operator characteristic curve (ROC) generally equal to C-statistics with a 95% confidence interval for each model was calculated by IBM SPSS version 17.0[19].

Table 1. Patient and procedural characteristics.

Variables	Patients (N=647)
Demographic data	
Age (years)	62.6 ± 10.2
Age ≥ 70 years	166 (25.7)
Male	369 (57.0)
Height (cm)[1]	159 ± 8
Body weight (kg)[1]	56.6 ± 12.2
Body Mass Index (kg/m^2)[1, 2]	22.4 ± 4.0
Smoking status[1]	
Current smoking	27 (4.7)
Quit	251 (43.9)
Never	294 (51.4)
NYHA class III/IV	33 (5.1%)
Serum creatinine (mg/dL)	1.04 (0.88 - 1.40)

Data are present as mean ± standard deviation or median (Q_1-Q_3) as appropriate.
1 Number of missing data: height (110), body weight (1), smoking status (76), hematocrit (2), LDL (419), HDL (420), previous cardiac angioplasty (4), left ventricular ejection fraction (85), radiocontrast dose (2), intravenous fluid (263), urine output (282) and current medication (4).
2 BMI is calculated as body weight (kg)/height (m)^2.
3 Some patients had more than one disease.
4 Low osmolar radiocontrast agents were used in all patients.
ACEIs; Angiotensin converting enzyme inhibitors, ARBs; Angiotensin receptor blockers, BMI; Body Mass Index, CrCl; creatinine clearance (the Cockcroft and Gault formula), eGFR; estimated glomerular filtration rate (the abbreviated MDRD equation), HDL; high density lipoprotein, LDL; low density lipoprotein, LVEF; Left ventricular ejection fraction, NYHA; New York Heart Association functional classification.

Table 1. Patient and procedural characteristics.

Variables	Patients (N=647)
Demographic data	
eGFR (mL/min/1.73 m^2)[3]	63.93 ± 24.30
eGFR < 60 mL/min/1.73 m^2	287 (45.6)
CrCl (mL/min)[1, 4]	54.64 ± 23.64
Hematocrit[1]	37.50 ± 5.3
LDL (mg/dL)[1]	109.52 ± 37.89
HDL (mg/dL)[1]	45.04 ± 12.99
Previous cardiac angioplasty[1]	74 (11.4)
LVEF (%)[1]	53.40 ± 16.40
Past Medical History[3]	
Hypertension	346 (53.5)
Diabetes mellitus	170 (26.3)
Dyslipidemia	225 (34.8)
Ischemic heart disease	361 (55.8)
Chronic heart failure	119 (18.4)
Valvular heart disease	251 (38.8)
Atrial fibrillation	149 (23.0)
Chronic kidney disease	93 (14.2)
Current Medications[1]	
Aspirin	389 (60.5)
Clopidogrel	266 (41.4)
Warfarin	86 (28.9)
ACEIs/ARBs	353 (54.9)
Beta-blockers	401 (62.4)
Calcium channel blockers	117 (18.2)
Oral Nitrates	185 (28.8)
Digoxin	102 (15.9)
Diuretics	349 (54.3)
Furosemide	255 (39.4)
Statins	366 (56.6)
Proton-pump inhibitors	153 (23.8)
Metformin	69 (10.7)
Sulfonylureas	85 (13.1)
Insulin	36 (5.6)
Procedure data	
Cardiac angiography	446 (68.9)
Cardiac angioplasty	201 (31.1)
Radiocontrast[1,4] (mL)	30 (20 - 80)
minimum	10
maximum	460
Intravenous fluid (mL/day)[1]	900 (646 - 1,400)
Urine output (mL/day)[1]	1,000 (602 - 1,530)

Data are present as mean ± standard deviation or median (Q_1-Q_3) as appropriate.

5 Number of missing data: height (110), body weight (1), smoking status (76), hematocrit (2), LDL (419), HDL (420), previous cardiac angioplasty (4), left ventricular ejection fraction (85), radiocontrast dose (2), intravenous fluid (263), urine output (282) and current medication (4).

6 BMI is calculated as body weight (kg)/height $(m)^2$.

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Table 2. Number of serum creatinine monitoring and contrast-associated acute kidney injury.

Day	SCr monitoring (%) (N=647)	Number of CA-AKI*,**
1	403 (62.3)	35
2	241 (37.2)	19
3	149 (23.0)	2
4	114 (17.6)	4
5	95 (14.7)	4
6	102 (15.8)	5
7	106 (16.4)	9

*CA-AKI was defined as an increase in SCr ≥0.3 mg/dL within 2 days or an increase in SCr ≥50% within 7 days after radiocontrast administration., **only new CA-AKI were counted.
SCr; serum creatinine, CA-AKI; contrast-associated acute kidney injury.

3. RESULTS

3.1. Baseline patient and procedural characteristics

There were 647 patients with a mean age of 62.6± 10.2 years, and 55% were male (Table 1.). The mean eGFR was 69.93±24.30 mL/min/1.73 m^2. There were 287 patients (45.6%) with an eGFR less than 60 mL/min/1.73 m^2. The three most common underlying diseases were ischemic heart disease (55.8%), hypertension (53.5%), and valvular heart disease (38.8%), respectively. Forty hundred and forty-six patients (68.9%) underwent cardiac angiography. A low osmolar radiocontrast agent and 0.9% saline solution were administed to all patients before or after the procedure with a variable rate and amount.

3.2. Serum creatinine monitoring

Most patients (76.5%) had SCr monitoring within two days after receiving radiocontrast (Table 2.). About 78% of patients had SCr followed up 1-2 times, and less than 5% had SCr monitored more than four times within seven days after the procedure (Supplementary Table 1A).

3.3. Occurrence of CA-AKI

CA-AKI occurred in 78 patients (12.1%), and more than 50% of these patients had detected CA-AKI within the first two days. The longer-term monitoring of SCr resulted in additional CA-AKI detected shown in Table 2.

3.4. Performance of pre-procedural variable models to predict contrast-associated acute kidney injury.

Of those 647 patients, 643 records were used to test Inohara's risk model[12]. 638 records were used to test Tziakas' risk model[14]. Only 193 records were used to test Chen's risk model[10] due to the unavailability of HDL laboratory reports. The c-statistic of Chen's risk model was highest at 0.571, followed by Inohara's risk model (c-statistic=0.551) and Tziakas' risk model (c-statistic= 0.530) (Table 3. and Figure 1.). The result of subgroup of patients who underwent cardiac angiography or angioplasty were comparable.

4. DISCUSSION

In this study, three preprocedural CA-AKI risk models examination resulted in the range of c-statistic 0.530-0.57 with a comparable value among a subgroup of patients who underwent cardiac angiography and angioplasty. The results indicated that the discriminative ability of the three risk models was low and not clinically useful for elective cardiac angiography or angioplasty in Thai patients[20-21]. The performance of the tested models usually decreases when tested in different cohorts[22]. The explanation might be the differences in patient characteristics as unreported of valvular heart disease in Chen's and Iohara's studies and only 7% reported in Tziakas's study[10,12,14]. In addition, in the present study, 68.9% of patients underwent elective cardiac angiography, but all patients in the previous development risk models underwent angioplasty[7,19,21,23] which may affect the performance of the tested models. The volume of fluid hydration and diuretic use might contribute to the risk of CA-AKI but have not been included in the risk models[7]. Ma B et al. examined the risk score of Tziakas et al. in Canadians who underwent cardiac angioplasty and showed that this risk model also had low discrimination (Supplementary Table 2A.)[22].

Three CA-AKI prediction models selected in this study have been based on the following criteria. The preprocedural risk models can apply to patients who plan to undergo elective cardiac angiography or angioplasty. We excluded the procedural risk models because some variables can be obtained only after the procedure. Therefore, they have little benefit in the surveillance and prevention of CA-AKI. The prediction models of CA-AKI in patients with ST-segment elevation myocardial infarction (STEMI) who required primary angioplasty were excluded due to limited time for CA-AKI risk evaluation and prevention. Secondly, validated predictive risk models should yield at least 0.7 or an acceptable value of the c-statistic of discrimination of the model[24]. The variables in the model should be ready to use for evaluation with no additional information required. Lastly, it should not be time-consuming with variables not greater than 10 in the model.

A systematic review of risk prediction models with

Table 3. Performance of three pre-procedural contrast-associated acute kidney injury models.

Risk score	Number of patients	C-statistic	95% CI
Chen[8]	All 193	0.571	0.436-0.706
	angiography 126	0.557	0.407-0.707
	angioplasty 67	0.706	0.404-1.000
Inohara[10]	All 643	0.551	0.484-0.619
	angiography 442	0.554	0.475-0.633
	angioplasty 201	0.558	0.438-0.678
Tziakas[11]	All 638	0.530	0.460-0.601
	angiography 437	0.521	0.440-0.602
	angioplasty 201	0.584	0.439-0.729

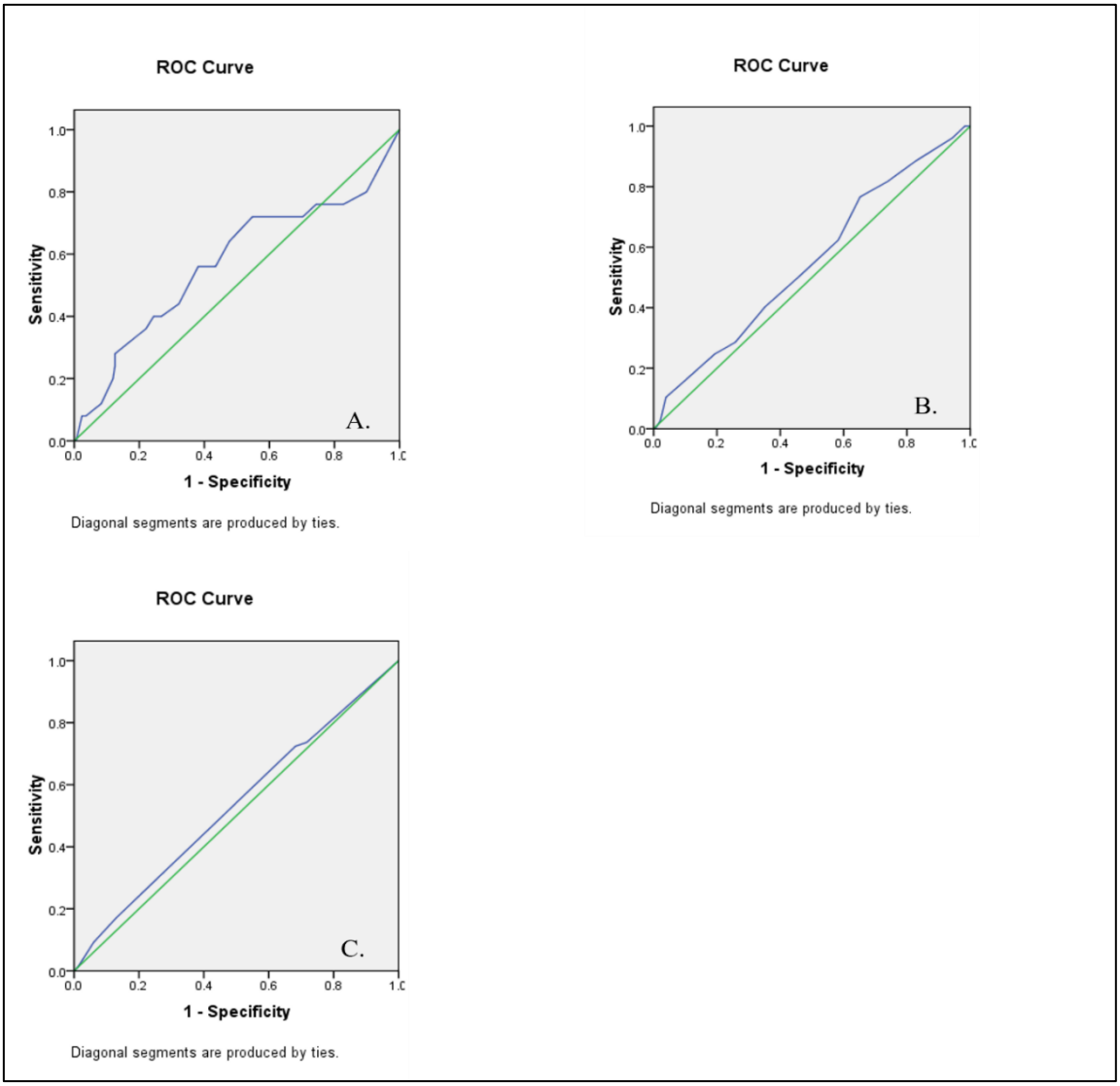

Figure 1. A. Chen's risk score (AUC 0.571; 95% CI 0.436-0.706); B. Inohara's risk score (AUC 0.551; 95% CI 0.484-0.619); C. Tziakas' risk score (AUC 0.530; 95% CI 0.460-0.601).

internal validation for contrast-associated nephropathy demonstrated high discrimination[7]. Chen YL et al. performed the split-sample internal validation of the two groups in which the baseline clinical and procedural characteristics had no significant differences. Chen's prediction model yielded the same c-statistic of 0.82 in training and validation datasets[10]. However, the external validation of Chen's prediction model demonstrated low discriminative power with c-statistic 0.555, 0.478, and 0.49 by Liu Y-H et al., Serif L et al., and Yin W et al., respectively (Supplementary Table 2A.)[9,25-26]. Our study observed low discriminatory ability with c-statistic 0.571 in Chen's model. Although performing internal validation, Inohara's prediction model had not reported the c-statistic and patient characteristics in the validation dataset.[12] as well as Chen's prediction model, external

validation by Yin W et al. demonstrated low discrimination with c-statistic 0.52 (Supplementary Table 2A.)[25]. Tziakas' prediction model also was validated externally in another three settings. The first external validation demonstrated excellent discriminating power with c-statistic 0.864. The validation cohort was also from the setting of the derivation study cohort and had similar baseline characteristics[14]. The second external validation performed in a multicenter across four countries had moderate discrimination with a c-statistic of 0.741[15]. The third external validation exhibited a low capability of discriminating power with c-statistic 0.502, even though the study was performed in the same setting as in the first external validation at different times (Supplementary Table 2A.)[9]. Our finding also demonstrated low discriminatory ability with c-statistic 0.530 in Tziakas' model.

Although there are many CA-AKI definitions[27], we used KDIGO's CA-AKI, a broadly accepted definition, to standardize and compare across studies[4,28]. These three risk models showed less discrimination capability. Perhaps, due to the difference in CA-AKI definition from the original studies[9,22,25]. Because c-statistics is a function of sensitivity and specificity of risk score, differences in the definition of CA-AKI can affect its value[7].

The rate of CA-AKI in this study was 12.1%. The incidence of CA-AKI in patients who underwent cardiac angiography or angioplasty range from 1.7% to 23%[22, 29-35]. Reports of incidence of CA-AKI have varied, depending on the population, the baseline risk factors, and the duration of SCr monitoring[1].

This first study reported the incidence of CA-AKI, regarding KDIGO's definition, the most widely accepted guideline, in Thai elective cardiac angiography or angioplasty patients. This study had some limitations inherent to the retrospective study. First, the SCr monitoring in each patient during the high-risk period was not uniformly measured. The follow-up SCr in patients with a high risk of developing CA-AKI justified by the physician's perspective was more often than usual. On the contrary, some patients who had not been monitored SCr or had less monitoring as required might have delayed diagnosis or failure to diagnose CA-AKI. CI-AKI usually occurs within 2-3 days after exposure to radiocontrast. Creatinine levels typically rise within 24 hours and peak three to five days afterward[27-36]. Our study found that CA-AKI still occurred from day 3 to 7 after contrast media administration. Therefore, SCr should be monitored closely within seven days after the procedure. Second, mainly because HDL data was not readily available, there were only 193 (29.8%) patients to examine Chen's risk score. Several reasons explained why many patients did not have lipid panel measurements. Patients referred to the hospital for angiography or angioplasty purposes were tremendous. Second, about 40% of patients received coronary angiography before valvular surgery. These patients were young adults with low atherosclerotic risks. Apart from ischemic heart disease (IHD), warfarin use, and statin use, the main clinical and procedural characteristics of these 193 patients were similar to overall patients (data not shown). Ischemic heart disease and statin usage were common in the subgroup (65.3% and 65.3%) compared to all patients (55.8% and 56.6%). Because a history of myocardial infarction (MI) was a predictor of Chen's risk score, the number of patients with such a history influenced the score that might alter the performance of the risk score. However, there was no significant difference in the history of MI among the overall patients (30.4%) and the subgroup patients (34.7%). In the subgroup, 106 patients (84.8%) with IHD had chronic statin use that was not significantly different from overall patients, 290 individuals (80.4%). While high-dose statin for secondary prevention in statin-naïve patients was advocated as one prevention measure for CA-AKI[37], the rates of CA-AKI in patients with IHD and statin-naive were 0 and 1.7 percent (p=0.33) in the subgroup of 193 patients and total participants. Therefore, IHD and statin usage might not affect the performance of the risk score. The difference in warfarin used between the subgroup and overall patients might not affect the c-statistics of the risk score since it has not been considered a factor associated with CA-AKI[1,5,38]. Consider the c-statistics of Chen's risk score's 95 percent confidence interval. This risk score had a low probability of being clinically useful (c-statistic >0.75) in Thai patients[21]. An adequate number of patients in further studies are needed to confirm this assumption.

Lastly, this was a single-center study, so generalizing our findings to the different populations should be done with caution. Compared to CA-AKI studies in Thai elective cardiac angiography or angioplasty patients, all the studies were small (n=61-305) and done in medical schools from 2008 to 2016[13,39-44]. These studies usually selected high-risk groups as those with diabetes mellitus [44] or chronic kidney disease[41-43], and aimed to investigate the effectiveness of preventive measures of CA-AKI[39, 41,43]. One prospective study validated Mehran's risk score for CA-AKI after cardiac angiography or angioplasty[13], the most well-known procedural risk model[45]. The analysis illustrated the high discrimination with a c-statistic of 0.86. The traditional definition of CA-AKI, SCr increases at least 25% or 0.5 mg/dL within 48 hours, were used. Majortity of the patients were elderly (the mean age was 67 years), and two-thirds were over 75 years old. Additionally, the incidence of CA-AKI was only 6.5%, which was less than expected due to few samples. It may be appropriate to use the predictive risk of CA-AKI in Thai patients after the procedure if Mehran's predictive risk model has highly discriminating capability in the large sample size and the standardized CA-AKI definitions used. Overall, patients in these studies were older than our patients, and there were male more than females [13,39-44]. Baseline renal function as eGFR or CrCl of the

patients were lower to our patients in three studies that included only patients with CKD[41-43], not differ from our patients in two studies[13,39], and better than our patients in one study[44]. Most studies included more patients with hypertension, diabetes mellitus, and dyslipidemia[13,39-44]. Low osmolar contrast agents were mainly used in all studies[13,39-44], recommended and preferred by the Society for Cardiovascular Angiography and Interventions (SCAI)[6,46]. At this time, the professionals should assess kidney function to identify those at high risk of CA-AKI before having cardiac angiography or angioplasty[1,6,37,47].

5. CONCLUSION

In the present study, CA-AKI occurred in 12.1% of patients. The pre-procedural risk models by Chen, Inohara, and Tziakas illustrated low discrimination in predicting CA-AKI. Therefore, these predictive risk models of CA-AKI may have limited application in Thai elective cardiac angiography or angioplasty. Before coronary angiography or angioplasty, the health care professionals should confirm the kidney function of the patients at high risk of CA-AKI. This research is just a validation study in Thai cardiac angiography or angioplasty of these three risk models. Another validation should be done in many different groups in a large and sufficient sample of Thai cardiac angiography or angioplasty to provide an average c-statistic for Thai patients. A prospective study is warranted, allowing for collection parameters that affect the CA-AKI, such as hydration fluid volume and the management of a consistent SCr measurement.

6. ACKNOWLEDGEMENT

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Funding
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Conflict of interest
None to declare.

Ethics approval
The research ethics committee, Faculty of Medicine, Chiang Mai University, approved the study protocol (study code: NONE-2559-04380). The study conforms to the principles outlined in the Declaration of Helsinki.

Implication for health policy makers/practice/research/ medical education
Chen's, Inohara's and, Tziakas' predictive risk models have moderate discriminative ability in Thai elective cardiac angiography or angioplasty. Development and validation of a good clinical applicable pre-procedural CA-AKI predictive risk model is still needed.

Author contribution
SS, PS conceived and designed the analysis. SS Collected the data. SS, KL and PS contributed data and analysis tools. SS performing the analysis. SS, KL and PS wrote the paper.

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