

Nomogram for the Prediction of Contrast-associated Acute Kidney Injury in Patients with Hypoalbuminemia Undergoing Coronary Angiography

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Research article

Keywords: Contrast-associated kidney injury, Hypoalbuminemia, Nomogram, Coronary angiography, Percutaneous coronary intervention

Posted Date: June 11th, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-34291/v1>

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Version of Record: A version of this preprint was published on August 31st, 2020. See the published version at <https://doi.org/10.1186/s12872-020-01689-6>.

Abstract

Background: Risk stratification is recommended as the key step to prevent contrast-associated acute kidney injury (CA-AKI) by allowing for prevention among at-risk patients undergoing coronary angiography (CAG) or percutaneous coronary intervention (PCI). Patients with hypoalbuminemia are prone to CA-AKI and do not have their own risk stratification tool. Therefore, we developed and validated a nomogram for predicting CA-AKI in patients with hypoalbuminemia undergoing CAG/PCI.

Methods: A total of 1272 consecutive patients with hypoalbuminemia undergoing CAG/PCI were enrolled and randomly assigned (2:1 ratio) to a development cohort (n = 848) and a validation cohort (n = 424). CA-AKI was defined as a serum creatinine (SCr) increase of ≥ 0.3 mg/dL or 50% from baseline within the first 48 to 72 hours following CAG/PCI. A nomogram was established with independent predictors according to multivariate logistic regression and a stepwise approach. The discrimination of the nomogram was assessed by the area under the receiver operating characteristic (ROC) curve and was compared to the classic Mehran CA-AKI score. Calibration was assessed using the Hosmer–Lemeshow test.

Results: Overall, 8.4% (71/848) of patients in the development cohort and 11.2% (48/424) of patients in the validation cohort experienced CA-AKI. The simple nomogram included estimated glomerular filtration rate (eGFR), serum albumin (ALB), age and the use of intra-aortic balloon pump (IABP); showed better predictive ability than the Mehran score (C-index 0.756 vs. 0.693, $p = 0.02$); and had good calibration (Hosmer–Lemeshow test $p = 0.187$). Decision curve analysis showed that the nomogram was more clinically useful than the Mehran score.

Conclusions: Our data suggested that the simple nomogram might be a good tool for predicting CA-AKI in high-risk patients with hypoalbuminemia undergoing CAG/PCI, but our findings require further external validation.

Trial registration number NCT01400295

Background

Contrast-associated acute kidney injury (CA-AKI) is one of the major complications that occurs after contrast exposure [1] and is significantly associated with higher mortality and worsened clinical outcomes [2]. Guidelines recommend that patients undergoing coronary angiography (CAG) or percutaneous coronary intervention (PCI) should be assessed for the risk of CA-AKI [3].

Hypoalbuminemia is a frequent problem in patients with cardiovascular disease, with a prevalence ranging from 10–40% in coronary artery disease patients [4–7]. Previous studies have shown that hypoalbuminemia is an independent risk factor for CA-AKI [8] after adjusting for chronic kidney disease (CKD), age or congestive heart failure (CHF). Moreover, the development of AKI is associated with worse survival than the absence of AKI in patients with baseline hypoalbuminemia [9].

Although patients complicated with hypoalbuminemia are at high risk of CA-AKI, based on a systematic search, there is no prediction model to identify such patients. A recent review concluded that only a few published models are available for routine clinical use [10]. Therefore, we conducted this study to establish a simple nomogram for the assessment of CA-AKI risk among patients with hypoalbuminemia undergoing CAG/PCI.

Methods

Patients

This prospective cohort study reviewed all consecutive patients aged ≥ 18 years with baseline hypoalbuminemia [hypoalbuminemia was defined as serum albumin less than 3.5 g/L (< 35 mg/dl)] who were undergoing CAG or PCI and who were included in a prospective observation cohort (PREdictive Value of COntrast voluMe to creatinine Clearance Ratio, PRECOMIN, NCT01400295) in Guangdong Provincial People's Hospital between January 2010 and October 2012 [11]. The exclusion criteria included pregnancy, lactation, intravascular administration of contrast medium within the previous 7 days or 3 days post operation, no use of low-osmolality contrast agents, cardiovascular surgery or endovascular repair, end-stage renal disease or renal replacement, missing preoperative or postoperative creatinine data, malignancy, and no use of isotonic saline for hydration. The Ethics Committee of the Guangdong Provincial People's Hospital approved this study. All the patients involved provided written informed consent.

Finally, approximately 1272 patients were included in the analysis. All eligible patients were randomly (2:1) assigned to a development cohort ($n = 848$) and a validation cohort ($n = 424$). The mean follow-up time was 7.7 (6.8; 8.8) years. All follow-up events were carefully monitored and recorded by trained nurses through office visits or telephone interviews.

Endpoint and definitions

The endpoint of this study was CA-AKI defined as a Scr elevation of ≥ 0.3 mg/dL or 50% from baseline within the first 48 to 72 hours following contrast exposure [12]. CKD was defined as an estimated glomerular filtration rate (eGFR) calculated by the Modification of Diet in Renal Disease (MDRD) formula < 60 mL/min/1.73 m². The definitions of intra-aortic balloon pump (IABP), hypotension, diabetes, and CHF were the same as those used in the Mehran score [13]. Serum albumin (ALB) was analyzed by an automatic biochemical analyzer (Beckman Coulter AU5800, Ireland). The left ventricular ejection fraction (LVEF) was evaluated in all patients using echocardiography within 24 hours before the procedure. Follow-up of mortality was carefully monitored and recorded by trained nurses through office visits and telephone interviews at 1 and 6 months and every 1 year after enrollment until April 2019.

Statistical analysis

Normally distributed continuous variables were compared with an unpaired, 2-tailed t test and are expressed as the mean \pm SD, while nonnormally distributed variables were compared through the

Wilcoxon rank-sum test and are expressed as the median \pm interquartile. Categorical variables were analyzed using the χ^2 test or Fisher's exact test and expressed as percentages. Kaplan-Meier curves were used to explore the association between the CA-AKI and long-term mortality. Multivariate Cox proportional hazards regression was used to evaluate the impact of CA-AKI on the long-term mortality [14, 15].

The associations between CA-AKI and variables in the development cohort were assessed by univariable logistic analysis. Variables with missing values $> 15\%$ were not considered candidates. Significant predictors from the univariable logistic analysis were then included in the multivariable logistic analysis to fit a prediction model. A backward stepwise approach was performed to create a reduced model by successively removing nonsignificant covariates ($P > 0.1$) until all the remaining predictors were statistically significant. For all logistic regression analyses, odds ratios (ORs) with 95% confidence intervals (95% CIs) were calculated. Collinearity between variables was also evaluated. A nomogram was then formulated based on the results and by using the rms package of R. To form the nomogram, each regression coefficient in multivariate logistic regression was proportionally converted into a 0- to 100-point scale. The variable with the highest β coefficient (absolute value) was assigned 100 points. The total points were calculated by adding points of each variable, which were finally converted to predicted probabilities. To assess the clinical use of the nomogram, the final nomogram was tested in the validation cohort. The performance of the nomogram was assessed using the area under the receiver operating characteristic (ROC) curve and concordance index (C index). The nomogram was internally validated with 1000 bootstrap samples to decrease the overfit bias [16, 17]. Calibration was assessed using the Hosmer–Lemeshow test and shown with a calibration curve. The area under the curve (AUC) was calculated in both the development and validation cohorts and compared to that of the Mehran score. Finally, decision curve analysis was performed to compare the clinical discriminative abilities between the new nomogram and the Mehran model.

Missing data were not imputed. In all analyses, $P < 0.05$ was considered statistically significant. All analyses were conducted with R software (version 3.6.2; R Foundation for Statistical Computing, Vienna, Austria) and SPSS (version 25.0).

Results

Baseline characteristics

During this study period, 1272 consecutive patients who had preprocedure hypoalbuminemia were enrolled and divided into the development cohort ($n = 848$) and the validation cohort ($n = 424$). In the total cohort, the incidence of CA-AKI was 9.36% (119/1272). The incidence of CA-AKI in the development cohort and validation cohort was 8.4% (71/848) and 11.2% (48/424), respectively. There were 286 (22.5%) female patients. The mean age was 66.00 ± 10.63 years, and the mean Scr was 102.09 ± 53.63 $\mu\text{mol/L}$. No significant difference was identified between the development and validation cohorts, except for in hydration volume (Table 1).

Table 1
Baseline characteristics of the development cohort and validation cohort

	Development (n = 844)	Validation (n = 428)	p
Age, y	66.02 (10.44)	65.96 (11.02)	0.921
Female, n (%)	204 (24.2)	82 (19.2)	0.051
Weight, n	63.48 (10.10)	62.62 (10.49)	0.361
SBP, mmHg	127.66 (20.48)	128.85 (20.78)	0.329
DBP, mmHg	74.30 (12.08)	74.92 (12.31)	0.385
HR, bpm	75.61 (13.73)	75.60 (14.41)	0.989
Medical history			
CKD, n (%)	251 (29.7)	105 (24.5)	0.059
AMI, n (%)	378 (45.1)	202 (47.3)	0.495
Hypertension, n (%)	494 (58.5)	250 (58.5)	1
Prehypotension, n (%)	13 (1.5)	9 (2.1)	0.621
Hyperlipidemia, n (%)	99 (11.7)	40 (9.3)	0.233
Anemia, n (%)	346 (41.6)	179 (42.2)	0.878
DM, n (%)	219 (25.9)	96 (22.5)	0.2
CHF, n (%)	198 (23.6)	87 (20.4)	0.227
LVEF, n	56.28 (12.98)	55.39 (12.16)	0.262
NYHA	1.92 (0.76)	1.87 (0.67)	0.317
Laboratory examination			
eGFR, mL/min/1.73 m ²	72.30 (22.57)	73.42 (21.68)	0.395
Scr, µmol/L	103.03 (59.22)	100.22 (40.16)	0.379
ALB, g/l	31.59 (2.72)	31.60 (2.74)	0.96
Lpa, mg/dl	34.55 (36.49)	34.10 (38.28)	0.849

Abbreviations: CA-AKI: contrast-associated acute kidney injury; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; CKD: chronic kidney disease; AMI: acute myocardial infarction; DM: diabetes mellitus; CHF: congestive heart failure; LVEF: left ventricular ejection fraction; NYHA: NYHA classification grading of cardiac function; eGFR: estimated glomerular filtration rate; ALB: serum albumin; Lpa: lipoprotein a; BUN: blood urea nitrogen; Dose: dose of contrast media; IABP: intra-aortic balloon pump; Scr: serum creatinine

	Development (n = 844)	Validation (n = 428)	p
BUN, mg/dl	5.61 (3.03)	5.67 (3.02)	0.75
Na, mmol/L	138.82 (3.40)	138.42 (3.43)	0.097
K, mmol/L	3.74 (0.46)	3.72 (0.43)	0.547
Medications			
Metformin, n (%)	31 (3.7)	16 (3.7)	1
ACEI/ARB, n (%)	735 (87.1)	388 (90.7)	0.075
Diuretics, n (%)	187 (22.2)	97 (22.6)	0.603
Beta blocker, n (%)	678 (80.3)	362 (84.6)	0.076
Procedure			
Dose, mL	133.99 (66.22)	131.97 (63.40)	0.603
IABP, n (%)	43 (5.1)	18 (4.2)	0.574
Hydration volume, mL	874.12 (516.30)	816.60 (418.54)	0.047
Other			
CA-AKI, n (%)	71 (8.4)	48 (11.2)	0.129
Mehran score	5.75 (4.74)	5.25 (4.61)	0.075
Abbreviations: CA-AKI: contrast-associated acute kidney injury; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; CKD: chronic kidney disease; AMI: acute myocardial infarction; DM: diabetes mellitus; CHF: congestive heart failure; LVEF: left ventricular ejection fraction; NYHA: NYHA classification grading of cardiac function; eGFR: estimated glomerular filtration rate; ALB: serum albumin; Lpa: lipoprotein a; BUN: blood urea nitrogen; Dose: dose of contrast media; IABP: intra-aortic balloon pump; Scr: serum creatinine			

Compared to patients without CA-AKI, patients with CA-AKI tended to have lower eGFR, LVEF, ALB and higher age, heart rate (HR), and SCR in the development cohort (Supplementary Table 1). Patients with CA-AKI were more likely to be complicated with CKD, CHF, acute myocardial infarction (AMI), hypertension, anemia and other conditions.

Development and validation of a CA-AKI-predicting nomogram

The results of univariate logistic analysis about associations with CA-AKI are shown in Table 2. Through multivariate logistic analysis and stepwise approach, age (OR: 1.036 95% CI: 1.006–1.007), IABP (OR: 8.267 95% CI: 4.007 – 16.978), eGFR (OR: 0.982 95% CI: 0.970–0.994) and ALB (OR: 0.875 95% CI: 0.801 – 0.955) were selected as predictors of CA-AKI (Table 3).

Table 2
Univariate logistic regression

	OR	95% CI	P
eGFR	0.968	0.957–0.979	< 0.001
Scr	1.006	1.003–1.010	< 0.001
Age	1.058	1.030–1.088	< 0.001
AMI	2.113	1.290–3.520	0.003
NYHA	2.290	1.587–3.308	< 0.001
CKD	3.664	2.237–6.066	< 0.001
Hypertension	2.404	1.397–4.342	0.002
IABP	12.788	6.558–24.868	< 0.001
CHF	3.848	2.34–6.338	< 0.001
LVEF	0.966	0.949–0.984	< 0.001
HR	1.018	1.005–1.031	0.005
Anemia	1.802	1.106–2.956	< 0.001
BUN	1.131	1.067–1.197	< 0.001
Diuretics	1.658	1.084–2.465	0.015
Beta-blocker	0.51	0.302–0.885	0.014
Hydration volume	1.001	1.000-1.001	< 0.001

Abbreviations: CA-AKI: contrast-associated acute kidney injury; eGFR: estimated glomerular filtration rate; AMI: acute myocardial infarction; Dose: dose of contrast media; IABP: intra-aortic balloon pump; CHF: congestive heart failure; LVEF: left ventricular ejection fraction; HR: heart rate; ALB: serum albumin; BUN: blood urea nitrogen; infarction; Scr: serum creatinine

Table 3
Multivariate logistic regression

	OR	95% CI	p
eGFR	0.982	0.97–0.994	0.004
ALB	0.875	0.801–0.955	0.002
IABP	8.267	4.007–16.978	< 0.001
Age	1.036	1.006–1.067	0.018

Abbreviations: eGFR: estimated glomerular filtration rate; ALB: serum albumin; IABP: intra-aortic balloon pump;

These independently associated risk factors were used to form a simple nomogram for CA-AKI (Fig. 1). In the development cohort, the nomogram demonstrated good discriminative power in estimating the risk of CI-AKI, with an unadjusted C-statistic of 0.816 (95% CI, 0.763–0.862) and a bootstrap-corrected C statistic of 0.802. Compared to the Mehran score, the nomogram had good discrimination (AUC 0.816 VS 0.775, $p = 0.082$, Fig. 2). In addition, calibration plots graphically showed good agreement regarding the presence of CA-AKI between the risk estimation and the observed frequency (Fig. 3). The Hosmer–Lemeshow statistic of multivariable analysis suggested a good fit ($\chi^2 = 3.65$, $P = 0.887$) in the development cohort.

In the validation cohort, the simple nomogram demonstrated a C-statistic of 0.756 (95% CI 0.685–0.832), which was better than that of the Mehran score (AUC: 0.693, 95% CI 0.608–0.779) among patients ($P = 0.02$, Fig. 4). This means that there is no significant difference between the model's predicted value and the actual predicted value. Moreover, there was also a good calibration curve for risk estimation (Fig. 3). The Hosmer–Lemeshow statistic of multivariable analysis did not perform a lack of fit ($\chi^2 = 11.27$, $P = 0.187$) in the validation cohort. Decision curve analysis was used to facilitate the comparison between the new nomogram and the Mehran score. The clinical usefulness of new nomogram was better than that of the Mehran score in development and validation cohort (Supplementary Fig. 1A and B).

Follow-up of clinical outcomes

We divided patients into 2 groups according to whether they developed CA-AKI. The median follow-up period was 7.7 (6.8; 8.8) years. The rates of mortality were 30.2% and 17.1% in the CA-AKI group and non-CA-AKI group, respectively. According to the log-rank analysis (Fig. 5), patients with CA-AKI presented a worse long-term outcome ($P < 0.01$). By multivariate Cox regression analysis, CA-AKI was independently associated with the long-term mortality (Hazard ratio:1.53, 95% CI 1.03–2.25) after adjusting for diabetes, age, gender, CHF, AMI, hypertension, IABP and eGFR (Supplementary Table 2).

Discussion

Our study was the first to develop a CA-AKI risk stratification model in a population with hypoalbuminemia. We established a simple nomogram that included four powerful predictors for clinical use (IABP, ALB, eGFR, age). Compared to the classical Mehran score, the new simple nomogram had good discrimination and calibration in predicting CA-AKI.

In our study, the incidence of CA-AKI was 9.36%, which was relatively high but similar to the high-risk population, such as patients with CKD or AMI [18, 19]. Several studies have demonstrated that hypoalbuminemia is an independent risk factor for CA-AKI [8] and is closely related to poor prognosis in coronary heart disease [20]. Yu et al. [9] reported that patients with hypoalbuminemia had a high incidence of AKI in the hospital. Furthermore, in our study, the development of CA-AKI was associated with a poor prognosis according to the results of the 7-year follow-up, which suggested the importance of CA-AKI prevention.

Risk assessment in high-risk groups is a primary aim and important for the prevention of CA-AKI, so a large number of models have been proposed [10]. Although the classic Mehran score or other models had good predictive efficiency, most models included 5–8 variables, and some factors required the subjective judgment of clinicians. In recent years, predictive models have been established for different populations at high risk of CA-AKI, such as patients with CKD [21], AMI [22] or diabetes mellitus [23]. However, there was no predictive model for patients with hypoalbuminemia. Because of the high incidence of CA-AKI in patients with hypoalbuminemia, it is important to develop a simple risk score for these patients undergoing CAG/PCI.

For predicting CA-AKI, the currently available models seldom included albumin [10] because serum albumin was a novel laboratory risk factor. Murat et al. [8] suggested that albumin is an independent and good predictor of CA-AKI among patients with ACS undergoing PCI. Low serum albumin concentrations reflect the inflammatory state of the body, which may lead to CA-AKI [24].

The use of IABP was the strongest predictor of CA-AKI in the present model. IABP, a reflection of hemodynamics, was an independent predictor of CI-AKI, which has been reported in previous studies [21, 25]. Bartholomew et al. [26] first reported a close association between the use of IABP and CA-AKI, and then Mehran et al. [13] first included IABP in a model of CA-AKI prediction. Perioperative hemodynamic disorders may lead to ischemia-reperfusion injury, which may have a potential contribution to AKI.

In the present risk model, age was an independent predictor of the occurrence of CI-AKI [13, 27]. This independent predictive ability may be related to a degenerative changes in the structure and function of kidneys with increasing age. Baseline eGFR was also a common risk factor for CA-AKI following CAG [28]. eGFR represents worse kidney function and a higher risk of acute kidney injury [29].

Diabetes and contrast volume were not included in our nomogram, although these variables were included in previous models [30]. In our study, diabetes and contrast volume were not independent risk factors for CA-AKI based on statistical analysis. Similar to our finding, Sabeti et al. [31] showed that diabetes mellitus was not an independent risk factor for CA-AKI. A recent review concluded that diabetes

is not independently associated with the risk of developing CA-AKI and only increases susceptibility in patients with underlying kidney disease [32]. Regarding contrast volume, several studies suggested that the development of AKI following contrast exposure is significantly determined by the presence of comorbidities and hemodynamic instability rather than contrast media [33, 34]. Hydration has been considered an effective treatment for CA-AKI, but the actual effect of this strategy is still controversial. Perioperative hydration can expand plasma volume with suppression of the renin-angiotensin-aldosterone system, down-regulation of tubuloglomerular feedback, a decrease of contrast medium concentration in the tubule lumen and vasa recta, and counteraction of medullary vasoconstriction activation[35]. It is still the cornerstone of CIN prevention. But, the rate and duration of hydration remain inconsistent. Meanwhile, the AMACING trial challenged the tenet that intravenous fluids are effective. There was no significant difference in the incidence of CI-AKI between the hydration group and the non-hydration group (2.7% vs 2.6%)[36]. In our study, the volume of hydration was not significantly different in multivariate regression, so this variable was not included in our model. By using our nomogram, clinicians can identify high-risk patients with CA-AKI early and treat them in a timely manner.

Limitations

Our study had some limitations. First, this study was based on data from a single center. However, our cohort is one of the largest CA-AKI databases among patients with hypoalbuminemia.

Second, our model is not as effective as other models, but it has fewer objective variables and higher clinical operability. Importantly, our model has good predictive and evaluative effectiveness in hypoalbuminemia populations.

Third, the nomogram established has not been externally verified. However, we randomly divided all the patients into a development group and a validation group according to a 2:1 ratio, which showed that our model had good stability.

Finally, several patients were discharged 3 days after the operation, so serum creatinine was not measured after 3 days in these patients. This might decrease the development of CA-AKI.

Abbreviations

CA-AKI: contrast-associated acute kidney injury; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; CKD: chronic kidney disease; AMI: acute myocardial infarction; DM: diabetes mellitus; CHF: congestive heart failure; LVEF: left ventricular ejection fraction; NYHA: NYHA classification grading of cardiac function; eGFR: estimated glomerular filtration rate; ALB: serum albumin; Lpa: lipoprotein a; BUN: blood urea nitrogen; Dose: dose of contrast media; IABP: intra-aortic balloon pump; Scr: serum creatinine

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Guangdong Provincial People's Hospital.

Consent for publication

Not required.

Availability of data and materials

Data relevant to this study are available from the corresponding authors upon reasonable request.

Competing interests

All authors declare that they have no competing interests.

Funding

This work was supported by the Beijing Lisheng Cardiovascular Pilot Foundation [grant no. LHJJ201612127] and the National Science Foundation of China (grant no. 81670339). The funding body did not participate in the design of the study; the collection, analysis, and interpretation of the data; or in the writing of the manuscript.

Authors' contributions

Substantial contributions to the conception and design of the study (LWL, JL, LL, BW); data collection (GS, ZG, YH, FS, LZ, GC, BL); data analysis and/or interpretation of data for the work (LWL, LL, LY, JYC); drafting of the work or revising it critically for important intellectual content (LWL, LL, JL, LY, JYC); final approval of the version to be published (all the authors).

Acknowledgements

None.

Authors' information

Jiyan Chen takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

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Conflicts of interest: None.

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Figures

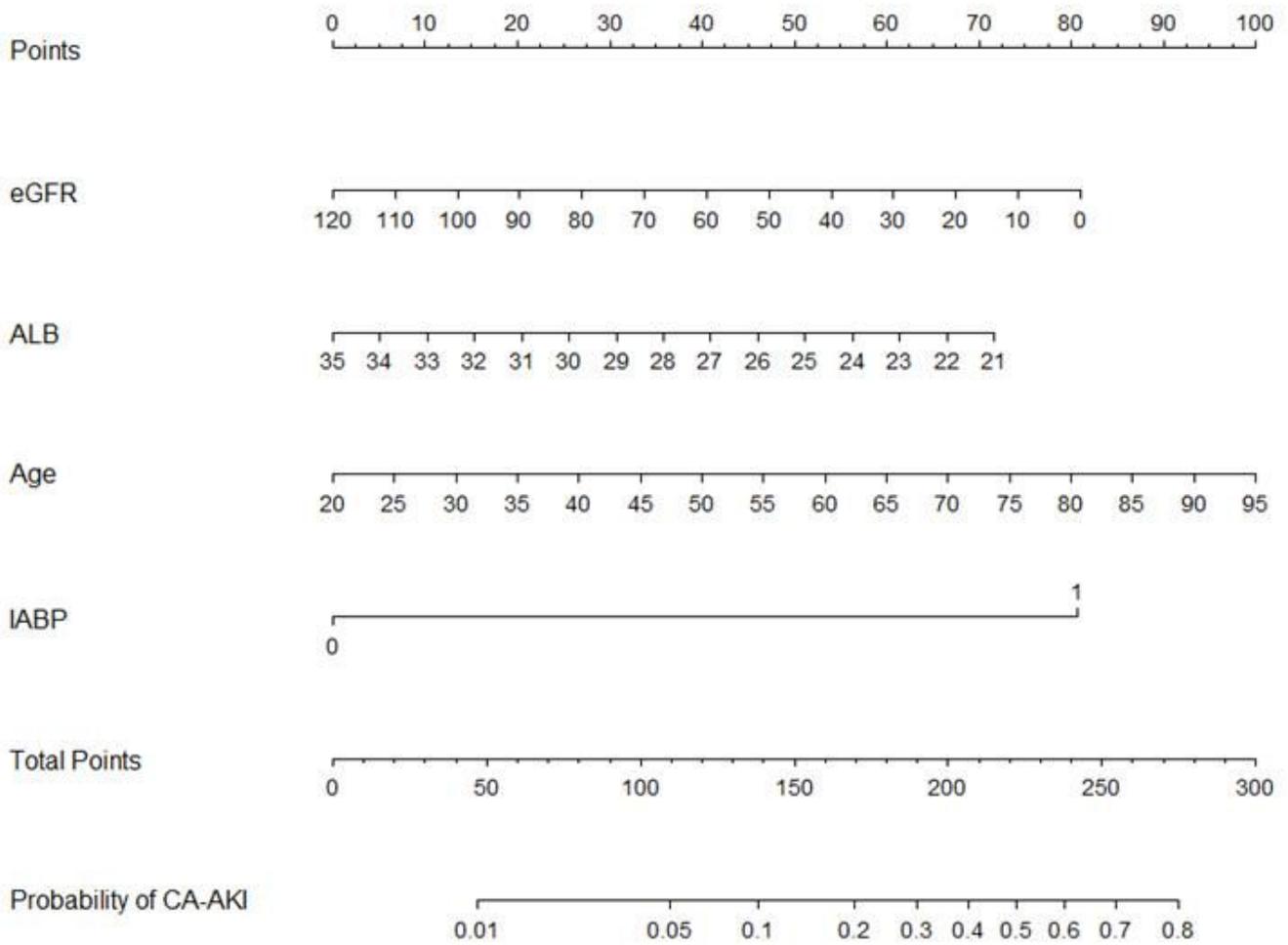


Figure 1

Nomogram to estimate the risk of CA-AKI

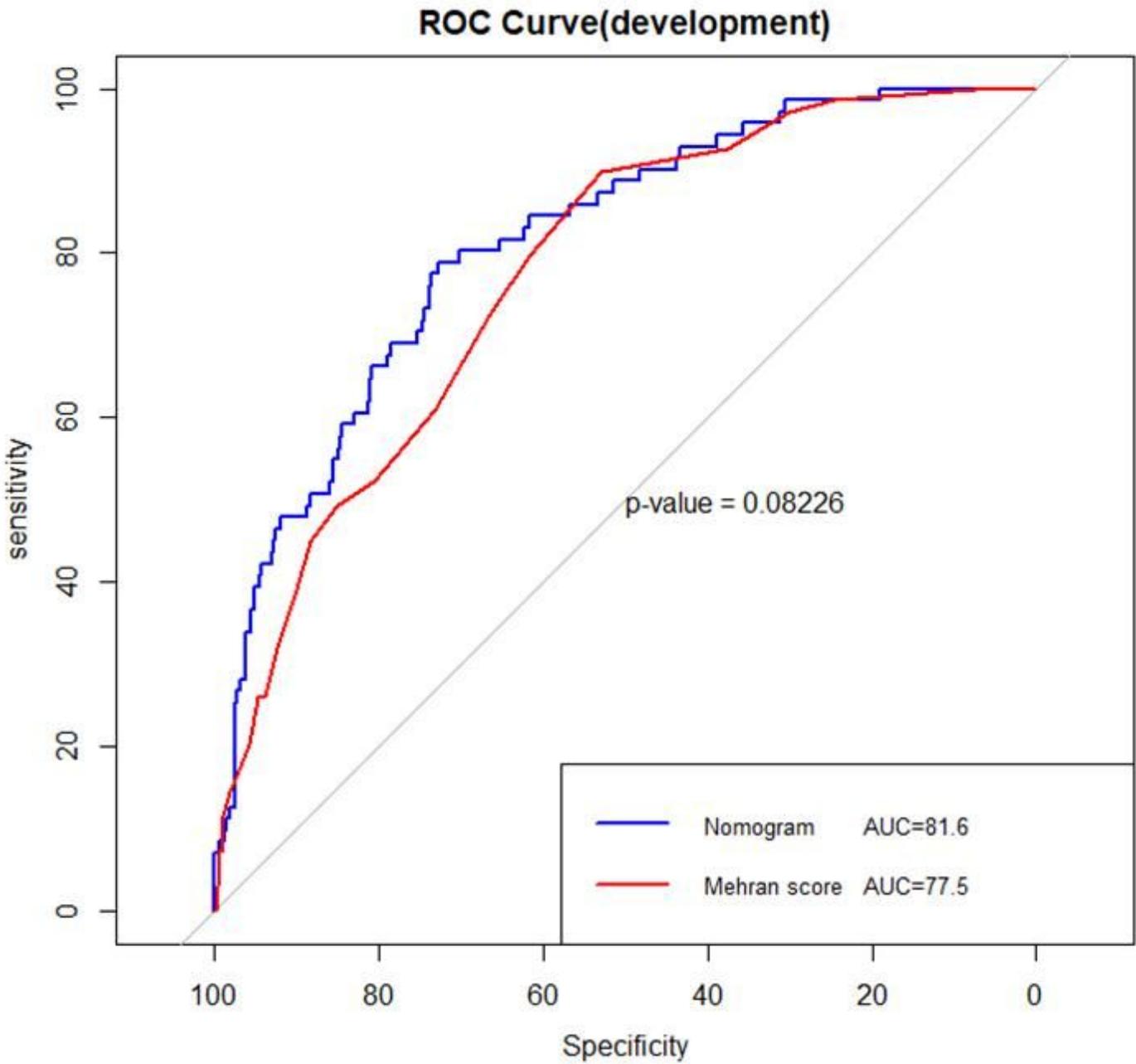


Figure 2

The receiver operator characteristic curves of the nomogram and the Mehran score in the development cohort.

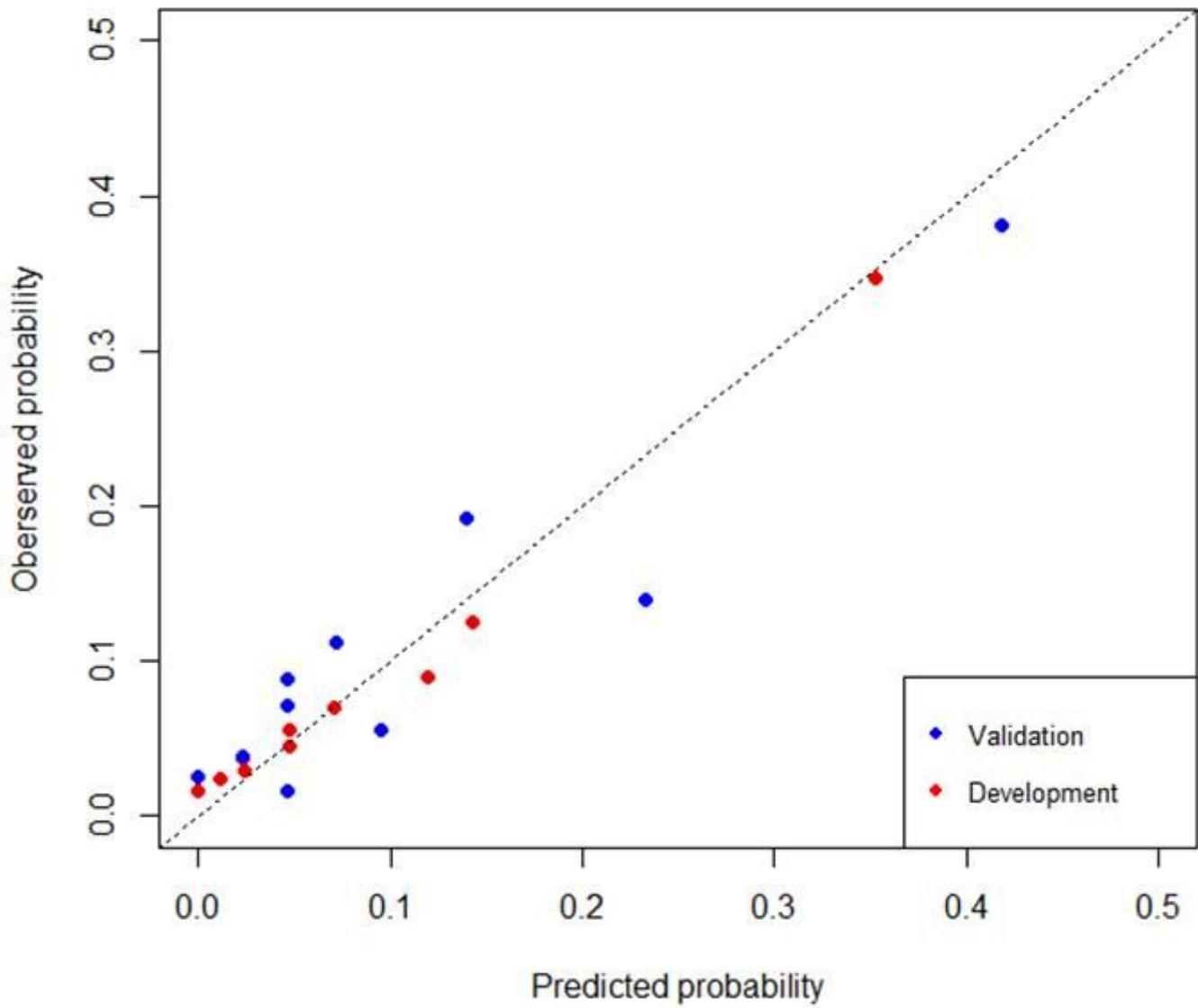


Figure 3

Validity of the predictive value of the nomogram in the development and validation cohort.

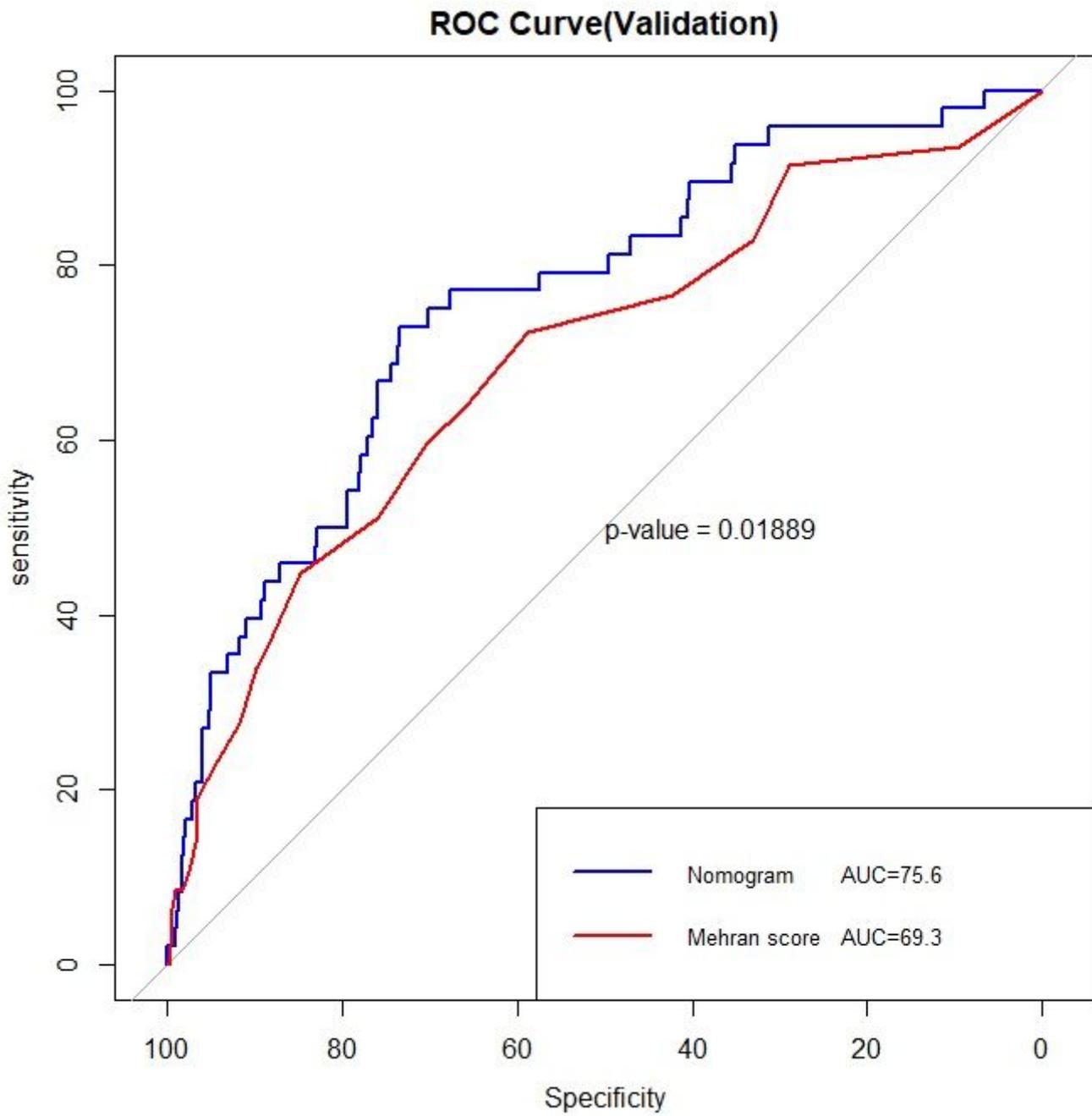


Figure 4

The receiver operator characteristic curves of the nomogram and the Mehran score in the validation cohort.

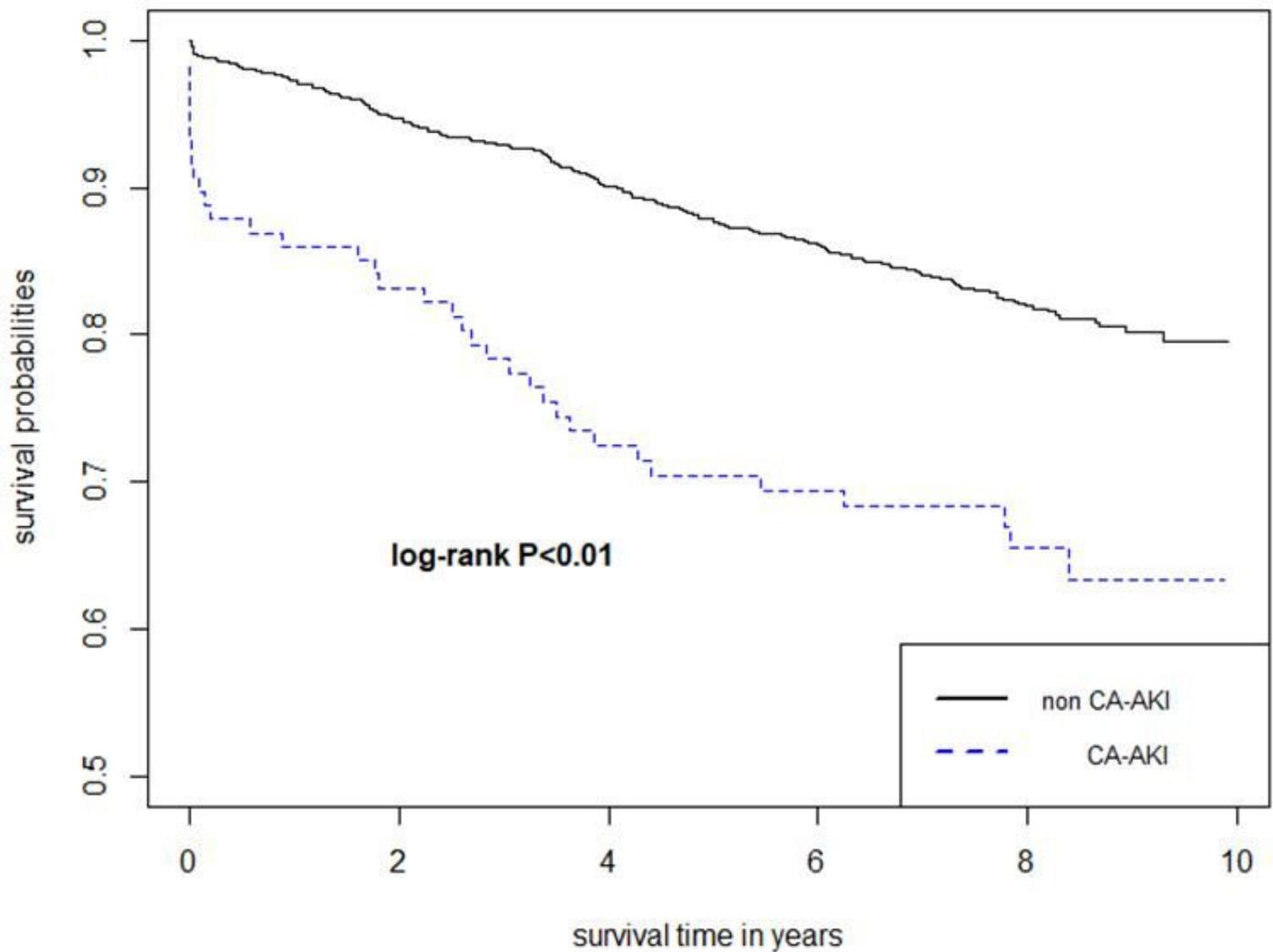


Figure 5

Association between CA-AKI and long-term mortality in patients with hypoalbuminemia

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