International Journal of General Medicine

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Renal and Cardiovascular Outcomes of IABP-Assisted PCI in Renal Dysfunction: A Propensity-Matched Study and MACEs Prediction Model Development

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Background: The renal safety and cardiovascular outcomes of intra-aortic balloon pump (IABP) support during percutaneous coronary intervention (PCI) in patients with renal dysfunction remain controversial.

Objective: To investigate the effects of IABP on renal function and major adverse cardiovascular events (MACEs) in PCI patients with renal dysfunction, and to establish an individualized prognostic prediction model.

Methods: In this retrospective cohort study, 253 PCI patients with renal dysfunction from Meizhou People's Hospital (January-December 2023) were analyzed. Propensity score matching (1:4 IABP/non-IABP) balanced baseline characteristics. Least absolute shrinkage and selection operator (LASSO) regression identified predictors for MACEs, followed by nomogram development and validation.

Results: Post-matching (IABP = 16, non-IABP = 64), despite higher baseline risk in the IABP group, no intergroup differences occurred in postoperative creatinine, eGFR, or MACEs incidence (all $P > 0.05$). However, this group exhibited greater eGFR decline from baseline. Multivariate analysis identified five independent MACEs predictors: preoperative B-type natriuretic peptide (BNP), postoperative BNP, preoperative neutrophil-to-lymphocyte ratio (NLR), postoperative blood urea nitrogen (BUN), and prealbumin levels. The prediction model achieved AUCs of 0.869 (*95% CI*: 0.774–0.964) and 0.843 (*95% CI*: 0.678–1.000) in training and validation sets, with decision curve analysis confirming clinical utility at 10%–80% risk thresholds.

Conclusion: IABP may exacerbate subclinical renal injury in PCI patients with renal dysfunction without mitigating MACEs risk. The validated nomogram provides individualized risk stratification to guide clinical management.

Keywords: intra-aortic balloon pump, renal dysfunction, percutaneous coronary intervention, prognostic model, propensity score matching, major adverse cardiovascular events

Introduction

Renal dysfunction is an independent risk factor for adverse outcomes in cardiovascular disease patients, with risk severity inversely correlated with estimated glomerular filtration rate (eGFR) levels.[1–3] Approximately two-thirds of patients with kidney disease also have concomitant coronary artery disease, which is the leading cause of death in this population.[4] Early revascularization may delay disease progression and improve clinical outcomes in this population.[5] However, such patients frequently present with complex coronary lesions (eg, multivessel disease, severe calcification, and peripheral artery disease),[6,7] which significantly increase percutaneous coronary intervention (PCI) procedural risks.

Percutaneous mechanical circulatory support (pMCS) technologies, including intra-aortic balloon pump (IABP), Impella, TandemHeart, and Extracorporeal Membrane Oxygenation, have emerged as a crucial strategy to enhance the

Received: 7 May 2025
Accepted: 9 August 2025
Published: 18 August 2025

safety and procedural success of high-risk PCI patients. The primary objectives of utilizing pMCS are to maintain hemodynamic stability, ensure coronary perfusion, and minimize end-organ hypoperfusion. By providing circulatory support, these devices enable interventional cardiologists to perform complex coronary interventions that would otherwise be considered prohibitively risky.[8] For cardiogenic shock patients with chronic kidney disease, current research reveals a mortality benefit: pMCS recipients show significantly lower in-hospital mortality than those without pMCS support.[9] However, there is a lack of dedicated data on the role of pMCS as an adjunctive therapy during PCI specifically in patients with renal dysfunction.

Compared with other pMCS devices, IABP provides the least hemodynamic support but is most widely used in clinical practice due to its simplicity of management and fewer complications.[10,11] While IABP may theoretically preserve renal function through hemodynamic optimization, current evidence regarding its clinical impact on kidney outcomes remains scarce.[12] The absence of randomized controlled trials substantially limits our understanding of the renal effects of IABP to hypothesis-driven observations derived from retrospective analyses and pathophysiological extrapolations.[13] Currently, no specific recommendations for or against the use of IABP in patients with renal dysfunction undergoing PCI. Divergent findings have been reported across clinical studies: studies by Miller et al[14] and Lansky et al[15] have shown that the use of the IABP may lead to renal dysfunction, whereas the landmark IABP-SHOCK II trial (2012) reported no significant renal impairment with IABP use.[16] Another study that summarized different renal function trajectories after LVAD placement further indicated that the impact of IABP use on renal function remains inconclusive.[17] Notably, IABP carries inherent risks potentially compromising renal homeostasis, including mechanical complications (valvular injury, vascular trauma, and limb ischemia) and hematological disturbances (hemorrhage and hemolysis).[18] This heterogeneity in reported outcomes underscores the critical need for dedicated investigation in patients with established renal dysfunction, the specific population targeted in our study.

Critically, prior investigations did not adequately stratify participants by baseline renal status (normal function vs established dysfunction). This limitation in study design likely contributes to the observed discrepancies in outcomes. To address this evidence gap, our study specifically enrolled patients with preexisting renal dysfunction. We employed propensity score matching (PSM) to minimize selection bias by adjusting for key covariates.[19] The co-primary objectives of this study were to evaluate renal outcomes after IABP-assisted PCI in patients with renal dysfunction and to construct a risk prediction model to identify patients at risk of experiencing major adverse cardiovascular events (MACEs).

Materials and Methods

Study Population

This retrospective study enrolled patients with renal dysfunction (eGFR <60 mL/min/1.73m² or serum creatinine exceeding the upper limit of the institutional reference range) who underwent PCI at the Cardiovascular Center of Meizhou People's Hospital between January and December 2023. The inclusion criteria were: (1) Coronary angiography-confirmed stenosis ≥70% in at least one major coronary vessel; (2) No requirement for hemodialysis. The exclusion criteria were: (1) Moderate-to-severe aortic regurgitation or aortic dissection; (2) Missing clinical data.

This study was performed in accordance with the ethical standards of the Declaration of Helsinki and approved by the Human Ethics Committee of Meizhou People's Hospital.

Methods

Data Collection

Demographic and clinical data were extracted, including age, sex, body mass index (BMI), comorbidities (hypertension, diabetes, etc)., smoking status, cardiogenic shock, blood pressure, uric acid, liver function, albumin, globulin, prealbumin, triglycerides, total cholesterol, low-density lipoprotein cholesterol (LDL-C), pre- and post-PCI troponin, B-type natriuretic peptide (BNP), serum creatinine, blood urea nitrogen (BUN), complete blood count, number of diseased coronary vessels, stent count, procedural duration, and contrast volume. The neutrophil-to-lymphocyte ratio (NLR) was calculated as the absolute neutrophil count divided by the absolute lymphocyte count. The eGFR was estimated using the

2021 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[20] Preoperative laboratory tests were obtained within 24 hours of admission, while postoperative assessments were performed within 4 hours after PCI.

Grouping

Patients were stratified into the IABP group, who received IABP support before or during PCI with device removal upon procedure completion, and the non-IABP group, which underwent standard PCI without mechanical circulatory support.

Endpoints

The primary outcome was MACEs, a composite of in-hospital: cardiovascular death, cardiac arrest, malignant arrhythmia, non-fatal stroke, and acute heart failure.

Statistical Analysis

Data were analyzed using R software (v.4.4.1). Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range [IQR]), compared via independent *t*-test or Mann–Whitney *U*-test. Categorical variables were expressed as frequency (%), compared using-χ^2 test or Fisher's exact test.

Propensity Score Matching Analysis

Cardinality matching was performed at a 1:4 ratio using the "MatchIt" package in R. This method employs mixed integer programming to select the largest possible balanced subset of the original cohort under strict predefined balance constraints,[21] requiring standardized mean differences (*SMD*) ≤0.10 for all covariates.[22] Unlike traditional nearest-neighbor matching, this approach operates without caliper widths or pair formation, prioritizes global balance over pairwise similarity, and executes matching without replacement. The dependent variable was IABP use, with covariates comprising age, gender, systolic/diastolic blood pressure, cardiogenic shock status, left ventricular ejection fraction, preoperative serum creatinine, and contrast volume.

Predictive Modeling

The Least absolute shrinkage and selection operator (LASSO) regression analysis was identified MACEs predictors. A nomogram was constructed and evaluated using Receiver operating characteristic (ROC) curve (discrimination), Calibration curve (goodness-of-fit), and Decision curve analysis (DCA) (clinical utility). A two-tailed P-value less than 0.05 was considered significant.

Results

Baseline Characteristics

A total of 253 patients undergoing PCI were included, of whom 38 (15.0%) received IABP support. Before PSM, the IABP group exhibited more severe hemodynamic impairment (systolic blood pressure: 114.76 ± 18.41 vs 127.88 ± 19.26 mmHg, $P < 0.001$) and worse cardiac function (Left ventricular ejection fraction: 38.50% vs 62.00%, $P < 0.001$). After 1:4 matching, baseline characteristics were well-balanced (all *SMD* < 0.1), with 80 patients (IABP group: n = 16; non-IABP group: n = 64) included in the final analysis (Table 1).

Clinical and Procedural Outcomes

Before PSM, compared with the non-IABP group, the IABP group had higher prevalence of hypertension ($P < 0.05$); elevated levels of total cholesterol, LDL-C, aspartate aminotransferase (AST), alanine aminotransferase (ALT), BUN, troponin, BNP, white blood cell (WBC) count, NLR, and postoperative creatinine ($P < 0.05$); lower serum albumin and prealbumin levels ($P < 0.05$); longer hospitalization duration ($P < 0.05$); and higher incidence of MACEs ($P < 0.05$).

After PSM, the IABP group still showed significantly higher AST, ALT, preoperative and postoperative troponin, preoperative and postoperative WBC count, preoperative NLR, postoperative BNP, postoperative BUN ($P < 0.05$), and longer hospitalization duration ($P < 0.05$). However, no significant differences were observed in postoperative creatinine or MACEs incidence (Table 2).

Table 1 Comparison of Baseline Data of the Study Population Before and After Propensity Score Matching

Baseline Data	Before PSM				After PSM			
	non-IABP (n=215)	IABP (n=38)	*P*	*SMD*	non-IABP (n=64)	IABP (n=16)	*P*	*SMD*
Age(years)	70 (61,77)	69 (59.50,76)	0.488	−0.1435	74.50 (65.75,80)	72 (67.50,78.50)	0.866	0.0464
Gender (n, %)			0.611	0.0514			1.000	0.0156
Male	170 (79.07)	32 (84.21)			59 (92.19)	15 (93.75)		
Female	45 (20.93)	6 (15.79)			5 (7.81)	1 (6.25)		
Cardiogenic shock (n, %)	1 (0.47)	22 (57.89)	**<0.001**	0.5743	0	0		0
Systolic Blood Pressure (mmHg)	127.88±19.26	114.76±18.41	**<0.001**	−0.7125	121.98±18.45	121.19±15.79	0.863	−0.0433
Diastolic Blood Pressure (mmHg)	75 (67,81.50)	70 (65,78)	**0.031**	−0.3688	71 (65,78)	71 (65.50,78.75)	0.928	−0.038
Left ventricular ejection function (%)	62 (52.50,65)	38.50 (28.50,50)	**<0.001**	−1.2455	47.38±13.25	46.69±13.33	0.855	−0.0482
Preoperative Serum creatinine (mol/L)	114.30 (104,141)	125 (105,157.20)	0.276	0.1696	116.25 (105,139.48)	116.85 (99.62,126.30)	0.400	−0.0424
Contrast Volume (mL)	60 (60,80)	80 (60,80)	**<0.001**	0.3998	60 (60,80)	80 (60,80)	0.169	0.0363

Note: Bold indicates statistical significance.

Risk Factors for MACEs

Within the entire cohort, a total of 63 MACEs were recorded. The specific breakdown of these composite endpoint components was as follows: cardiovascular death (n = 1), cardiac arrest (n = 5), malignant arrhythmia (n = 5), non-fatal stroke (n = 5), and acute heart failure (n = 47).

Following PSM, patients were stratified into MACEs (n = 19) and no-MACEs (n = 61) groups. Compared with the no-MACEs group, the MACEs group exhibited with lower left ventricular ejection fraction, serum albumin, and prealbumin levels ($P < 0.05$); higher total cholesterol, LDL-C, preoperative WBC count, preoperative NLR, postoperative BUN, and postoperative troponin ($P < 0.05$); and a greater proportion of patients with elevated pre operative and postoperative BNP levels ($P < 0.05$) (Table 3).

Subsequently, the matched dataset was split into training (n = 56, 70%) and validation (n = 24, 30%) sets. LASSO regression identified five independent predictors of MACEs, including preoperative BNP, postoperative BNP, preoperative NLR, postoperative BUN, and serum prealbumin (Table 4, Figure 1).

Nomogram Development and Validation

The final Lasso model employed $\lambda = 0.118$ (lambda. 1se), and a nomogram incorporating these five variables was constructed using logistic regression (Figure 2). Using bootstrap resampling (1,000 iterations), the optimism-corrected C-index was 0.7314 (*95% CI*: 0.5214–0.9413). While the wide confidence interval reflects sample size limitations, the point estimate supports discriminative capacity. The mean C-index obtained through ten-fold cross-validation was 0.7042. Model performance was assessed by ROC analysis with an area under the curve (AUC) of 0.869 (*95% CI*: 0.774–0.964) in the training set and 0.843 (*95% CI*: 0.678–1.000) in the validation set (Figure 3A and D). The Hosmer-Lemeshow test showed good fit (training set: $P = 0.795$; validation set: $P = 0.938$) (Figure 3B and E). DCA demonstrated the model provided net clinical benefit at threshold probabilities of 10–80% (Figure 3C and F).

Discussion

Using PSM analysis, this study revealed that despite worse baseline hemodynamics and cardiac function in the IABP group, there were no significant differences in postoperative serum creatinine levels or MACEs between groups after matching. Notably, preoperative BNP and NLR, along with postoperative BNP, BUN, and prealbumin levels, emerged as independent predictors of MACEs in renal dysfunction patients undergoing PCI. The developed nomogram demonstrated

Table 2 Comparison of Characteristics of the Study Population Before and After Propensity Score Matching

Variable	Before PSM				After PSM			
	non-IABP (n=215)	IABP (n=38)	*OR (95% CI)*	*P*	non-IABP (n=64)	IABP (n=16)	*OR (95% CI)*	*P*
Hypertension (n, %)	147 (68.37)	19 (50)	0.46 (0.23;0.94)	**0.044**	36 (56.25)	9 (56.25)	1.00 (0.32;3.16)	1.000
Diabetes (n, %)	85 (39.53)	20 (52.63)	1.69 (0.84;3.43)	0.183	21 (32.81)	9 (56.25)	2.59 (0.84;8.34)	0.149
Smoking (n, %)	36 (16.74)	6 (15.79)	0.95 (0.33;2.31)	1.000	14 (21.88)	4 (25)	1.21 (0.29;4.20)	0.749
BMI ($kg/m^{2)}$	24.30 (21.95,26.36)	23.32 (21.26,25.95)	0.97 (0.89;1.07)	0.288	23.33±3.18	25.31±5.30	1.15 (0.99;1.34)	0.170
Uric Acid (umol/l)	462.70 (405.55,529.50)	491.70 (389.33,606.97)	1.00 (1.00;1.00)	0.269	482.74±115.15	490.42±136.31	1.00 (1.00;1.01)	0.838
Preoperative Blood Urea Nitrogen (mmol/l)	6.86 (5.64,8.71)	8.36 (7.54,11.40)	1.14 (1.05;1.24)	**<0.001**	7.07 (5.94,8.97)	8.25 (7.46,8.59)	1.07 (0.90;1.26)	0.174
Preoperative eGFR	51.70 (40.05,61.38)	51.16 (36.11,60)	0.99 (0.97;1.02)	0.515	49.89±13.11	51.87±13.20	1.01 (0.97;1.06)	0.596
Creatine Kinase Isoenzyme MB (U/L)	15.70 (12.35,19.95)	73.10 (28.25,264.98)	1.04 (1.02;1.06)	**<0.001**	16.55 (12.55,20.05)	34.65 (21.12,68.33)	1.15 (1.05;1.26)	**<0.001**
Preoperative Troponin (ng/mL)	0.01 (0.01,0.03)	15.43 (2.07,50)	1.25 (1.12;1.39)	**<0.001**	0.02 (0.01,0.05)	4.69 (0.74,30.97)	19.6 (2.20;174)	**<0.001**
Preoperative BNP (pg/mL)	77.30 (23.90,229.55)	617.85 (244.55,1885.60)	1.00 (1.00;1.00)	**<0.001**	196.50 (76.65,871.55)	361.05 (160.58,696.38)	1.00 (1.00;1.00)	0.215
Preoperative White Blood Cells ($\times 10^9$/L)	7 (5.90,8.45)	12.75 (9.80,19.12)	1.68 (1.43;1.99)	**<0.001**	7.05 (5.80,8.33)	9.95 (8.40,12.88)	1.78 (1.29;2.46)	**<0.001**
Preoperative Red Blood Cells ($\times 10^{12}$/L)	4.39±0.73	4.47±0.70	1.16 (0.72;1.86)	0.529	4.38 (3.97,4.83)	4.23 (3.81,4.70)	0.70 (0.30;1.65)	0.467
Preoperative Hemoglobin (g/L)	135 (117,145)	135.50 (122.50,149)	1.01 (0.99;1.03)	0.295	131.38±18.60	132.88±16.19	1.00 (0.97;1.04)	0.751
Preoperative Red Cell Distribution Width (fL)	43.70 (41.45,45.30)	43.05 (42,45.92)	1.07 (0.97;1.17)	0.483	44.15 (42.77,46.50)	44.05 (43,45.78)	1.04 (0.90;1.19)	0.971
Preoperative Platelets ($\times 10^9$/L)	209 (171,246)	203 (175.25,259.75)	1.00 (1.00;1.01)	0.697	201.50 (162.50,238.50)	196.50 (163.50,270.25)	1.00 (0.99;1.01)	0.622
Preoperative NLR	3.02 (2.29,4.39)	6.87 (3.75,12.23)	1.20 (1.11;1.29)	**<0.001**	3.12 (2.30,4.33)	4.67 (3.30,7.98)	1.27 (1.05;1.54)	**0.039**
Triglycerides (mmol/l)	1.55 (1.12,2.24)	1.44 (1.15,2.13)	0.92 (0.69;1.23)	0.742	1.29 (0.91,1.87)	1.48 (1.17,2.46)	1.39 (0.86;2.23)	0.248
Cholesterol (mmol/l)	4.09 (3.45,4.92)	4.71 (3.86,5.53)	1.39 (1.06;1.83)	**0.012**	4.13±0.95	4.77±1.34	1.77 (1.03;3.03)	0.089
Low-Density Lipoprotein (mmol/l)	2.27 (1.95,3.02)	2.83 (2.20,3.53)	1.66 (1.13;2.44)	**0.006**	2.44±0.73	2.94±0.97	2.26 (1.08;4.75)	0.071
Serum Albumin (g/L)	38.60 (35.60,41)	33.05 (30.95,38.47)	0.83 (0.76;0.90)	**<0.001**	37.56±3.40	35.94±5.29	0.89 (0.77;1.04)	0.260
Serum Globulin (g/L)	26.90 (24.25,29.40)	26.70 (24.80,29.70)	1.01 (0.93;1.10)	0.707	27.11±4.09	29.16±4.94	1.11 (0.98;1.26)	0.140
Serum Prealbumin (mg/L)	241.80 (196.15,284.90)	176.75 (119.25,221.73)	0.98 (0.98;0.99)	**<0.001**	219.48±62.04	197.81±76.86	0.99 (0.99;1.00)	0.308
Aspartate Aminotransferase (U/L)	20 (16,25.05)	103.50 (48,382.50)	1.02 (1.01;1.02)	**<0.001**	22 (17.75,29.25)	45.25 (23,67)	1.06 (1.02;1.10)	**0.002**
Alanine Aminotransferase (U/L)	18 (14,27)	63 (24.25,122.50)	1.02 (1.01;1.02)	**<0.001**	19 (14,29.50)	24.50 (21.50,47.38)	1.03 (1.00;1.06)	**0.022**

(*Continued*)

https://doi.org/10.2147/IJGM.S532170

https://doi.org/10.2147/IJGM.S532170

Table 2 (Continued).

Variable	Before PSM				After PSM			
	non-IABP (n=215)	IABP (n=38)	*OR (95% CI)*	*P*	non-IABP (n=64)	IABP (n=16)	*OR (95% CI)*	*P*
Number of Coronary Artery Lesions (n, %)				0.693				0.465
1	11 (5.12)	2 (5.26)			3 (4.69)	2 (12.50)		
2	23 (10.70)	2 (5.26)	0.49 (0.05;5.21)		8 (12.50)	1 (6.25)	0.22 (0.01;3.70)	
3	181 (84.19)	34 (89.47)	0.98 (0.24;7.13)		53 (82.81)	13 (81.25)	0.37 (0.05;3.42)	
Main Coronary Artery Lesion (n, %)	55 (25.58)	15 (39.47)	1.90 (0.90;3.89)	0.117	16 (25)	8 (50)	2.95 (0.93;9.50)	0.069
Number of Coronary Stents Implanted	2 (1,2)	2 (1,2)	1.05 (0.70;1.56)	0.56	2 (1,2)	2 (1,2)	0.97 (0.54;1.72)	0.728
Surgical Time (min)	52 (40,70)	50 (36.25,84.25)	1.00 (0.99;1.02)	0.888	60 (45,80)	60.50 (40,85)	1.00 (0.98;1.02)	0.947
Postoperative Serum Creatinine (umol/l)	113.00 (101.25,134.00)	134.75 (105.95,167.50)	1.01 (1.00;1.02)	**0.016**	115.50 (101.67,137.92)	125.20 (106.85,141.62)	1.00 (0.99;1.02)	0.463
Postoperative Blood Urea Nitrogen (mmol/l)	6.29 (5.23,8.27)	9.84 (8.30,13.56)	1.30 (1.18;1.44)	**<0.001**	6.98 (5.48,9.12)	8.68 (7.46,10.75)	1.17 (1.00;1.37)	**0.019**
Postoperative eGFR	51.83±15.63	47.51±18.67	0.98 (0.96;1.00)	0.185	51.45 (15.33)	48.72 (15.03)	0.99 (0.95;1.02)	0.523
Postoperative Troponin (ng/mL)	0.04 (0.02,0.13)	34.93 (3.95,50)	1.23 (1.11;1.36)	**<0.001**	0.06 (0.02,0.36)	3.46 (1.02,49.68)	1.19 (0.98;1.45)	**<0.001**
Postoperative BNP (pg/mL)	89.10 (35,213.15)	619.40 (236.78,2727.90)	1.00 (1.00;1.00)	**<0.001**	180.30 (95.12,329.95)	315.10 (199.22,701.90)	1.00 (1.00;1.00)	**0.036**
Postoperative White Blood Cells ($\times10^9$/L)	7.64 (6.50,9)	12.25 (8.78,17.95)	1.61 (1.37;1.88)	**<0.001**	7.35 (6.25,8.85)	9.85 (7.90,14.40)	1.51 (1.19;1.90)	**0.001**
Postoperative Red Blood Cells ($\times10^{12}$/L)	4.22±0.71	4.23±0.60	1.03 (0.62;1.69)	0.910	4.22 (3.81,4.70)	4.25 (3.82,4.48)	0.66 (0.28;1.56)	0.528
Postoperative Hemoglobin (g/L)	126.49±19.82	128.84±17.68	1.01 (0.99;1.02)	0.461	129.31 (19.34)	129.69 (15.12)	1.00 (0.97;1.03)	0.934
Postoperative Red Cell Distribution Width (fL)	43.30 (41.45,45.05)	44.05 (42.20,45.85)	1.01 (0.99;1.02)	**0.045**	44.00 (42.18,46.00)	44.05 (43.15,45.12)	1.07 (0.93;1.22)	0.732
Postoperative Platelets ($\times10^9$/L)	192 (163,224)	177 (152,229.25)	1.00 (1.00;1.01)	0.601	193.48±60.77	208.50±79.88	1.00 (1.00;1.01)	0.490
Postoperative NLR	3.68 (2.51,4.89)	7.66 (4.69,13.13)	1.25 (1.16;1.36)	**<0.001**	3.95 (2.52,4.85)	4.37 (3.75,9.01)	1.11 (0.99;1.24)	0.062
Hospitalization Duration(days)	4(3,5)	9(6,12)	1.47 (1.30;1.67)	**<0.001**	4(3,5)	7(6,10)	1.47 (1.16;1.86)	**<0.001**
MACEs (n, %)	34 (15.8)	29 (76.3)	17.2 (7.46;39.45)	**<0.001**	20(31.3)	8(50)	2.2(0.723;6.70)	0.160

Note: Bold indicates statistical significance.

Abbreviations: BMI, Body mass index; BNP, B-type natriuretic peptide; eGFR, Estimated glomerular filtration rate; MACEs, Major adverse cardiovascular events; NLR, Neutrophil-lymphocyte ratio.

Table 3 Comparison of Characteristics Between MACEs and No-MACEs Groups

Variable	no-MACEs (n = 52)	MACEs (n = 28)	*P*
Age (years)	75 (68.75, 81)	71.5 (60.5, 77)	0.171
Gender (n, %)			1[a]
Male	48 (92.31)	26 (92.86)	
Female	4 (7.69)	2 (7.14)	
BMI (kg/m^2)	23.19 ± 3.43	24.72 ± 4.14	0.101
Hypertension (n, %)	30 (57.69)	15 (53.57)	0.906
Diabetes (n, %)	18 (34.62)	12 (42.86)	0.628
Smoking (n, %)	10 (19.23)	8 (28.57)	0.501
IABP performed (n, %)	8 (15.38)	8 (28.57)	0.266
Systolic Blood Pressure (mmHg)	123.63 ± 16.16	118.46 ± 20.53	0.255
Diastolic Blood Pressure (mmHg)	71 (65, 77)	70.5 (64.75, 85)	0.355
Left Ventricular Ejection Function (%)	52.98 ± 10.85	36.57 ± 10.23	**<0.001**
Uric Acid (umol/l)	471.6 ± 114.37	507.81 ± 125.3	0.21
Preoperative Serum Creatinine (umol/l)	115 (103, 132.55)	121.45 (104.8, 139.65)	0.672
Preoperative Blood Urea Nitrogen (mmol/l)	7.02 (5.67, 8.86)	7.97 (6.82, 9.64)	0.11
Preoperative eGFR	50.16 ± 12.09	50.52 ± 14.96	0.913
Creatine Kinase Isoenzyme MB (U/L)	16.15 (12.55, 21.3)	19.95 (17.2, 24.45)	**0.044**
Preoperative Troponin (ng/mL)	0.03 (0.01, 2.52)	0.18 (0.05, 0.74)	0.067
Preoperative BNP (n, %)			**0.01**
<100pg/mL	19 (36.54)	2 (7.14)	
>100pg/mL	33 (63.46)	26 (92.86)	
Preoperative White Blood Cells ($\times 10^9/L$)	6.95 (5.8, 8.33)	8.45 (6.62, 9.62)	**0.025**
Preoperative Red Blood Cells ($\times 10^{12}/L$)	4.31 ± 0.61	4.53 ± 0.83	0.212
Preoperative Hemoglobin (g/L)	129.71 ± 18.11	135.32 ± 17.71	0.185
Preoperative Red Cell Distribution Width (fL)	44.05 (43, 46)	44.95 (41.72, 46.5)	0.98
Preoperative Platelets ($\times 10^9/L$)	206.83 ± 67.5	199.61 ± 57.67	0.617
Preoperative NLR	3.5 (2.48, 4.34)	4.67 (3.02, 8.24)	**0.007**
Triglycerides (mmol/l)	1.33 (1.05, 2.13)	1.23 (0.88, 1.87)	0.471
Cholesterol (mmol/l)	4.05 ± 1.08	4.64 ± 0.92	**0.012**
Low-Density Lipoprotein (mmol/l)	2.51 ± 0.77	2.89 ± 0.74	**0.034**
Serum Albumin (g/L)	38.04 ± 3.74	35.73 ± 3.71	**0.01**
Serum Globulin (g/L)	27.59 ± 3.9	27.39 ± 5.08	0.861
Serum Prealbumin (mg/L)	229.28 ± 69.73	188.88 ± 46.92	**0.003**

(*Continued*)

Table 3 (Continued).

Variable	no-MACEs (n = 52)	MACEs (n = 28)	*P*
Aspartate Aminotransferase (U/L)	22 (17.75, 29.25)	27 (21.1, 45.75)	0.053
Alanine Aminotransferase (U/L)	19.5 (14, 28)	24 (17.75, 35.25)	0.122
Number of Coronary Artery Lesions (n, %)			0.73[a]
1	3 (5.77)	2 (7.14)	
2	7 (13.46)	2 (7.14)	
3	42 (80.77)	24 (85.71)	
Main Coronary Artery Lesion (n, %)	16 (30.77)	8 (28.57)	1
Number of Coronary Stents Implanted	2 (1, 2)	2 (1, 2)	0.812
Surgical Time (min)	60 (46.5, 77.75)	60.5 (40.5, 86.75)	0.816
Contrast Volume (mL)	60 (60, 80)	80 (60, 80)	0.093
Postoperative Serum Creatinine (umol/l)	109.5 (99.7, 137.02)	123.15 (112, 144.82)	0.054
Postoperative Blood Urea Nitrogen (mmol/l)	6.53 (4.87, 8.68)	8.38 (7.19, 11.91)	**0.001**
Postoperative eGFR	52.57 ± 15.07	47.81 ± 15.27	0.187
Postoperative Troponin (ng/mL)	0.06 (0.02, 0.49)	0.36 (0.09, 1.34)	**0.02**
Postoperative BNP (n, %)			**0.007**
<100pg/mL	17 (32.69)	1 (3.57)	
>100pg/mL	35 (67.31)	27 (96.43)	
Postoperative White Blood Cells ($\times 10^9$/L)	7.5 (6.4, 8.85)	7.8 (6.5, 10.93)	0.248
Postoperative Red Blood Cells ($\times 10^{12}$/L)	4.36 ± 0.72	4.65 ± 0.75	0.102
Postoperative Hemoglobin (g/L)	138.44 ± 23.18	140.07 ± 14.97	0.705
Postoperative Red Cell Distribution Width (fL)	43.95 (42.75, 46)	44.3 (42.15, 45.45)	0.968
Postoperative Platelets ($\times 10^9$/L)	192.4 ± 64.79	204.07 ± 65.17	0.447
Postoperative NLR	3.92 (2.52, 5.08)	4.25 (3.69, 5.3)	0.228

Note: a is Fisher's exact test; Bold indicates statistical significance.
Abbreviations: BMI, Body mass index; BNP, B-type natriuretic peptide; eGFR, Estimated glomerular filtration rate; IABP, Intra-aortic balloon pump; NLR, Neutrophil-lymphocyte ratio.

excellent discriminative ability (AUC > 0.84), calibration (Hosmer-Lemeshow test, $P > 0.7$), and clinical utility (DCA confirmed net benefit). To our knowledge, this is the first study to evaluate the clinical value of IABP specifically in a PCI population with renal dysfunction, and establish a validated MACEs risk prediction tool tailored for this high-risk subgroup.

Patients with renal dysfunction represent a high-risk population for cardiovascular diseases.[23] These individuals typically exhibit diffuse atherosclerosis with significant calcification, increased plaque vulnerability, and more severe microcirculatory dysfunction.[24] Given this distinct pathophysiological profile, revascularization plays a crucial role in improving clinical outcomes. Current evidence suggests that, compared with medical therapy alone, revascularization reduces myocardial infarction incidence and shows a trend toward lower all-cause mortality in this population.[5] Regarding revascularization strategies, PCI demonstrates advantages over coronary artery bypass grafting by offering

Table 4 Analysis Results of the Variables Screened by LASSO Regression in the Multivariate Logistic Regression

Considerations	β	SE	Waldχ^2	OR (95% CI)	P
(Intercept)	−1.546	1.629	0.901	0.213(0.005~4.087)	0.343
preBNP	0.447	2.007	0.049	1.563(0.044~125.868)	0.824
postBNP	0.788	1.992	0.157	2.2(0.043~142.077)	0.692
preNLR	0.187	0.155	1.451	1.205(0.931~1.727)	0.228
postBUN	0.194	0.12	2.611	1.214(0.971~1.572)	0.106
PA	−0.013	0.006	4.863	0.987(0.974~0.998)	0.027

Abbreviations: preBNP, preoperative B-type natriuretic peptide; postBNP, postoperative B-type natriuretic peptide; preNLR, preoperative neutrophil-lymphocyte ratio; postBUN, postoperative blood urea nitrogen; PA, serum prealbumin.

lower early mortality and reduced stroke risk.[25] In clinical practice, pMCS is frequently employed to facilitate high-risk PCI in these complex cases.[8]

As the most commonly used pMCS device, the impact of IABP on renal function remains controversial. Our study employed propensity score matching to minimize confounding factors and revealed no statistically significant differences were observed in serum creatinine levels or eGFR between the IABP and non-IABP groups. However, propensity-adjusted analysis revealed clinically divergent trajectories: the IABP group exhibited a mean eGFR decline of −3.06 mL/min/1.73m² (preoperative 51.78 ± 13.2 → postoperative 48.72 ± 15.03), while the non-IABP group showed a +1.56 mL/min/1.73m² improvement (49.89 ± 13.11 → 51.45 ± 15.33). This trajectory holds clinical relevance for renal outcomes because the magnitude of decline in IABP patients (−3.06 mL/min/1.73m²) may relate to increased complication morbidity, acute exacerbation of chronic kidney disease, and increased mortality.[26] This suggests that IABP may transiently increase renal burden and accelerate renal decline in vulnerable subgroups, aligning with findings from Miller et al[14] and Lansky et al.[15] Potential mechanisms include hemolysis-induced hemoglobinuria nephropathy,[27,28] systemic inflammation,[29,30] arterial embolization,[31,32] and compromised renal perfusion due to malposition or oversized balloons.[33] However, the 2012 IABP-SHOCK II trial reported no significant renal impairment with IABP use,[16] and Feng Yang et al found no substantial increase in renal risk.[34] Notably, these conflicting studies did not specifically analyze renal dysfunction subgroups. By controlling for confounders through PSM, our study provides novel evidence for this special population.

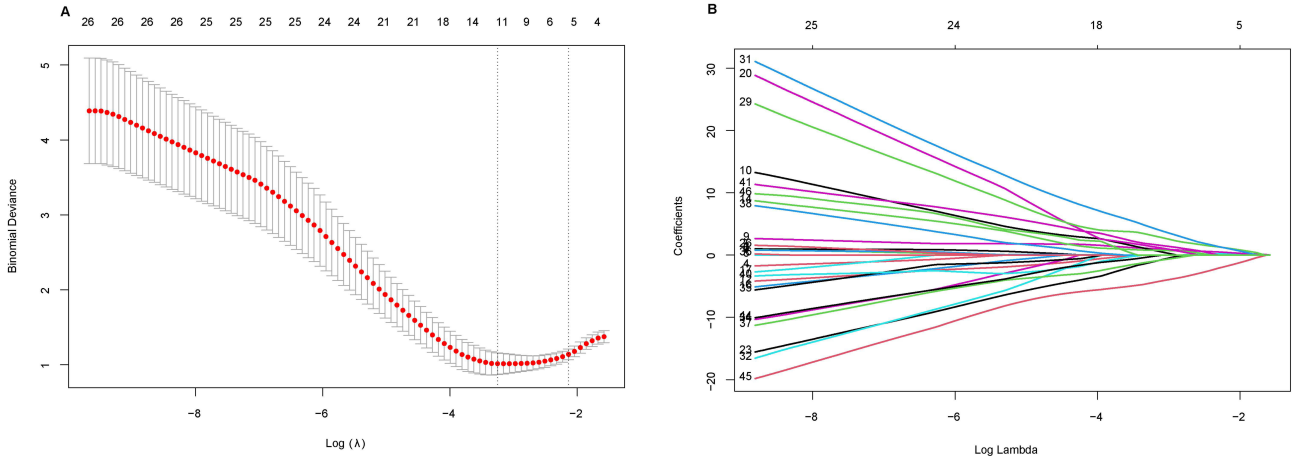

Figure 1 Feature selection using LASSO analysis. Lambda (tuning parameter) selection of deviance in the LASSO regression based on the one standard error criterion (right dotted line) and the minimum criterion (left dotted line) (**A**). LASSO coefficient profiles of the candidate features. The intersecting curves represent the number of features retained at that log (lambda) value, and five predictors with nonzero coefficients were selected according to one standard error criterion (**B**).

https://doi.org/10.2147/IJGM.S532170

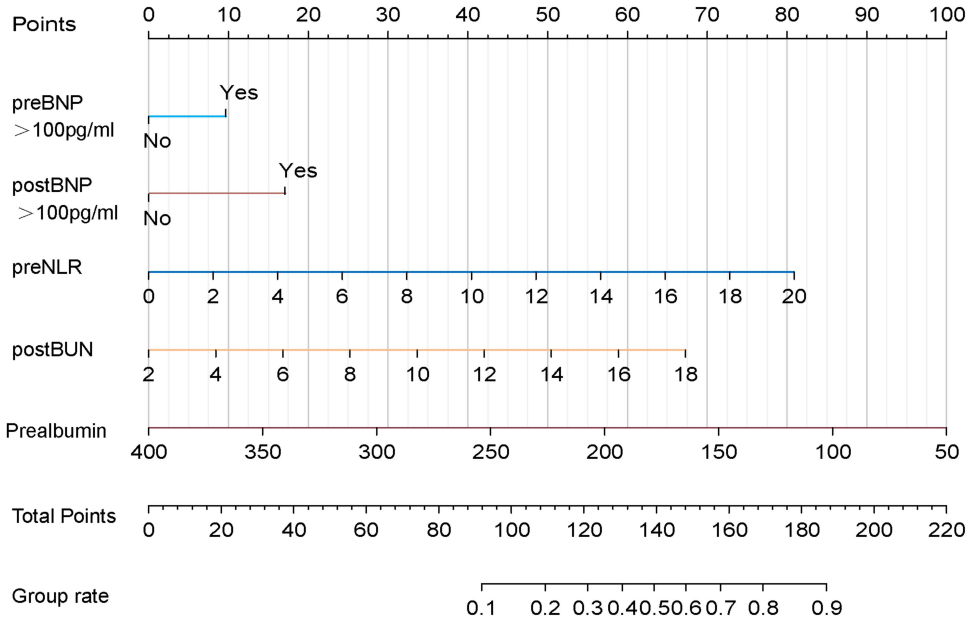

Figure 2 Nomogram for predicting the risk of MACEs in renal dysfunction patients undergoing PCI.

Although this study did not demonstrate a significant reduction in MACEs with IABP use, it is the first to integrate multidimensional indicators to construct a predictive nomogram for MACEs in renal dysfunction patients undergoing PCI. After PSM, multivariate analysis identified the following independent risk factors for MACEs: elevated preoperative and postoperative BNP, increased preoperative NLR, elevated postoperative BUN, and reduced serum prealbumin.

BNP, a hormone released in response to ventricular wall stress, reflects persistent hemodynamic overload. Elevated preoperative and postoperative BNP levels indicate sustained ventricular strain, reinforcing the concept of a deleterious cardio-renal feedback loop. Prior studies have consistently demonstrated that elevated preoperative BNP predicts adverse outcomes following cardiac surgery,[35] while perioperative BNP elevation correlates with postoperative complications and poor prognosis.[36,37] Our findings align with this evidence, supporting BNP's prognostic value in high-risk PCI patients with renal dysfunction. The NLR is a well-established marker of systemic inflammation. Elevated NLR has been associated with worse outcomes in various coronary syndromes,[38] including increased mortality and MACEs in patients undergoing coronary interventions.[39,40] A recent large-scale study confirmed that NLR remains an independent predictor of MACEs in PCI patients after multivariable adjustment,[41] further validating its inclusion in our predictive model. BUN not only reflects renal function but also serves as an indirect marker of neurohormonal activation and systemic perfusion status.[42] Since urea reabsorption in the renal tubules is a passive process linked to sodium and water retention, elevated BUN may indicate reduced cardiac output or intravascular depletion rather than intrinsic renal injury.[43] In acute myocardial infarction complicated by cardiogenic shock, elevated BUN is strongly associated with short-term mortality and MACEs.[44] Our study suggests that postoperative BUN elevation may reflect intraprocedural hemodynamic perturbations, contributing to subsequent adverse events. Serum prealbumin, a negative acute-phase reactant, is suppressed in inflammatory states due to cytokine-mediated inhibition of hepatic synthesis.[45] Low prealbumin levels have been consistently linked to poor prognosis in various cardiovascular conditions. Akashi et al demonstrated that reduced prealbumin at admission predicts long-term adverse outcomes in acute heart failure,[46] while Yu et al found that preoperative prealbumin ≤20 mg/dL increases postoperative infection risk and prolongs mechanical ventilation in cardiac surgery patients.[47] Our study extends these observations to the PCI setting, where lower prealbumin levels correlate with higher MACEs risk, likely reflecting underlying malnutrition and chronic inflammation.

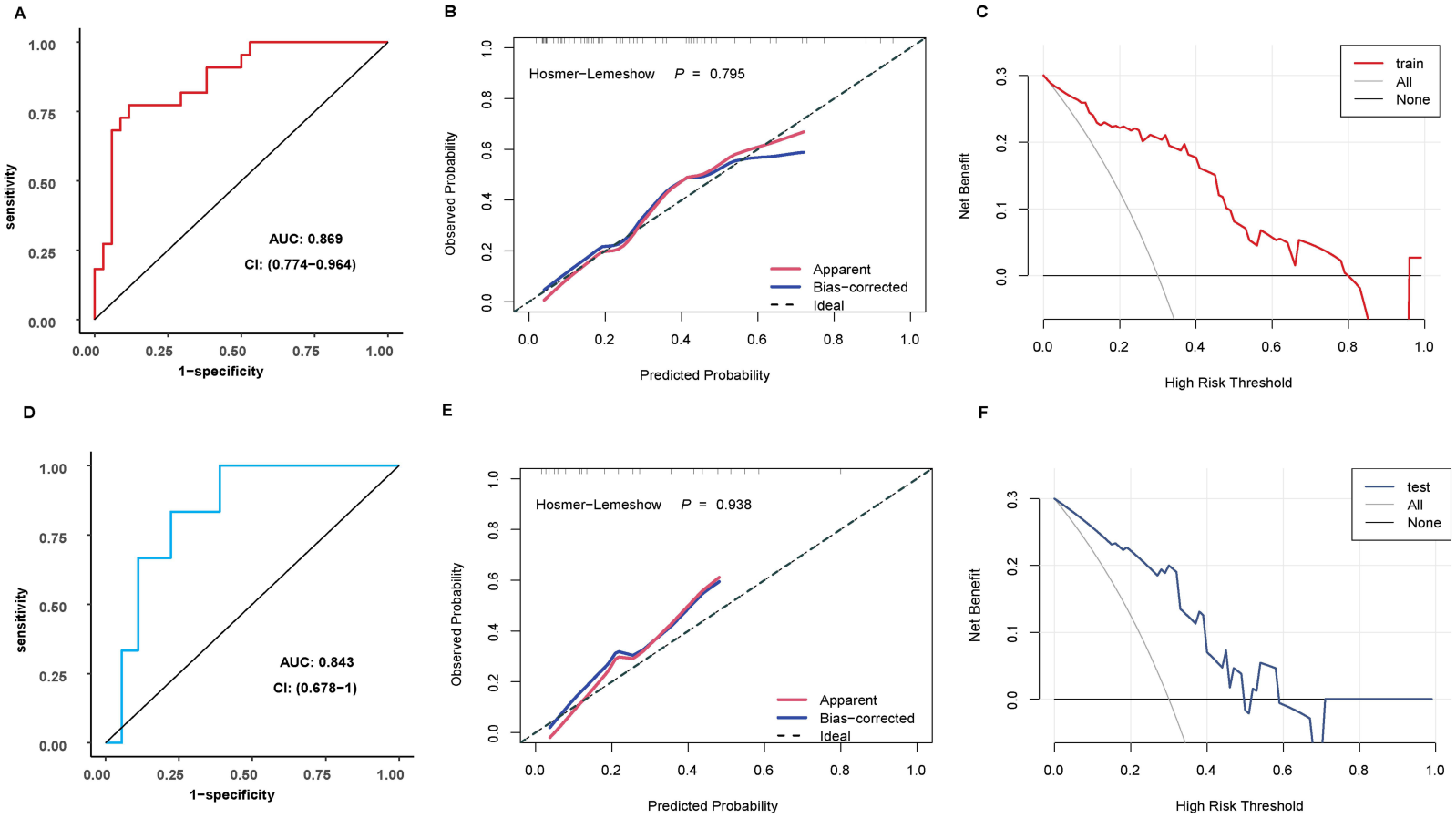

Figure 3 Evaluation of the nomogram. The ROC curve (**A**), the calibration plot (**B**), and the decision curve analysis (**C**) of the nomogram in the training set. The ROC curve (**D**), the calibration plot (**E**), and the decision curve analysis (**F**) of the nomogram in the validation set.

https://doi.org/10.2147/IJGM.S532170

Limitations

This study has several limitations that warrant consideration. First, the study design was a single-center retrospective analysis. Although PSM was employed to minimize selection bias, residual confounding factors may still influence the results. Second, the modest cohort size—reflecting the inherently low prevalence of renal dysfunction PCI patients receiving IABP therapy—limits statistical precision and generalizability. Nevertheless, this enhances the study's real-world clinical validity by capturing authentic decision-making scenarios for this critical subgroup. Third, the prediction model was developed and validated using data from a single center, which may limit its external validity. Future multicenter studies with large populations should address these limitations to enhance generalizability beyond single-center settings.

Conclusion

After balancing covariates via PSM, our analysis suggests that IABP use during PCI in renal dysfunction patients may contribute to subclinical renal impairment without significantly reducing MACEs incidence. These findings underscore the need for clinicians to carefully weigh the hemodynamic benefits of IABP against its potential renal risks, particularly in patients with preexisting renal insufficiency. However, these conclusions should be interpreted with caution given the study's limitations: a single-center design with modest sample size and low MACEs rates, which may affect statistical power and generalizability. And to enhance risk stratification, we developed a clinically practical nomogram integrating key predictors: pre-/post-operative BNP, preoperative NLR, postoperative BUN, and serum prealbumin. This model demonstrates excellent predictive accuracy, providing clinicians a tool for early identification of high-risk patients and guidance of personalized treatment strategies. We explicitly call for external validation in multicenter cohorts to verify nomogram robustness.

Data Sharing Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This retrospective study was approved by the Institutional Review Board of the Ethics Committee of Medicine, Meizhou People's Hospital (Approval Number: 2022-C-109). Given the retrospective nature of the study and the use of de-identified patient data, the requirement for informed consent was waived by the Institutional Review Board. The study was conducted in accordance with the ethical standards of the Declaration of Helsinki and its subsequent amendments.

Acknowledgments

The authors would like to thank other colleagues who were not listed in the authorship of Center for Cardiovascular Diseases, Meizhou People's Hospital, for their valuable comments on the manuscript.

Author Contributions

All authors made a significant contribution to the work reported, including conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the manuscript; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by the Medical and Health Program of Meizhou (Grant No. 2023-B-3).

Disclosure

The authors declare that they have no competing interests in this work.

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