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Contrast-induced nephropathy after endovascular interventions in peripheral artery disease: Predictive value of the Mehran score

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ABSTRACT

Objectives: This study aims to evaluate the predictive value of the Mehran score for contrast-induced nephropathy (CIN) following peripheral transcatheter angioplasty (PTA) in patients with peripheral artery disease (PAD).

Patients and methods: Between January 2020 and July 2024, a total of 103 patients (74 males, 29 females; mean age: 62.6±10.1 years; range, 44 to 75 years) who underwent PTA were retrospectively analyzed. The Mehran score was calculated for all patients meeting inclusion criteria. Contrast-induced nephropathy was defined as an absolute increase in serum creatinine ≥0.5 mg/dL or a relative increase ≥25% within 48 to 72 h post-procedure. The predictive value of the Mehran score was calculated using the receiver operating characteristic (ROC) curve analysis. Multivariate logistic regression identified independent risk factors for CIN.

Results: Contrast-induced nephropathy occurred in 19 patients (18.4%). Patients who developed CIN received significantly higher contrast volumes ($p<0.05$). The optimal Mehran score cut-off for predicting CIN was 11.2, with an area under the curve (AUC) of 0.712 (95% confidence interval [CI]: 0.612-0.826; sensitivity 69%, specificity 73%). Independent predictors of CIN included advanced age, diabetes mellitus, higher contrast volume, baseline glomerular filtration rate, and the Mehran score. Patients with CIN had longer hospital stays (3.9±1.7 *vs.* 1.9±0.7 days, $p=0.002$) and higher amputation rates (10.6% *vs.* 2.9%, $p=0.004$).

Conclusion: The Mehran score is a valuable tool for predicting CIN after PTA in PAD patients. Risk stratification using the Mehran score may guide preventive strategies, although PAD-specific models incorporating additional parameters are warranted.

Keywords: Contrast-induced nephropathy, Mehran score, percutaneous transcatheter angioplasty, peripheral artery disease.

Peripheral artery disease (PAD) is a prevalent vascular disorder associated with increased morbidity and mortality, with higher prevalence in older populations.[1,2] Untreated PAD carries a high risk of complications. Endovascular interventions, such as peripheral transcatheter angioplasty (PTA), have become preferred treatment options due to lower complication rates compared with medical therapy or surgical revascularization.[3] However, contrast media used during these procedures may precipitate contrast-induced nephropathy (CIN), defined as an increase in serum creatinine ≥0.5 mg/dL or ≥25% within 48 to 72 h post-procedure. Contrast-induced nephropathy occurs more frequently in elderly or comorbid patients.[4-7]

Risk factors for CIN include advanced age, pre-existing renal impairment, diabetes mellitus (DM), anemia, heart failure, hemodynamic instability, and high contrast volume. The Mehran score, developed to predict post-coronary intervention CIN, incorporates both clinical and procedural parameters, including hypotension, intra-aortic balloon pump requirement, congestive heart failure, chronic kidney disease, diabetes, age >75 years, anemia, and contrast volume.[7,8]

In the literature, the predictive value of the Mehran score in PAD patients undergoing PTA has not been extensively evaluated. In the present study, we, therefore, aimed to assess the utility of the Mehran score for predicting CIN after PTA in this patient population.

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PATIENTS AND METHODS

This single-center, retrospective, cohort study was conducted at Bakırçay University Çiğli Training and Research Hospital, Department of Cardiology between January 2020 and July 2024. Initially, patients ≥18 years old who underwent PTA for PAD were screened. Only patients who had documented serum creatinine within seven days prior to PTA and at least one post-procedure creatinine measurement within 48 to 72 h were included. End-stage renal disease on dialysis, acute kidney injury (AKI) at baseline, regular use of nephrotoxic medications such as non-steroidal anti-inflammatory drugs (NSAIDs) or incomplete clinical/laboratory data were excluded from the study. Finally, a total of 103 patients (74 males, 29 females; mean age: 62.6±10.1 years; range, 44 to 75 years) were included. Demographic, clinical, and procedural data, including age, sex, body mass index (BMI), comorbidities such as DM, hypertension, congestive heart failure, anemia, chronic kidney disease, procedure type, contrast type and volume, procedure duration, and hemodynamic parameters were obtained from electronic medical records. Written informed consent was obtained from each patient. The study protocol was approved by the İzmir Bakircay University Non-Interventional Clinical Research Ethics Committee (Date: 07.11.2024, No: 1839). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Mehran score calculation, definitions, and outcome measures

The Mehran score was calculated using pre- and intra-procedural clinical and procedural variables. The patients were categorized into low (0-5),

Table 1
Baseline characteristics of the study population

	CIN (n=19)			NKF (n=84)			
	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			64.5 ±9.9			62.2 ±10.7	0.568
Sex							
Male	13	68.4		61	72.6		0.212
Systolic BP (mmHg)			128.8 ± 17.4			129.6 ±18.3	0.656
Diastolic BP (mmHg)			74.7 ±13.5			81.4 ±19.9	0.046*
Diabetes mellitus (%)	14	73.6		43	51.1		0.012*
Hypertension (%)	13	68.4		41	48.8		0.008*
CAD (%)	9	47.3		27	32.1		0.032*
CKD (%)	5	26.3		10	11.9		0.062
Iliac-femoral (%)	2	10.5		11	13.1		0.468
SFA (%)	11	57.8		53	63.1		0.282
BTK (%)	5	26.3		20	23.8		0.404
LVEF (%)			52.5 ± 8.8			56.2 ±9.2	0.108
Creatinine (baseline) (mg/dL)			1.29±0.28			1.12±0.24	0.044*
GFR (mg/dL/1.73 m^2)			57 ± 10.2			68 ± 13.1	0.002*
Mehran score			11.5±3.5			9.8 ±2.8	0.001*
Hemoglobin (mg/dL)			11.7 ±2.7			12.6±2.4	0.033*
Contrast volume (mL)			204 ±48			174±33	<0.001*
In-hospital stay (days)			3.9±1.7			1.8±0.9	<0.001*
Mortality (1 year)	2	10.5		4	4.7		0.256
BARC >2 bleeding (%)	2	10.5		6	7.1		0.542

CIN: Contrast-induced nephropathy; NKF: Normal kidney function; SD: Standard deviation; BP: Blood pressure; CAD: Coronary artery disease; CKD: Chronic kidney disease; SFA: Superficial femoral artery; BTK: Below-the-knee; LVEF: Left ventricular ejection fraction; GFR: Glomerular filtration rate; BARC: Bleeding Academic Research Consortium.

Table 2
Univariate and multivariate analysis of contrast induced nephropathy predictors

	Univariate analysis			Multivariate analysis		
	Odds ratio	95% CI	*p*	Odds ratio	95% CI	*p*
Age	1.2	0.4-1.6	0.042	NS	NS	NS
Contrast volume	3.3	1.6-6-1	<0.001	2.2	1.2-3.5	<0.001*
Glomerular filtration rate	4.2	2.0-6-9	<0.001	2.5	1.7-3.6	<0.001*
Baseline creatinine	2.1	1.1-3.8	0.004	1.5	0.9-2.9	0.030*
Mehran score	2.7	1.4-4.9	<0.001	1.8	1.1-3.2	0.007*
Diabetes mellitus	1.8	0.9-3.3	0.014	1.2	0.6-2.2	0.049*
Anemia	1.9	0.7-3.7	0.008	1.2	0.8-2.0	0.041*
LVEF	1.1	0.3-1.8	0.048	NS	NS	NS

CI: Confidence interval; LVEF: Left ventricular ejection fraction.

moderate (6-10), high (11-15), and very high (≥16) risk groups.

Contrast-induced nephropathy was defined as an increase in serum creatinine ≥0.5 mg/dL or ≥25% within 48 to 72 h post-PTA. For sensitivity analysis, the Kidney Disease: Improving Global Outcomes-Acute Kidney Injury (KDIGO-AKI) criteria were also applied.[7]

The primary outcome measure was CIN development. Secondary outcome measures included hospital length of stay, acute dialysis requirement, and 30-day and one-year all-cause mortality.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 26.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed in as mean ± standard deviation (SD) or median and interquartile range (IQR), while categorical variables were expressed in number and frequency. Continuous data were compared using the Student t-test or Mann-Whitney U test, while categorical data were analyzed using the chi-square test. The receiver operating characteristic (ROC) analysis determined the predictive value of the Mehran score for CIN. Variables with a *p* value of <0.10 in the univariate analysis or clinical relevance were included in the multivariate logistic regression analysis to identify independent CIN predictors. A *p* value of <0.05 was considered statistically significant with 95% confidence interval (CI).

RESULTS

Comorbidities included smoking (74.7%), DM (55.3%), hypertension (52.4%), and chronic kidney disease (14.6%). The incidence of CIN was 18.4% (n=19), with a mean Mehran score of 8.9±2.1 (Table 1). The mean procedural duration was 41.3±17.7 min, the mean contrast volume was 141.7±41.3 mL, and the mean length of hospital stay was 2.1±0.8 days.

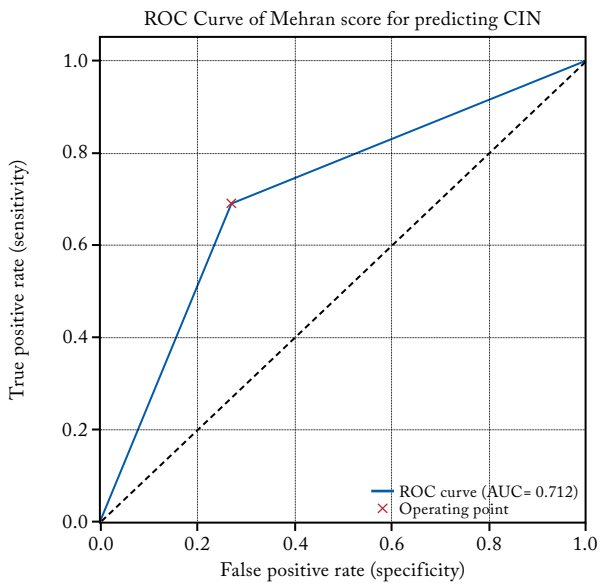

Figure 1. The ROC curve of the Mehran score predicting the development of contrast-induced nephropathy.

ROC: Receiver operating characteristic; CIN: Contrast-induced nephropathy; AUC: Area under the curve; AUC: 0.712, sensitivity 69%; specificity 73%.

Patients who developed CIN had longer hospital stays (3.9±1.7 *vs.* 1.9±0.7 days, p=0.002) and higher amputation rates (10.6% *vs.* 2.9%, p=0.004). Baseline glomerular filtration rate (GFR) was significantly lower in the CIN group (p=0.026). The ROC analysis showed an area under the curve (AUC) of 0.712 (95% CI: 0.612 to –0.826; sensitivity 69%, specificity 73%), with an optimal Mehran score cut-off of 11.2 (Figure 1). Multivariate analysis identified anemia, high contrast volume, DM, baseline GFR, baseline creatinine, and Mehran score as independent predictors of CIN (Table 2).

DISCUSSION

In the present study, we assessed the utility of the Mehran score for predicting CIN after PTA in PAD patients. In our cohort, CIN incidence was 18.4%, consistent with previously reported rates of 10 to 30%.[7] The Mehran score demonstrated a significant predictive value, with a cut-off of 11.2 (AUC=0.712), aligning with prior studies in coronary interventions and transcatheter aortic valve implantation (TAVI) procedures.[8-10]

Contrast-induced nephropathy is primarily attributed to contrast-induced renal toxicity and ischemia, with patient-related factors (age, DM, heart failure, and impaired renal function) and procedural factors (contrast volume and hemodynamic instability) contributing to risk. In our study, CIN patients had a higher prevalence of DM, reduced left ventricular ejection fraction (LVEF), and lower baseline renal function. In general, PAD patients have high rates of DM, smoking, and concomitant renal and coronary disease, potentially explaining increased heart failure prevalence and poorer outcomes.[8,10-13]

In the current study, higher contrast volume was associated with CIN, while procedure-related hypotension or blood loss was not significantly associated. Lower hemoglobin levels were observed in the CIN group. Contrast-induced nephropathy still remains a leading cause of hospital-acquired renal failure.[14-16] Post-procedural renal dysfunction has been linked to increased morbidity and mortality, although one-year mortality was not statistically different in our cohort. Amputation rates were higher in the CIN group, likely reflecting higher comorbidity burden.

In this study, the incidence of CIN was 18.4%, and affected patients experienced prolonged hospitalization, higher amputation rates, and worse baseline renal function. The ROC analysis identified a Mehran score cut-off of 11.2 with good predictive accuracy, while multivariate analysis confirmed anemia, high contrast volume, DM, baseline GFR, creatinine, and Mehran score as independent predictors of CIN. These findings are consistent with previous reports highlighting the prognostic impact of modifiable risk factors and validated risk scores in predicting CIN after cardiac and transcatheter interventions.[7,17-19] Early identification of high-risk patients and optimization of preventable risk factors remain crucial to improve outcomes.

Multivariate analysis identified anemia, high contrast volume, DM, baseline GFR, baseline creatinine, and Mehran score as independent predictors of CIN. Consistent with previous studies, Karakişi et al.[20] reported that the incidence of AKI following coronary artery bypass grafting (CABG) was 7.6%, with previous myocardial infarction and postoperative creatinine increase as independent predictors of AKI. Similarly, Yurdam et al.[21] demonstrated that, in patients undergoing PCI for chronic total occlusion, higher contrast volume, elevated blood glucose levels, reduced baseline renal function, and lower LVEF were independent predictors of CIN. These findings underscore the importance of careful pre-procedural risk assessment and optimization of modifiable factors to minimize CIN incidence.

The Mehran score is a validated tool for predicting CIN and future adverse outcomes after percutaneous interventions. In our study, anemia, DM, baseline GFR, baseline creatinine, contrast volume, and Mehran score independently predicted CIN. These findings support its utility in risk stratification for PAD patients undergoing PTA. Preventive strategies, including contrast minimization and pre-procedural intravenous hydration, should be considered in high-risk patients.

The present study is limited by its single-center, retrospective design, small sample size, variability in hydration protocols, heterogeneity in contrast type and volume, and incomplete long-term renal follow-up. Further well-designed studies are needed to establish more reliable conclusions on this subject.

In conclusion, the Mehran score is a valuable tool for predicting CIN in PAD patients undergoing endovascular interventions. It can guide risk stratification and preventive strategies, while PAD-specific risk models incorporating additional parameters are still warranted.

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