Research Article / Araştırma Makalesi

Interarm Blood Pressure Difference as a Predictor of Contrast-Induced Acute Kidney Injury in Patients Undergoing Peripheral Vascular Interventions

Periferik Vasküler Girişim Uygulanan Hastalarda Kontrast Nefropatisinin Bir Prediktörü Olarak Kollar Arası Kan Basıncı Farkı

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Abstract

Background: Contrast-induced acute kidney injury (CI-AKI) can be a serious complication for patients with peripheral artery disease(PAD) undergoing peripheral vascular interventions(PVI). An interarm blood pressure difference (IABPD)≥10 mmHg has been identified as an independent risk factor for cardiovascular disease and mortality. This study aimed to evaluate the predictive value of IABPD for the risk of CI-AKI in PAD patients undergoing PVI.
Materials and Methods: This prospective study included 171 consecutive patients who underwent PVI. IABPD was defined as the difference in systolic blood pressure between the two arms and was considered significant if it was≥10 mmHg. Patients were categorized into two groups based on the occurrence of CI-AKI.
Results: The incidence of CI-AKI after PVI was 21 %. The CI-AKI(+) group had a significantly higher incidence of IABPD >10 mmHg (28% vs. 8%, p<0.001). Multivariable logistic regression analysis identified IABPD (OR:1.135, 95% CI:1.037-1.243, p=0.006) as an independent predictor of CI-AKI. Additionally, hypertension (OR:2.308, 95% CI:1.091-4.885, p=0.03), higher mean blood pressure (OR:1.055, 95% CI 1.001-1.111, p=0.04), lower eGFR (OR 0.963, 95% CI 0.948-0.978, p<0.001), higher CRP levels (OR:1.028, 95% CI:1.006-1.050, p=0.01), and lower LVEF (OR:0.969, 95% CI 0.938-0.998, p=0.04) were significant predictors of CI-AKI. Furthermore, having TASC C-D lesions compared to TASC A-B was associated with a higher risk of CI-AKI (OR:3.304, 95% CI 1.197-9.117, p=0.02).
Conclusions: This study demonstrated that IABPD is significantly associated with the development of CI-AKI in patients undergoing PVI for PAD. Assessing IABPD in patients before PVI could help clinicians identify those at an elevated risk for developing CI-AKI.

Keywords: Contrast-induced acute kidney injury, Interarm blood pressure difference, Peripheral artery disease, Peripheral vascular intervention

Öz

Amaç: Kontrast nefropatisi (KN), periferik arter hastalığı (PAH) nedeniyle periferik vasküler girişim (PVI) uygulanan hastalarda ciddi bir komplikasyon olabilir. İki kol arasındaki kan basıncı farkı (IABPD) ≥10 mmHg, kardiyovasküler hastalık ve mortalite için bağımsız bir risk faktörü olarak tanımlanmıştır. Bu çalışma, PAH nedeniyle PVI uygulanan hastalarda CI-AKI riskini öngörmede IABPD'nin prediktif değerini değerlendirmeyi amaçladı.
Materyal ve Metod: Bu prospektif çalışmaya, PVI uygulanan 171 ardışık hasta dahil edildi. IABPD, iki kol arasındaki sistolik kan basıncı farkı olarak tanımlandı ve ≥10 mmHg olduğu durumlarda anlamlı kabul edildi. Hastalar, KN gelişimine göre iki gruba ayrıldı.
Bulgular: PVI sonrası CI-AKI insidansı %21 olarak bulundu. KN (+) grubunda, IABPD ≥10 mmHg oranı anlamlı derecede daha yüksekti (%28 vs. %8, p<0.001). Multivaryant lojistik regresyon analizi, IABPD'yi (OR: 1.135, %95 CI: 1.037-1.243, p=0.006) KN için bağımsız bir prediktör olarak tanımladı. Ayrıca, hipertansiyon (OR: 2.308, %95 CI: 1.091-4.885, p=0.03), daha yüksek ortalama kan basıncı (OR: 1.055, %95 CI: 1.001-1.111, p=0.04), daha düşük eGFR (OR: 0.963, %95 CI: 0.948-0.978, p<0.001), daha yüksek CRP seviyeleri (OR: 1.028, %95 CI: 1.006-1.050, p=0.01) ve daha düşük LVEF (OR: 0.969, %95 CI: 0.938-0.998, p=0.04) de KN için anlamlı prediktörler olarak bulundu. Ek olarak, TASC C-D lezyonlarına sahip olmak, TASC A-B'ye kıyasla daha yüksek KN riski ile ilişkilendirildi (OR: 3.304, %95 CI: 1.197-9.117, p=0.02).
Sonuç: Bu çalışma, IABPD'nin, PAH nedeniyle PVI uygulanan hastalarda KN gelişimi ile anlamlı bir şekilde ilişkili olduğunu göstermiştir. PVI öncesinde IABPD değerlendirilmesi, KN gelişim riski yüksek olan hastaların belirlenmesine yardımcı olabilir.

Anahtar Kelimeler: Kontrast nefropatisi, İki kol arasındaki kan basıncı farkı, Periferik arter hastalığı, Periferik vasküler girişim

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Introduction

Contrast-induced acute kidney injury (CI-AKI) is one of the more well-known threats following cardiac, vascular, and radiologic procedures that demand the application of contrast agents(1-3). Although CI-AKI is typically transient and resolves within two weeks in most cases, it has been linked to increased risks of adverse events, mortality, and higher hospital costs(4,5). Consequently, CI-AKI is often utilized as a quality outcome measurement following these interventions. During peripheral vascular interventions (PVIs) for peripheral arterial disease(PAD), iodinated contrast media (ICM) is commonly employed to visualize vascular anatomy, assess lesions, and guide endovascular treatments. However, ICM is believed to CI-AKI in approximately 10% of PVI cases, a condition with ties to significant mortality and morbidity(6). Early detection of CI-AKI in patients undergoing PVI is vital for halting its progression and ensuring a preferred outcome.

Interarm blood pressure differences (IABPDs) of less than 10 mmHg are generally considered normal. Typically, an IABPD of 10 mmHg or greater is classified as elevated(7). A meta-analysis of four cross-sectional studies among low-risk populations found a pooled prevalence of 19.6% for IABPDs of 10 mmHg or more(8). Several studies have established that IABPDs are correlated with cardiovascular mortality and morbidity(9,10). Additionally, many studies have demonstrated that increased IABPDs linked to a higher risk of aortic dissection and aneurysms, subclinical atherosclerosis, left ventricular hypertrophy, and asymptomatic intracranial and extracranial arterial stenosis(11-14). Previous research also indicates that the risk of PAD in patients possessing higher IABPD is increased(15). However, despite its quick and easy application at the bedside, this clinical tool is not commonly utilized in modern medical fields. The goal of this research was to analyze the predictive value of IABDP regarding the threat of CI-AKI in PAD patients undergoing PVI.

Materials and Methods

Study population

A total of 208 consecutive patients with suspected PAD who were referred for PVI between October 2024 and January 2025 were initially screened. Sixty-five of the potential participants were excluded from the study due to: active infection (n=11), malignancy (n=6), vasculitis (n=5), severe kidney disease (GFR < 15 mL/min/1.73m²) (n=13), aortic coarctation (n=1), or thoracic aortic dissection (n=1). Ultimately, data from 171 patients were analyzed in this study, which was conducted based upon the doctrines and ethics set out by the Declaration of Helsinki. It also received approval from the institutional review board. Each of the participants provided written informed consent. The data that support the findings of this study are available from the corresponding author upon reasonable request.

Laboratory data and definitions

Patient medical records provided laboratory data having to do with hemoglobin, troponin, creatinine, serum glucose, total cholesterol (Total-C), LDL-C, HDL-C, triglycerides, high-sensitivity C-reactive protein (hsCRP), albumin, and counts of white blood cells, neutrophils, and lymphocytes. To measure serum hsCRP levels, the nephelometric method was implemented using an IMMAGE 800 analyzer (Beckman Coulter, CA, USA). Kidney function was gauged through estimating the glomerular filtration rate (eGFR) determined through application of the Modification of Diet in Kidney Disease (MDRD) equation. This method involves the following formula: eGFR (mL/min/1.73 m2) = 175×(Serum Creatinine) $^{-1.154}$ ×(Age)$^{-0.203}$ × (0.742 if female) × (1.212 if African American). Hypertension (HT) was classified as originating from diastolic blood pressure ≥90 mmHg, systolic blood pressure ≥140 mmHg, or a history with antihypertensive medication. Diabetes mellitus (DM) was identified as fasting serum glucose ≥126 mg/dL, glycated hemoglobin ≥6.5%, or a history of hypoglycemic medication use. Hyperlipidemia was classified by Total-C >200 mg/dL and/or LDL-C >140 mg/dL. Qualifications for being categorized as a cigarette smokers were a history of smoking at least ten cigarettes per day for a year or more without making any attempt to break the habit. Patients did not receive any preventive treatment such as pre- or intra-procedural intravenous hydration for CI-AKI. Following angiography, all patients received continuous intravenous isotonic saline hydration (0.9% NaCl) at a standard rate (typically 1–1.5 mL/kg/h) for at least 12 hours, as part of the routine post-procedural care. All patients were given a non-ionic iso-osmolar contrast agent.

Measurement of Blood Pressure and Interarm Blood Pressure Difference

IABPD was calculated through the implementation of various blood pressure devices that had been authorized by the European Society of Hypertension (Omron HEM-7001-E; Omron Corp., Tokyo, Japan). The research made use of a standard cuff (12–13 cm x 35 cm) in accordance with the parameters of the European Society of Hypertension, with two additional cuffs available for arms that were either thicker or thinner. Patients were able to rest for five minutes before blood pressure (BP) was taken, and their arms remained parallel to their heart during this time. BP was measured simultaneously in both arms three different times at five-minute intervals, with the average BP for both left and right being calculated from these measurements. The discrepancy in systolic BP between the two arms IABPD was considered significant if it was calculated to be ≥10 mmHg. This cutoff value aligns with the guidelines laid out by The National Institute for Health and Clinical Excellence, with most previous research choosing to abide by them. Consequently, it provided the basis for participant classification (9,10). mean blood pressure (MBP) was estimated

from Systolic Blood Pressure (SBP) and diastolic blood pressure (DBP) as DBP + (SBP–DBP)/3

Diagnosis of Contrast-induced acute kidney injury

Patients were monitored for 72 hours following the angiographic procedure. Serum creatinine levels were measured at baseline and then once daily for three consecutive days to detect the development of CI-AKI. The diagnosis of CI-AKI was established based on criteria requiring either a minimum 25% rise in creatinine or an absolute increase of 0.5 mg/dL within 72 hours following the administration of CM. The patients were split into two separate groupings: those without confirmed CI-AKI (CI-AKI-) and those with confirmed CI-AKI (CI-AKI+).

Peripheral angiographic evaluation

Two interventional cardiologists analyzed the peripheral angiography images, categorizing occlusions of 70% or greater as indicative of severe peripheral vascular disease. The aortoiliac, femoropopliteal, and infrapopliteal arteries were assessed and stratified based on the severity of the conditions identified, following the classification system outlined in the Trans-Atlantic Inter-Society Consensus II (TASC-II) document. Lesion characteristics were determined from the angiographic findings. Treatment for these lesions included procedures such as balloon angioplasty and stent placement. Failing to navigate the guidewire through the occluded lesion during the percutaneous intervention or to achieve distal perfusion after the PVI resulted in a designation of procedural failure.

Statistical analysis

The way by which CI-AKI developed became the basis for categorizing the features of the study population. To test normality, the Kolmogorov-Smirnov test was employed. Continuous research data were conveyed as mean and standard deviation values or median and interquartile range (IQR) according to their normality. Categorical data, on the other hand, were represented by values that were either absolute and percentage-based. Mann–Whitney U test and Independent samples t-test were utilized to compare independent continuous data groups, while Fisher's exact test and Pearson's chi-squared were applied to compare categorical data groups. Univariate and multivariable logistic regression analyses were helpful in determining the independent predictors of the development of CI-AKI. Sensitivity analyses were conducted to ensure the robustness of the findings. Key predictors of CI-AKI identified in the multivariable logistic regression model were re-evaluated by excluding outliers or extreme values in variables such as eGFR and CRP. Odds ratio (OR) was used for Model's coefficients, and 95% was the chosen confidence interval (CI). For each of these analyses, 2-tailed probability (p) values less than .05 were indicative of statistical significance. A post hoc power analysis was conducted based on the proportion of patients with IABPD >10 mmHg in the CI-AKI (+) and (–) groups (28% vs. 8%). Using these values and a total sample size of 171, the calculated power was 99.99% (α=0.05), indicating sufficient statistical power to detect a significant difference. Jamovi and R 4.01 software (Vienna, Austria) was the vehicle by which all statistical analyses were conducted with "Hmisc", "ggplot", "rms" packages.

Results

The study included 171 patients who underwent PVI for PAD. The incidence of CI-AKI was 21% (n=36) (Figure 1). Patients were categorized into two groups: CI-AKI (+) (n=36) and CI-AKI (–) (n=135). Basic demographic and clinical information of these patients can be reviewed in Table 1.Those belonging to the CI-AKI (+) grouping were, on average, older than those that belonged to the CI-AKI (–) grouping (67.2 ± 7.2 years vs. 59.7 ± 10.2 years, p<0.001). HT and chronic heart failure were more prevalent in the CI-AKI (+) group (69% vs. 40%, p<0.001 and 28% vs. 5%, p<0.001, respectively). LVEF was significantly lower in the CI-AKI (+) group compared to the CI-AKI (–) group (55% [35–65] vs. 65% [60–65], p<0.001).

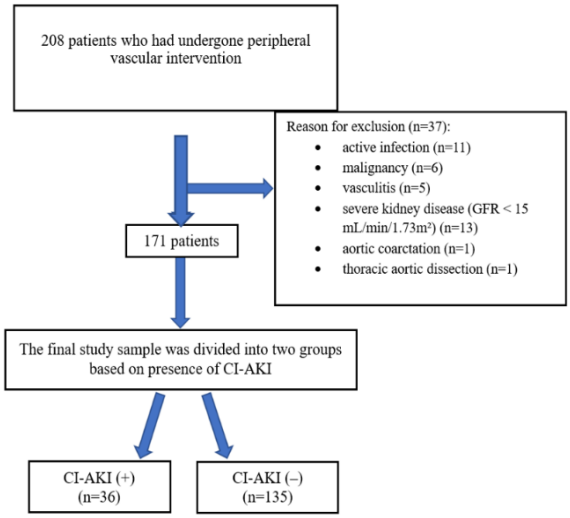

Figure 1. Consort flow diagram for inclusion in the study

The CI-AKI (+) group had a higher prevalence of severe PAD, as indicated by the higher proportion of TASC C-D lesions (89% vs. 63%, p<0.001). The incidence of IABPD >10 mmHg was also significantly higher in the CI-AKI (+) group (28% vs. 8%, p<0.001) (Figures 2, 3).MBP was significantly higher in the CI-AKI (+) group for both the right and left brachial arteries (right brachial: 99.33 [94.66–101.66] mmHg vs. 97.35 [92.78–99.97] mmHg, p=0.009; left brachial: 96.90 [93.25–99.31] mmHg vs. 95.46 [91.20–97.44] mmHg, p=0.004).Patients in the CI-AKI (+) group had a significantly longer hospital stay compared to the CI-AKI (–) group (8 [7–10] days vs. 3 [2–6] days, p<0.001).

Table 1. Demographic and clinical characteristics of study patients based on development CI-AKI

Variables	CI-AKI (+) n= 36 (%21)	CI-AKI (–) n= 135 (%79)	p
Age (years)	67.2±7.2	59.7±10.2	**<0.001**
Gender (male), n (%)	33 (92)	115 (85)	0.083
BMI, kg/m²	25.6±3.6	25.9±3.2	0.584
BSA, m²	1.85±0.1	1.84±0.1	0.690
HT, n (%)	25 (69)	54 (40)	**<0.001**
DM, n (%)	20(55)	60 (44)	0.122
CAD, n (%)	21 (58)	72 (53)	0.357
CHF, n (%)	10 (28)	7 (5)	**<0.001**
Cerebrovascular Disease, n (%)	1 (2)	3 (2)	0.848
Smoking, n (%)	25 (69)	82 (60.7)	0.057
EF (%)	55(35-65)	65(60-65)	**<0.001**
Location of disease, n (%)			
Aortailiac	9 (25)	40 (30)	0.204
Femoro-popliteal	21 (58)	82 (61)	
infra-popliteal	5 (14)	22 (8)	
Balloon size	5 (5-6)	5 (5-6)	0.165
TASC C-D vs A-B	32 (89)	86 (63)	**<0.001**
IABPD>10 mmHg	10 (28)	12 (8)	**<0.001**
Stent usage, n (%)	17 (47)	54 (40)	0.261
RRT, n (%)	3 (8)	0 (0)	**<0.001**
In-hospital mortality, n (%)	2 (6)	0 (0)	**<0.001**
Amputation, n (%)	5 (14)	4 (3)	**<0.001**
Right brachial SBP (mmHg)	134(130-138)	132.2(127.4-137.2)	0.056
Left brachial SBP (mmHg)	126 (122-132)	128.8(124.2-132.6)	0.052
Right brachial DBP (mmHg)	81 (76-85)	79.2(74.2-83.3)	0.054
Left brachial DBP (mmHg)	79.2 (74.2-82.1)	80.8(75.7-83.8)	0.058
Right brachial MBP (mmHg)	99.33 (94.66-101.66)	97.35 (92.78-99.97)	**0.009**
Left brachial MBP (mmHg)	96.90 (93.25-99.31)	95.46 (91.20-97.44)	**0.004**
Length of hospital stay	8 (7-10)	3(2-6)	**<0.001**

BMI: Body Mass Index; BSA: Body Surface Area; CAD: Coronary Artery Disease; CHF: Chronic Heart Failure; CI-AKI: Contrast-induced acute kidney injury; CRP: C-reactive protein; DM: Diabetes Mellitus; EF: Ejection Fraction; Hb: Hemoglobin; HT: Hypertension; IABPD: Interarm Blood Pressure Difference; LDL: Low-Density Lipoprotein; MBP: mean blood pressure RRT: Renal Replacement Therapy; SBP: Systolic Blood Pressure; TASC: Transatlantic Intercommunal Consensus Document

Rates of renal replacement therapy (RRT), in-hospital mortality, and amputation were also significantly higher in the CI-AKI (+) group (8% vs. 0%, p<0.001; 6% vs. 0%, p<0.001; and 14% vs. 3%, p<0.001, respectively).

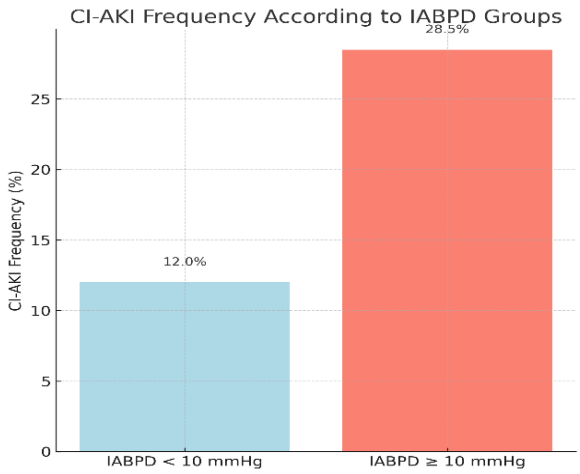

Figure 2. Bar Chart showing the rate of CI-AKI development patients according to the presence of IABPD

Laboratory findings indicated that those within the CI-AKI (+) grouping had significantly lower hemoglobin levels (12.3 [11.4-14.2] vs. 14.2 [12.3-15] , p<0.001), lower eGFR (61.3 ± 27 vs. 94.1 ± 24.8 , p<0.001), higher creatinine levels (1.28 [1-1.56] vs. 0.82 [0.7-0.97], p<0.001), and higher CRP levels (11.1 [4.16-34.6] vs. 3.8 [3.1-11.4], p<0.001) than the CI-AKI (–) grouping (Table 2).

Multivariable logistic regression analysis identified IABPD >10 mmHg (OR 1.135, 95% CI 1.037-1.243, p=0.006) as an independent predictor of CI-AKI. Additionally, hypertension (OR 2.308, 95% CI 1.091-4.885, p=0.029), higher MBP (OR 1.055, 95% CI 1.001-1.111, p=0.045), lower eGFR (OR 0.963, 95% CI 0.948-0.978, p<0.001), higher CRP levels (OR 1.028, 95% CI 1.006-1.050, p=0.014), and lower LVEF (OR 0.969, 95% CI 0.938-0.998, p=0.043) were significant predictors of CI-AKI. Furthermore, having TASC C-D lesions compared to TASC A-B was associated with a higher risk of CI-AKI (OR 3.304, 95% CI 1.197-9.117, p=0.021) (Table 3) ROC curve analysis showed that an IABPD cutoff of >5 mmHg predicted CI-AKI with an AUC of 0.678 (95% CI: 0.613–0.743; p < 0.001), 83.6% sensitivity, and 55.0% specificity (Figure 4).

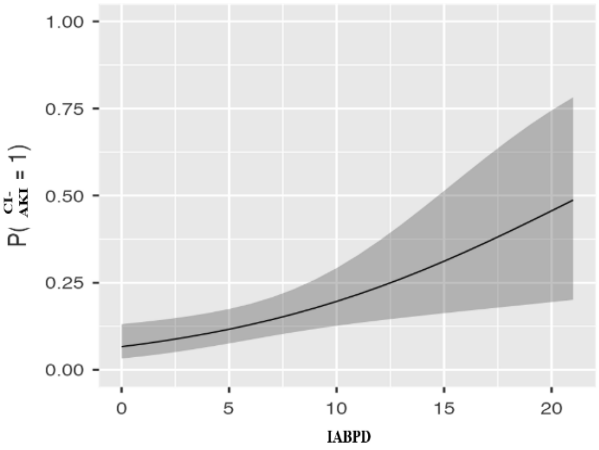

Figure 3. Marginal means plot showing the relationship between the probability of CI-AKI and IABPD

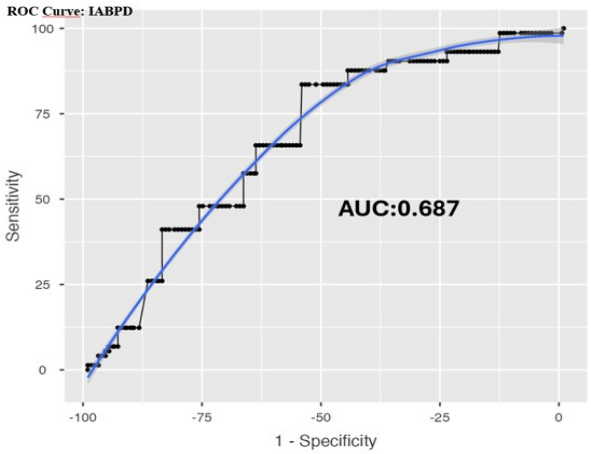

Figure 4. ROC curve analysis showed that an IABPD predicted CI-AKI with an AUC of 0.678 , 83.6% sensitivity, and 55.0% specificity.

Table 2. Laboratory finding comparisons between the groups based on development of CI-AKI.

Variables	CI-AKI (+) n= 36 (%21)	CI-AKI (–) n= 135 (%79)	p
WBC (10^3/µL)	9.2 (7.3-10.7)	8.7 (7.5-10.4)	0.310
Hemoglobin (g/dL)	12.3 (11.4-14.2)	14.2 (12.3-15)	<0.001
AST (IU/L)	20 (17-28.1)	19 (17.5-23.5)	0.427
ALT (IU/L)	17.5 (12.2-27)	16.6 (12-22.6)	0.243
Total Cholesterol (mg/dL)	189.2±43.2	201.5±48.7	0.094
Triglyceride (mg/dL)	142 (114-210)	129 (96-195)	0.274
HDL-C (mg/dL)	40.7±7.8	43±9.5	0.117
LDL-C (mg/dL)	118.3±36.5	126.3±38.9	0.177
eGFR (ml/min/1.73 m^2)	61.3±27.4	94.1±24.8	<0.001
Creatinine (mg/dL)	1.28 (1-1.56)	0.82 (0.7-0.97)	<0.001
Na (mEq/L)	138 (136-140)	138 (135-140)	0.458
K (mmol/L)	4.33±0.6	4.30±0.39	0.503
CRP (mg/L)	11.1 (4.16-34.6)	3.8 (3.1-11.4)	<0.001
Albumin (g/dL)	3.7 (3.3-3.9)	3.9 (3.7-4.3)	<0.001
Glucose (mg/dL)	128 (100-159)	106 (89-166)	0.027

ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; CI-AKI: Contrast-induced acute kidney injury CRP: C reactive protein; eGFR: estimated glomerular filtration rate; HDL-C: high-density lipoprotein cholesterol; K: Potassium; LDL-C: low-density lipoprotein; Na: Sodium; WBC: white blood cell

Table 3. Multivariable analysis for prediction of CI-AKI.

Variables	Multivariable analysis			
	p	OR	95% Confidence Interval (CI)	
			Lower	Upper
Age	0.068	1.044	0.997	1.094
Sex (male)	0.057	4.294	0.956	19.293
Hypertension	0.029	2.308	1.091	4.885
IABPD>10 mmHg	0.006	1.135	1.037	1.243
MBP (mmHg)	0.045	1.055	1.001	1.111
eGFR (ml/min/1.73 m^2)	<0.001	0.963	0.948	0.978
Albumin (g/dL)	0.150	0.554	0.248	1.237
CRP (mg/L)	0.014	1.028	1.006	1.050
Hemoglobin (g/dL)	0.754	1.042	0.804	1.352
EF (%)	0.043	0.969	0.938	0.998
TASC CD vs AB	0.021	3.304	1.197	9.117

CRP: C-reactive protein; CI-AKI: Contrast-induced acute kidney injury EF: ejection fraction; IABPD: Interarm Blood Pressure Difference; MBP: mean blood pressure OR: odds ratio; TASC: Transatlantic Intercommunal Consensus Document

Discussion

This study investigated the relationship between IABPD and CI-AKI in patients undergoing PVI. Our findings demonstrated that IABPD ≥10 mmHg was significantly associated with CI-AKI and remained an independent predictor after adjusting for other known risk factors. Additionally, our study demonstrated LVEF, pre-intervention TASC score, preprocedural eGFR, presence of CKD, HT, MBP and CRP levels can also independently predict CI-AKI following PVI.

CI-AKI is a serious complication of PVIs, with complex and multifactorial pathophysiology. Contributing factors include contrast dose, cholesterol embolization, bleeding-related anemia, and infection (16–18). CI-AKI primarily results from tubular epithelial and endothelial injury, oxidative stress, and impaired renal microcirculation (19). Contrast agents may exacerbate these effects by increasing reactive oxygen species and reducing nitric oxide availability, leading to vasoconstriction (20). Numerous risk factors, such as chronic kidney disease, heart failure, older age, and the use of nephrotoxic agents, have been associated with increased CI-AKI risk (21). Our findings are consistent with this literature: lower baseline eGFR and reduced LVEF were both independently associated with CI-AKI in our cohort (3, 22). Inflammation also affects the pathogenesis of CI-AKI. Accumulating evidence is revealing that oxidative stress and a heightened inflammatory response are significant contributors to the growth and advancement of CI-AKI (23).

Inflammatory cells infiltrating tissue that has been damaged exacerbates renal injury. Multiple inflammatory cytokines, including tumor necrosis factor-α and interleukins-1 and -6 lead to impairment of tubular and endothelial cells, renal medullary hypoxia, and eventually impair renal function(24). Furthermore, inflammatory mediators' secretion induces vasoconstriction of intrarenal vessels, resulting in decreased pressure from glomerular filtration (25). The study also noted that CRP levels, indicating the level of inflammation in patients, were connected to the development of CI-AKI after PVI. Previous studies have also emphasized the role of baseline hydration status in the development of CI-AKI. Notably, inferior vena cava (IVC) diameter has been proposed as a reliable echocardiographic parameter to estimate volume status. Gungoren et al. demonstrated that reduced IVC diameter was associated with an increased risk of CI-AKI in patients undergoing cardiac catheterization (26). Although IVC assessment was not part of our protocol, this parameter could be considered in future studies for more comprehensive risk stratification.

We found that patients with TASC C-D lesions had a significantly higher risk of developing CI-AKI. This may be attributed to the increased complexity of interventions in these patients, which often require longer procedures and higher volumes of contrast media. Moreover, the presence of more advanced and diffuse atherosclerotic disease may exacerbate endothelial dysfunction and inflammation, contributing to renal injury. Previous studies have also shown that TASC C-D lesions are associated with poorer outcomes and greater procedural difficulty (27,28).

MBP, reflecting systemic hemodynamic load, emerged as another independent predictor of CI-AKI in our study. Previous research has shown that MBP plays a key role in maintaining renal perfusion and preventing AKI, particularly in settings involving hemodynamic instability or increased venous congestion (29). Although MBP and IABPD are distinct parameters, both may reflect underlying vascular stiffness, asymmetric atherosclerosis, or impaired autoregulation factors that contribute to renal hypoperfusion and injury. Therefore, combining MBP with IABPD in pre-procedural assessment may offer a more nuanced and practical approach to identifying patients at high risk for CI-AKI in the clinical setting (30).

IABPD is a non-invasive parameter that is easy to measure, though its clinical significance is not yet fully understood. The European Society of Hypertension has long recommended the assessment of IABPD as part of the physical examination for patients with arterial hypertension, suggesting that high IABPD findings ought to initiate a more thorough evaluation in search of vascular abnormalities (31). However, the American College of Cardiology and the American Heart Association do not currently address interarm blood pressure differences in their guidelines (32). If IABPD exceeds the physiological differences due to anatomical variations between the arms, it is counted as an indicator of atherosclerosis. It is also worth noting that a significant association exists between IABPD and the ankle-brachial index (11). Previous research has also indicated that patients with elevated IABPD have an increased risk of developing PAD (15). An IABPD > 10 mmHg is widely recognized as clinically significant. In our study, 44 patients (12.8%) had an IABPD exceeding 10 mmHg, a percentage notably higher than those of other studies(33). This difference may be due to our specific patient population, which was comprised of individuals undergoing PVI for PAD.

IABPD serves not only as an indicator of atherosclerotic diseases, but it also affects prognosis. A cohort study conducted by Clark and colleagues noted that individuals possessing an IABPD of 10 mmHg or greater, or 15 mmHg or greater, were at a greater risk of all-cause mortality over a ten-year period(34). In line with these findings, a meta-analysis composed of nine separate cohort studies showed that the collective hazard ratio for cardiovascular and total mortality saw a jump to 2.98 in individuals with a higher IABPD (31). Durmus et al. examined the relationship between the SYNTAX score and IABPD in their retrospective study, which revealed that those with a high IABPD (≥10 mmHg) had significantly higher SYNTAX scores than those with a low IABPD (<10 mmHg)(35). Additionally Park et al. demonstrated that among patients treated with PCI, the incidence of major adverse cardiovascular events was significantly higher in those with an IABPD of 10 mmHg or greater compared to those with an IABPD of less than 10 mmHg(36). Furthermore, in their recently-published work on 2120 patients with ST-segment elevation myocardial infarction receiving primary percutaneous coronary intervention, Simsek et al demonstrated

that an IABPD ≥10 mmHg serves to independently predict the development of CI-AKI in this patient population (37). Our results support this notion that a strong correlation exists between IABPD and the development of CI-AKI following PVI. Thus, IABPD may serve as a clinically important, simple, quick, and easy-to-measure tool for predicting the risk of CI-AKI in PAD patients undergoing PVI. The underlying pathophysiological process detected by IABPD could be related to increased inflammation, similar to that which was observed in atherosclerosis.

Limitations

There were a few noteworthy limitations in this research. Firstly, as a prospective, single-center, and observational study, there remains a risk of selection bias due to the specialized nature of our center and the patient population it serves. The small sample size no doubt negatively affects the generalizability of the findings. In addition, several studies have shown that the white coat effect could influence higher systolic IABPD. The use of antihypertensive therapy in some patients may have also influenced IABPD levels. Furthermore, our patient population consisted of individuals with peripheral arterial disease, which may explain the higher incidence of IABPD compared to other studies. The delay in detecting serum creatinine changes beyond the 72-hour post-contrast administration window could have led to an underestimation of CI-AKI incidence, particularly in cases where renal function deteriorated after discharge. The volume of CM used during angiography is critical in the genesis of CI-AKI. However, our study lacks data on the amount of CM administered to patients. An additional of our study is the imbalance in baseline renal function between the two groups, with the CI-AKI (+) group having significantly higher baseline serum creatinine levels. Although we excluded patients with severe renal impairment (eGFR <15 mL/min/1.73 m²), higher baseline creatinine levels could still contribute to an increased risk of CI-AKI. We addressed this potential confounder by including eGFR in our multivariable logistic regression analysis and conducting sensitivity analyses. Nevertheless, this factor should be considered when interpreting our results. Moreover, our study did not evaluate baseline hydration status using echocardiographic parameters such as inferior vena cava diameter, which has been shown to predict the development of CI-AKI in prior studies. Lastly, more studies will help elucidate the pathophysiological roles of IABPD.

Conclusion

This study demonstrates that IABPD is significantly linked to the development of CI-AKI in PAD patients undergoing PVI. Predicting the threat of acquiring CI-AKI enables the optimization of procedures by shortening their duration, reducing the volume of CM administered, and implementing preventive measures to avoid CI-AKI. These findings underscore the need for further prospective research to validate the use of IABPD in clinical practice and to explore the underlying mechanisms linking IABPD to adverse renal outcomes.

Ethical Approval: *This study adhered to the principles of the Declaration of Helsinki and received ethical approval from the Kartal Koşuyolu Training and Research Hospital (Approval no: 2024/19/927).*

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