Factors Predictive of Contrast-induced Acute Kidney Injury in the Setting of Coronary No-reflow Phenomenon

Koroner no-reflow fenomeninde kontrast ilişkili akut böbrek hasarını öngördüren faktörler

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ABSTRACT

Introduction: In subjects undergoing percutaneous coronary intervention (PCI), coronary no-reflow phenomenon is generally determined through evaluation of TIMI (Thrombolysis In Myocardial Infarction) score. Contrast-induced acute kidney injury (CI-AKI) has also been a frequent problem in these patients. The purpose of this study is aimed to evaluate potential risk factors of CI-AKI in subjects with coronary no-reflow phenomenon.

Methods: We enrolled consecutive patients with coronary artery disease (CAD) undergoing PCI between the years 2014 and 2019 in cardiology clinic. Independent variables associated with CI-AKI in patients with no reflow phenomenon following PCI were evaluated using multivariate logistic regression analysis. A p value <0.05 was accepted as the statistical significance limit.

Results: Among a population of 3034 patients, 93 (3%) were diagnosed as having coronary no-reflow (64% male, mean age of 64±12 years). CI-AKI occurred in 22% of the patients with coronary no-reflow (n:20). The multivariate analysis has demonstrated significant associations of post PCI TIMI flow 0-1, contrast volume and pulmonary arterial hypertension (PHT) with the evolution of CI-AKI in the setting of no-reflow phenomenon (p:0.023, p:0.017 and p<0.001).

Conclusion: In CAD patients managed with PCI, PHT, contrast volume, post-PCI TIMI flow as well as the presence of coronary ectasia may pose a significant risk for CI-AKI development in subjects with coronary no-reflow phenomenon.

Key words: No-reflow phenomenon**,** coronary artery disease, contrast-induced, acute kidney injury

ÖZET

Giriş: Perkutan koroner girişim (PKG) uygulananlarda, koroner no-reflow fenomeni genellikle TIMI (Miyokard Enfarktüsünde Tromboliz) skorunun değerlendirilmesi ile belirlenir. Kontrast kaynaklı akut böbrek hasarı (CI-AKI) da bu hastalarda sık görülen bir problem olmuştur. Bu çalışmada, koroner no-reflow fenomeni olan hastalarda CI-AKI gelişimi için potansiyel risk faktörlerini değerlendirmeyi amaçladık.

Yöntemler: Kardiyoloji kliniğimize 2014-2019 yılları arasında PKG uygulanan ardışık koroner arter hastalığı (KAH) hastalarını dahil ettik. Çok değişkenli lojistik regresyon analizi ile PKG sonrası no-reflow fenomeni gelişen hastalarda CI-AKI ile ilişkili bağımsız değişkenler değerlendirildi. İstatistiksel anlamlılık sınırı olarak bir P değeri <0.05 kabul edildi.

Bulgular: 3034 hastadan oluşan popülasyondan 93'üne (% 3) koroner no-reflow (% 64 erkek, ortalama yaş 64 ± 12) tanısı kondu. CI-AKI koroner no-reflow saptanan hastaların % 22'sinde görüldü (n = 20). Çok değişkenli analiz, PKG sonrası TIMI akım 0-1, kontrast miktarı ve pulmoner arteriyel hipertansiyonun (PHT) koroner no-reflow fenomeninde CI-AKI' nin gelişimi ile anlamlı ilişkilerini göstermiştir (p:0.023, p:0.017 ve p<0.001).

Sonuç: PKG ile yönetilen KAH hastalarında, PHT, PKG sonrası TIMI akım ve ayrıca mevcut koroner ektazi varlığı, koroner no-reflow fenomeni olan hastalarda LVEF değerlerinden bağımsız olarak CI-AKI gelişimi için önemli bir risk oluşturmaktadır.

Anahtar Kelimeler: No-reflow fenomeni, koroner arter hastalığı, kontrast ilişkili, akut böbrek hasarı.

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INTRODUCTION

Contrast-induced acute kidney injury (CI-AKI) has been considered as a life-threatening complication of percutaneous coronary procedures. Its incidence has been reported to range from 2% to 30% based on different study populations and definitions (1-2). Importantly, CI-AKI development following PCI has been suggested to elicit adverse outcomes including prolonged hospitalization, risk of re-infarction, need for revascularization, progression of renal failure and mortality (3).

The Contrast Media Safety Committee (CMSC) of the European Society of Urogenital Radiology (ESUR) previously defined CI-AKI as a serum creatinine (sCr) absolute increment of ≥0.5 mg/dl (≥ 44 μmol/L) (or a percent increase of ≥25%) from baseline values within 72 hours following intravascular administration of iodine contrast media provided that other potential triggers of AKI are fully excluded (4).

The most notable risk factors for the development of CI-AKI were previously reported to be the presence of certain diseases including chronic renal failure and diabetes mellitus (DM) as well as the amount of contrast agent used (5). Therefore, effective treatment and reduction of these risk factors might serve as the most effective strategy to prevent CI-AKI in this setting. In particular, CI-AKI might mechanistically be due to a variety of factors including, inflammatory process, medullary hypoxia, endothelial dysfunction, impaired renal blood flow as well as direct tubular epithelial damage (6).

On the other hand, coronary no-reflow phenomenon has been one of the most devastating complications of PCI with an overall incidence of 2% to 5% in patients undergoing PCI (7). This phenomenon generally appears to be more prevalent in the presence of high thrombotic burden (as in the settings of acute myocardial infarction (AMI)) and degenerated saphenous vein grafts) (8). In this context, angiographic (TIMI 0-1) flow is generally considered as no-reflow phenomenon in the absence of thrombus, dissection, vasospasm or significant residual stenosis while TIMI-2 flow is generally defined as coronary slow flow and TIMI 3 as normal flow (8).

Even though the exact mechanisms of coronary no-reflow phenomenon currently remain unknown, disruption of the coronary microvascular architecture possibly due to certain factors including endothelial swelling, platelet aggregation, and myocardial edema might account for its evolution (9).

In the present study, we aimed to determine the risk factors for the development of CI-AKI in the setting of coronary no-reflow phenomenon.

METHODS

The present study is a single-center retrospective, cross sectional and observational research (clinical data belong to years between 2014 and 2019) performed in our cardiology clinics. In order not to affect the results due to the COVID-19 pandemic, the records of patients after 2019 were not included. The study was endorsed by the Institutional Ethical Committee. In general, CAD patients (>18 years of age) who had undergone PCI were enrolled in the study. Major valve diseases, cancer patients, pregnant women and re-MI were excluded from the study. Patient data, basic clinical features, procedural characteristics, TIMI grade following PCI, treatment strategies in the hospital setting were then evaluated.

All patients were administered acetylsalicylic acid (ASA)(with loading and maintanence doses of 300 mg and 100 mg/day, respectively) and clopidogrel (with loading and maintanence doses of 300 or 600 mg and 75mg /day, respectively). Unfractionated heparin was injected in a single bolus dose (70 U/kg) and further doses were injected when needed to maintain an activated clotting time of > 250 s. All subjects also

received atorvastatin (with a loading dose of 40–80 mg) in the absence of strict contraindications including severe hepatic dysfunction, history of drug reaction, etc. The decision of further treatment strategies (diuretics, β-blockers and glycoprotein IIb/IIIa inhibitors, etc) was at the discretion of the treating clinician. The creatinine and other laboratory parameters were measured at baseline and 72 hours after PCI.

Water soluble, nonionic, low, or iso-osmolar X-ray contrast medium, iohexol (350 mg iodine/ml, Omnipaque, GE Healthcare, Cork, Ireland), were administered during the coronary procedures. Patients with a high risk for CI-AKI were managed in a standard manner (intravenous hydration of 0.9% NaCl at 0.5 or 1 mL / kg / h for 24 h). N-acetylcysteine was not a part of our institutional protocol largely based on the fact that it was previously demonstrated in a randomized study as ineffective in the prevention of CI-AKI (10). Similarly, sodium bicarbonate (0.84%) infusion and ascorbic acid were not included either.

Coronary flow patterns according to the TIMI classification were evaluated and recorded before and after the coronary procedures In this setting, epicardial coronary flow patterns rather than the myocardial flow were evaluated. Angiographic appearances of different TIMI grades follow as:

TIMI 0: No distal flow

TIMI 1: Distal coronary opacification is hardly visible with no sufficient perfusion.

TIMI 2: Distal coronary wash-out is slower as compared with other coronary arteries.

TIMI 3: The rate of filling and wash-out of the coronary artery is normal.

Statistical Analysis

The normal distribution was assessed using the Shapiro-Wilk test. In the two-group comparisons, Student T test was used for variables that conformed to the normal distribution, and Mann-Whitney U test was used for variables that did not match the normal distribution. In more than two group comparisons, one-way analysis of variance was used for variables that conform to the normal distribution, and Kruskal-Wallis test was used for variables that did not fit the normal distribution. While investigating the relationships between quantitative variables, the coefficient of Pearson correlation was calculated for variables that match the normal distribution, and the coefficient of Spearman correlation was calculated for variables that did not fit the normal distribution. The relationships between qualitative variables were evaluated by Pearson chi-square test.

The risk factors of no-reflow phenomenon were detected using logistic regression analysis. The intraclass correlation coefficient was calculated to determine inter- and intra-observer concordance. The mean and standard deviation were used in the variables that were consistent with the normal distribution, and the median and quarters were used in the variables that did not match the normal distribution as the descriptive statistics for the quantitative variables. Frequency and percentage are noted for qualitative variables. The level of significance was considred as 0.05 in all statistical evaluations. All statistical analyses were done using TURCOSA statistical software (Turcosa Analytics Ltd Co, Turkey, www.turcosa.com.tr).

RESULTS

The demographic characteristics of participants are presented in table 1. Demographic characteristics of patients with and without CI-AKI were similar (Table 1).

Among the echocardiographic variables, we observed that presence of pulmonary PHT was significantly associated with CI-AKI evolution (Table 2 and 5). In contrast, large left atrial diameter was not found to be significant in multivariate logistic regression analysis (OR: 1.132, 95%CI: 0.929-1.418, p: 0.243) (Table 5).

Table 1. Baseline demographic parameters of the study population

Variables	No CI-AKI (n:73)	CI-AKI (n:20)	p
Age	64±12	70±12	0,061
Gender			
Male	48(66%)	12(60%)	0,636
HT	43(59%)	15(75%)	0,178
DM	19(26%)	10(50%)	0,056
Smoking	36(49%)	10(50%)	0,957
Family history	14(19%)	7(38%)	0,148
Hyperlipidemia	9(12%)	5(25%)	0,182
Obesity	7(10%)	1(5%)	0,492
CRD history	7(10%)	1(5%)	0,492

CI-AKI, Contrast induced acute kidney injury; HT, Hypertension; DM, Diabetes Mellitus; CRD, Chronic Renal Disease; n, Patient number.

Table 2. Echocardiographic parameters of the study population

Variables	No CI-AKI (n:73)	CI-AKI (n:20)	p
LVEF (%)	46.86±10.03	44.75±10.88	0,415
LVEDD (mm)	49.23±4.80	51.25±3.70	0,083
LVESD (mm)	35.12±4.75	37.20±3.30	0,072
IVSD (mm)	11.30±1.63	11.15±1.31	0,703
PVSD (mm)	10.58±1.51	10.40±1.27	0,636
LAD (mm)	37.27±3.88	39.35±3.12	0,030
MAC	10 (13.6%)	5 (25%)	
AC	15 (20.5%)	6 (30%)	
AV (m/s)	1.36±0.31	1.40±0.25	0,663
AD (mm)	33.22±3.27	33.45±3.52	0,784
PAP (mmHg)	29.84±8.95	40.30±11.75	<0,001

CI-AKI, Contrast induced acute kidney injury; LVEF, Left ventricular ejection fraction; LVEDD, Left ventricular end-diastolic diameter ; LVESD, Left ventricular end-sistolic diameter; IVSD, Interventricular septum diameter; PVSD, Posterior ventricular septum diameter; LAD, Left atrium diameter; MAC, Mitral annular calcification; AV, aortic velocity; AC, Aortic valve calcification; AD, Ascending aorta diameter; PAP, Pulmonary artery pressure; n, Patient number.

Table 3. Angiographic parameters of the study population

Variables	No CI-AKI (n:73)	CI-AKI (n:20)	p
Indications for angiography			
CCS angina	2 (%2,74)	1(%5)	0,612
MPS positivity	2 (%2,74)	-	0,454
Exercise Test positivity	1 (%1,37)	-	0,590
Low EF	-	1 (%5)	0,055
Anterior MI	31 (%42,47)	7 (%35)	0,547
Inferior MI	22 (%30,14)	6 (%30)	0,391
Lateral MI	11 (%15,07)	2 (%10)	0,562
NSTEMI	2 (%2,74)	2 (%10)	0,560
USAP	2 (%2,74)	1 (%5)	0,612
Procedure timing (h)	1,75	2,05	0,343
Lesion type			
A	4 (%5,48)	-	
B	36 (%49,32)	15 (%75)	0,103
C	33 (%45,3)	5 (%25)	
Severity of stenosis (%)	96.01±6.89	95.50±8.019	0,777
Diameter of coronary artery (mm)	3.02±0.58	2.88±0.45	0,333
Lesions			
LMCA	3(%4,11)	1(%5)	0,770
LAD	61(%83,56)	16(%80)	0,847
CX	45(%61,64)	13(%65)	0,427
RCA	47(%64,38)	16(%50)	0,416
SB	47(%64,38)	11(%55)	0,668
Coronary ectasia	10(%13,7)	7(%35)	0,029
Coronary calcification	5(%6,85)	1(%5)	0,766
Post-PCI TİMİ flow			
0-1	22	14	0,001
2-3	51	6	
Stent procedure	63 (DES)	17 (DES)	
Stent length	38.56 (±17.24)	46.25(±23.38)	0,110
Balloon pre-dilatation	72	20	0,599
Balloon post-dilatation	66	20	0,150
Arrhythmia	29	5	0,226
Coronary diss./rupture	3	1	0,862
SB occlusion	10	4	0,485
Contrast volume (ml)	247.60	324.50	0,003
BP at procedure (mmHg)			
Sistolic	110.82±16.05	108.00±19.36	0,507
Diastolic	70.75±11.30	60.50±13.09	0,447
AF in the first 72 hours	5	2	0,131

CI-AKI, Contrast induced acute kidney injury; CCS, Canadian cardiovascular society; MPS, Myocardial perfusion scintigraphy ; EF, Ejection fraction; MI, Myocardial infarction; NSTEMI, Non ST elevation myocardial infarction; USAP, Unstable angina pectoris; LMCA, Left main coronary artery; LAD, Left anterior descending coronary artery; CX, Circumflex coronary artery; RCA, Right coronary artery; SB, Side branch coronary artery; TİMİ, Thrombolysis in myocardial infarction score; CABG, Coronary artery by-pass graft operation; DES, Drug eluting stents; BP, Blood pressure; AF, Atrial fibrillation; h, hour; n, Patient number.

Table 4. Laboratory parameters of the study population

Variables	No CI-AKI (n=73)	CI-AKI (n=20)	p
Baseline laboratory results;			
CrCl (ml/min.)	82,68±21,57	75,43±25,40	0,204
Urea (mg/dl)	38,87±12,94	50,20±19,65	0,003
Creatinine (mg/dl)	0,93±0,24	1,02±0,47	0,282
Na (mEq/L)	137,67±3,94	136,80±4,33	0,399
K (mEq/L)	4,29±0,44	4,56±0,94	0,071
CRP (mg/L)	1,70±2,85	4,59±7,67	0,002
Tn I (ng/L)	3599,82±7972,41	3334,84±4424,86	0,899
Hb (g/dL)	14,13±4,19	12,91±1,72	0,207
Hct (%)	40,25±6,33	38,35±4,73	0,215
PLT (mL*10³)	277,09±99,03	263,75±102,85	0,598
MPV (fL)	9,65±1,18	9,98±1,65	0,329
Total cholesterol (mg/dL)	180,49±52,66	175,30±56,55	0,794
Triglycerides (mg/dL)	135,57±59,71	147,60±74,50	0,450
LDL (mg/dL)	116,04±40,10	118,20±40,16	0,820
HDL (mg/dL)	38,16	41,05	0,120
Albumin (g/dl)	3,68±0,40	3,53±0,10	0,151
Total protein (g/dl)	6,58±0,63	6,56±0,57	0,879
TSH (mU/L)	1,62±2,13	1,73±1,97	0,832
72.hour laboratory results;			
72.hour Urea (mg/dl)	39,95±16,60	81,75±48,80	<0,001
72.hour creatinine (mg/dl)	0,87±0,20	1,83±1,12	<0,001
72.hour Na (mEq/L)	138,72±3,25	136,30±3,54	0,005
72.hour K (mEq/L)	4,02±0,44	4,50±0,80	<0,001
72.hour CRP (mg/L)	8,83±9,53	9,80±9,04	0,685
72.hour Tn I (ng/L)	9205,91±9738,37	8210,28±6888,24	0,719
72.hour Hb (g/dL)	12,63±3,17	12,03±2,181	0,422
72.hour Hct (%)	36,42±4,93	35,56±5,97	0,519
72hour PLT (mL*10³)	233,91±74,53	243,75±71,63	0,600
72.hour MPV (fL)	12,36±16,03	10,15±1,72	0,543

CI-AKI, Contrast induced acute kidney injury; CrCl, Creatinine clearance ; Na, Sodium ; K, Potassium; CRP, C reactive protein ; Tn I, High sensitivity cardiac troponin I; Hb, Hemoglobin; Hct, Hematocrit; PLT, Platelets; MPV, Mean platelet volume; LDL, Low density lipoprotein; HDL, High density lipoprotein; TSH, Thyroid stimulating hormone; n, Patient number.

Regarding angiographic variables, presence of coronary ectasia, post PCI (pPCI) TIMI flow 0-1, and increased contrast volume were found to be statistically significant in terms of CI-AKI evolution (Table 3 and 5).

Subjects suffering CI-AKI had higher levels of C-reactive protein (CRP) and urea in the pre-procedural laboratory analysis. However, the two groups did not differ significantly in terms of other parameters. Importantly, the groups differed significantly with regard to electrolytes evaluated at 72 hours after the procedure (Table 4).

Table 5. Multivariate logistic regression analysis of risk factors on no-reflow phenomenon

Variable	OR	%95 CI	P value
Coronary ectasia	0,122	0,019-0,657	0,018
Contrast volume	1,008	1,001-1,014	0,017
PAP	1,129	1,060-1,218	<0,001
LAD	1,132	0,929-1,418	0,243
Dystal TIMI	5,277	1,332-24,737	0,023

PAP; pulmonary artery pressure, LAD; left atrial diameter, Dystal TIMI; post PCI TIMI flow

DISCUSSION

No-reflow phenomenon following PCI has been an ominous sign that hampers microvascular perfusion even after restoration of epicardial blood flow. However, current information on its mechanisms and management strategies is still limited. The most efficient strategy is the prevention of this phenomenon through a variety of strategies including targeting reduction of thrombus burden in the pre-PCI setting. However, in most cases, there exists no signs to an impending no-reflow phenomenon. Therefore, most therapeutic strategies generally target dilatation of coronary microvascular bed (with agents including adenosine, etc.) after angiographic emergence of this phenomenon.

Similarly, CI-AKI may potentially lead to increased morbidity and mortality (11). Fundemental mechanisms of no-reflow phenomenon including increased blood viscosity in the coronary microvascular system and the immune system activation are quite analogous to those of CI-AKI evolution (12). Therefore, clarification of the potential association between these two conditions is of crucial importance for the early diagnosis and management of these conditions with considerable morbidity and mortality rates.

CI-AKI in the setting of no-reflow phenomenon might arise through a variety of mechanisms: PHT due to acute left heart failure (HF) (with consequent right HF and systemic congestion) as well as the use of diuretics might impair kidney functions in the setting of no-reflow phenomenon. Right HF and associated systemic venous congestion might potentially lead to renal hypoperfusion and congestion, and was previously reported to be the an important trigger of renal dysfunction (13). However, this type of renal injury is generally reversible in most cases.

It is well known that reduction glomerular filtration rate (GFR) with ageing might increase the risk of CI-AKI (14). However, the groups did not differ significantly with regard to age.

On the other hand, risk of CI-AKI appears to be significantly enhanced in subjects with pre-existing renal injury including diabetic nephropathy. However, the absolute causative factors of CI-AKI in this setting is not absolutely known, and trials on how DM serves as a facilitating factor for CI-AKI evolution provide limited information (15). In the present study, even though the frequency of DM did not appear to be statistically significant between the groups, the severity of DM was found to be more pronounced in the no-reflow group.

As mentioned previously, pathophysiology of CI-AKI relies on various mechanisms including vasoconstriction, medullary ischemia, and direct adverse impact of iodinated contrast media (CM). In the setting of CI-AKI, both patient and procedure-related risk factors have been suggested as potential pathogenetic contributors (16). In this context, we have

identified 'coronary ectasia' as a patient-related risk factor. It might be suggested that enhanced oxidative stress might serve as the common trigger of coronary ectasia, CI-AKI as well as no-reflow phenomenon. It might also be proposed that use of higher amounts of contrast agent in the CI-AKI group (that was one of the procedural risk factors) has been more prevalent in the setting of no-reflow phenomenon due to prolonged procedural times.

Importantly, we have also demonstrated that CI-AKI risk might significantly increase in subjects with TIMI flow 0-1 irrespective of LVEF values and TnI levels. This also suggests the common microvascular characteristics of these conditions. The laboratory tests have also revealed increased levels of urea and CRP in the CI-AKI group suggesting potential association of these markers with oxidative stress and cytotoxic effect through immune activation. Moreover, electrolyte disturbances at 72 hours might be due to CI-AKI itself.

Taken together, the present study has demonstrated that development of CI-AKI in patients with coronary no-reflow was as high as 22%, and the presence of PHT, TIMI flow 0-1and coronary ectasia were risk factors CI-AKI evolution. In this context, the present study might be considered as the first study that might shed light on this issue. In this way, early diagnosis and management of patients with coronary no-reflow phenomenon who are likely to develop CI-AKI, might significantly reduce morbidity and mortality in clinical practice.

Potential limitations of the present study might be the relatively low number of samples and evaluation by different operators (leading to interobserver variability).

CONCLUSION

In patients with coronary no-reflow phenomenon, PHT, contrast volume, post PCI TIMI flow and coronary ectasia might serve as risk factors for the development of CI-AKI.

Ethics approval and consent to participate: The study protocol conforms to the ethical guidelines of the1964 *Declaration of Helsinki* and its later amendments. This study was approved by the ethics committee of Trakya University with approval number TÜTF-BAEK 2018/305. The patients provided written consent in the study.

Conflict of Interest: The authors have no conflicts of interest to declare.

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