ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA MAKALESİ

Analysis of Contrast-Induced Nephropathy in Patient with Acute Myocardial Infarction Undergoing Percutaneous Coronary Intervention in the Emergency Department

Acil Serviste Akut Miyokard Enfarktüsü Tanısı Alıp Perkütan Koroner Girişim Uygulanan ve Kontrast Nefropati Gelişen Hastaların Analizi

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ABSTRACT

Aim: The purpose of this study was to determine the incidence and predictors of Contrast-induced acute kidney injury (CI-AKI) in emergency patients undergoing primary percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI).

Material and Methods: We enrolled electrocardiogram (ECG) findings, serum creatinine (Cr), troponin, alanine aminotransferase (ALT), aspartate aminotransferase (AST) levels at pre-PCI and Cr levels post-PCI following three days. Patients' N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, left ventricular ejection fraction (LVEF) and results of angiography was recorded in 422 patients. We analyzed the relationship between examined parameters and the occurrences of CI-AKI.

Results: CI-AKI occurred in 9.7% of patients. There was a statistically significant relationship between occurrences CI-AKI and age, gender, AST, ALT, creatinine levels at admission, cardiac dysfunction (P<0.001, P<0.01, P<0.01, P<0.05, P<0.001, P<0.05).

Conclusion: According to our study; age>70 years, abnormal pre-PCI creatinine, female gender, high basal AST-ALT levels, cardiac dysfunction may predict CI-AKI development in patients who undergoing PCI for treatment of AMI.

Keywords: Emergency**,** contrast nephropathy, percutaneous coronary intervention.

ÖZ

Amaç: Çalışmamızda, acil serviste akut miyokardiyal enfarktüs (AMI) tanısı alan ve perkutan koroner anjiyografi (PKA) uygulanan hastalarda, kontrast kaynaklı akut böbrek hasarının (CI-AKI) insidansını ve klinik prediktörlerini belirlemek amaçlandı.

Gereç ve Yöntemler: Çalışmamıza 422 hasta dâhil edildi. Hastaların, kan troponin, alanin aminotransferaz (ALT), aspartat aminotransferaz (AST) düzeyleri, başvuru sırasındaki ve PKA'dan üç gün sonraki serum kreatinin (Cr) düzeyleri, N-terminal pro-beyin natriüretik peptid (NT-proBNP) seviyeleri, elektrokardiyogram (EKG) bulguları, sol ventriküler ejeksiyon fraksiyonu (LVEF) ve anjiyografi sonuçlarını kaydettik. İncelenen parametreler ile CI-AKI oluşumları arasındaki ilişkiyi analiz ettik.

Bulgular: CI-AKI hastaların% 9.7'sinde görüldü. CI-AKI ile yaş, cinsiyet, AST, ALT, başvuru anındaki kreatinin düzeyleri, kardiyak disfonksiyon varlığı arasında istatistiksel olarak anlamlı bir ilişki vardı (P <0.001, P <0.01, P <0.01, P <0.05, P <0.001, P < 0.05).

Sonuç: Çalışmamıza göre; Yaş> 70, anormal pre-PCA kreatinin, kadın cinsiyet, yüksek bazal AST-ALT seviyeleri, kardiyak disfonksiyon, AMI tedavisi için PKA uygulanan hastalarda CI-AKI gelişimini öngörebilir.

Anahtar Kelimeler: Acil, kontrast nefropati, perkutan koroner anjiyografi

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Introduction

Contrast-induced acute kidney injury (CI-AKI) is the sudden change in renal function following contrast medium administration. It occurs 1-3 days after the procedure and usually returns to normal within 7 days (1,2). CI-AKI incidence varies between 2% and 50% (3).

Emergency percutaneous coronary intervention (PCI) is the best treatment approach in the treatment of acute myocardial infarction (AMI) (4,5). PCI restores the circulation of coronary arteries and improves the survival of the patient by correcting the feeding of the myocardium. CI-AKI due to contrast agent used during the procedure is one of the major complications affecting the mortality and morbidity of the patient. The incidence of CI-AKI that develops after PCI varies between 2-20% (6). CI-AKI rarely requires dialysis (7). The risk of developing CI-AKI is higher in emergency PCI than elective (8-10).

Although many risk factors affecting the development of CI-AKI after PCI have been identified, the relationship between them has not been fully clarified (11). Determining the predicting parameters and risk factors for CI-AKI that may develop in patients with emergency PCI is important for prognosis. In our study, we evaluated the AMI patients who undergoing emergency PCI. The purpose of this study was to determine the incidence and predictors of CI-AKI in patients undergoing primary PCI for AMI.

Material and Methods

The patients who presented to Emergency Department (ED)-which has an annual turnover of 250.000 patients - between 1 January 2017 and 31 April 2019 were screened. We studied 18 aged and above patients who diagnosed with ST elevation myocardial infarction (STEMI) in ED and underwent PCI within the first hour. Our study was designed retrospectively. Patients with cirrhosis, chronic renal failure (CRF) requiring dialysis and anemia which hemoglobin values<10 g/dl (Reference value= 12.6-17.0 g/dl) were excluded from the study.

422 patients were included in the study. Demographic characteristics of the patients, electrocardiogram (ECG) findings, pre-PCI blood creatinine, troponin, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and N-terminal pro-brain natriuretic peptide (NT-proBNP) values, vascular lesions which were detected in PCI and creatinine values of the first 3 days after PCI were recorded. The patients' age were divided into two groups as <70 age and over.

Lesions which were detected in PCI were grouped into left anterior descending artery (LAD), circumflex artery (Cx) and right coronary artery (RCA). Vessels with obstruction above 50% were evaluated as abnormal.

NT-proBNP or left ventricular ejection fraction (LVEF) was used to evaluate cardiac decompensation of patients. NT-proBNP> 2000 pg/ml (Reference value = 0-100 pg / ml) or LVEF <40% was considered abnormal.

CI-AKI was defined as 25% or 0.5 mg/dl increase from baseline Cr within 72 hours after PCI.

The relationship between CI-AKI development and the age, gender, ALT, AST, troponin, creatinine levels, cardiac dysfunction (decision based on NT-proBNP or LVEF) of the patients were analyzed by chi-square method.

The statistical analysis of our data was performed using the "SPSS for Windows Version 16.0" software. P <0.05 was considered statistically significant.

The ethical approval of the study was gotten from the local ethical committee with the approval number 12.08.2020/1034

Results

422 patients who were diagnosed as STEMI in the ED and undergoing PCI were included in our study. 308 (73%) of the patients were male and 114 (27%) were female.

The patients' age is minimum 29, maximum 92 and the mean age was 60.14 +/-12.5. According to the ECG findings in the ED, 228 (54%) of the patients were anterior, 169 (40%) were inferior, 14 (3.32%) were posterior and 11 (2.6%) were lateral STEMI. Laboratory parameter results of the patients are summarized in Table 1.

The maximum measured ALT level was 611 U/L, the mean value was 22.25 +/- 49.08 U/L (Reference value: 0-34 U/L). The maximum measured AST level was 800 U/L, the mean value was 66.46 +/-121.74 U/L (Reference value: 0-35 U/L). The maximum measured creatinine level of the patients at the time of admission was 4.8 mg/dl (Reference value: 0.66-1.09 mg/dl). The maximum measured creatinine level after PCI was 7.6 mg/dl. The development of acute renal failure requiring hemodialysis after PCI was seen in only 2 (0.47%) patients. Pre-procedure creatinine levels were high in these two patients.

CI-AKI occurred in 35(9.7%) patients. There was a statistically significant relationship between CI-AKI and gender, pre-PCI creatinine level, basal AST, ALT levels (P<0.01, P<0.001, P<0.01, P<0.05). When the age of the patients was divided into two groups <70 and above, a statistically significant difference was found between the two groups in terms of development of CI-AKI (P<0.001).

There was no statistically relationship between troponin elevation and CI-AKI (P=0.555).

No statistical relationship was found between CI-AKI and the diagnosis of the patients according to ECG findings (P =0.704). Cx, LAD and RCA lesions were evaluated in angiography of the patients. Accordingly, 200 (47.4%) patients had one vessel, 126 (29.9%) had 2 vessels, 90 (21.3%) had 3 vessels with more than 50% obstructive

Laboratory Parameters (Normal Value)	Number of Patients with normal value	Number of patient with abnormal value	Total number of patients
Alanine aminotransferase (0-34 U/L)	365 (86.5%)	57 (13.5%)	422 (100%)
Aspartate aminotransferase (0-35 U/L)	285 (67.5%)	137 (32.5%)	422 (100%)
Troponin	127 (30.1%)	295 (69.9%)	422 (100%)
Pre-PCI creatinine (0.66-1.09 mg/dl)	352 (83.4%)	70 (16.6%)	422 (100%)
Post-PCI creatinine	320 (75.8%)	102 (24.4%)	422 (100%)

Table 1. Laboratory Parameters of Patients

lesions. 6 (1.4%) patients had normal angiography. There was no statistically significant relationship between CI-AKI and the presence of lesions in the angiography or the number of lesional vessels (*P*=0.460, *P*=0.289).

303 patients with NT-proBNP or LVEF were enrolled. 90 (21.3%) had high NT-proBNP or LVEF <40%. There was statistically significant relationship between CI-AKI and cardiac dysfunction (*P* <0.05).

Discussion

CI-AKI is defined as renal dysfunction associated with the administration of contrast media, in the absence of any alternative etiology. For CI-AKI there are different definitions and different creatinine cut-off values in guidelines (6). In our study, CI-AKI was defined as 0.5 mg/dl or 25% in serum creatinine increase from pre-PCI creatinine level.

The incidence of CI-AKI after PCI is different in studies. In our study, incidence of CI-AKI independent of pre-procedure creatinine level was found 9.7%. Incidence of CI-AKI was found 13.1% in study of Mehran R. et al., 22.7% in study of Yuan Y. et al., 15.8% in study of Sun G. et al, and 19% in study of Marenzi G. et al (9,11-13). In study of Chong E et al., CI-AKI incidence was found 7.3% in patients who underwent emergency PCI after AMI with normal creatinine levels (10). In the study of Yuan hui Liu et al, the incidence of CI-AKI in patients with STEMI was found 9.2% (14). Many studies have been done to determine the risk factors for the development of CI-AKI in patients undergoing PCI. It was shown that there was a difference in the development of CI-AKI between the emergency and elective PCI (3). Age was found to be an important risk factor for CI-AKI (6,9,10,12,13). In our study, a significant relationship was found between advanced age and CI-AKI. In our study, the effect of gender on the development of CI-AKI was analyzed and a significant statistically differences was found in women. In study of Lucreziotti S et al., there was found that female gender increased the risk of CI-AKI after PCI in AMI (15).

Determination of basal creatinine value before the procedure may be useful in predicting the development of nephropathy. In study of Chalikias G. et al., the impaired renal function at baseline was found an independent risk factor for CI-AKI (6). Iakovou I. et al. studied 8628 patients who underwent PCI. In their study, pre-PCI chronic renal failure was found to be an independent predictor of CI-AKI (16). According to the study of Chong E. et al., the incidence of post-PCI contrast-induced nephropathy (CIN) development in patients with normal serum creatinine level was lower than in our study (10). The need of dialysis in patients with CI-AKI due to PCI is rare (6). In our study, the need of dialysis was <1%.

In our study, we did not find any statistical relationship between CI-AKI and ECG findings, the degree of obstruction in the vessels, the number of vessels with lesions. There was no difference in CI-AKI development between patients with normal and abnormal angiography. In study of Marenzi G., there was found correlation with CIN and anterior MI (9). In the study of Yuan Y. et al, stent application to LAD was considered to be an independent risk factor for CI-AKI (11).

Recent data proved the effect of BNP for predicting CIN. Study of Yuan-hui Liu et al. showed that the best cut-off NT-proBNP value for detecting CIN was 1800 pg/mL with 69% sensitivity and 70.0% specificity (14). In study of Kurtul A. et al, there was found that NT-proBNP ≥2149 pg/mL measured on admission had a 79.4% sensitivity and 74.3% specificity in predicting CIN (17). In our study, NT-proBNP elevation or low LVEF were evaluated as cardiac dysfunction. According to our study; there was statistically significant relationship between cardiac dysfunction and CI-AKI.

Limitations

Our study is retrospective and is not multi-centered. Hemodynamic instability parameters such as hypotension could not be evaluated. The number of patients developing CI-AKI is low.

Conclusion

In the ED, CI-AKI should be kept in mind in the AMI patients who underwent PCI. Early recognition, definition of risk factors and preventative therapies for CI-AKI in the ED is paramount to the patient's survival.

Definition of risk factors is important for the prognosis of patients and identification of treatment strategies for CI-AKI. Causes or risk classifications of CI-AKI have not been clarified yet. According to our study; age> 70 years, abnormal pre-PCI creatinine, female gender, high basal AST-ALT levels, cardiac dysfunction (high NT-proBNP and/or low EF) may predict CI-AKI development in patients who undergoing PCI for treatment of AMI.

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Authors' Contribution: All authors contrubuted for conception, design of the study, data collection, data analysis, and assembly. The manuscript was written and approved by all authors.

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All authors declared that they follow the rules of Research and Publication Ethics.

References

1- Barrett BJ, Parfrey PS. Clinical practice. Preventing nephropathy induced by contrast medium. *N Engl J Med*. 2006; 26;354(4):379-86.

2- Guitterez NV, Diaz A, Timmis GC, et al. Determinants of serum creatinine trajectory in acute contrast nephropathy. *J Interv Cardiol*. 2002; 15(5):349-54.

3- Kumar S, Nair RK, Aggarwal N, et al. Risk factors for contrast-induced nephropathy after coronary angiography.*Saudi J Kidney Dis Transpl.* 2017; 28(2):318-24.

4- Zijlstra F, Hoorntje JC, de Boer MJ, et al. Long-term benefit of primary angioplasty as compared with thrombolytic therapy for acutemyocardial infarction. *N Engl J Med.* 1999;341:1413-19.

5- Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. *Lancet.* 2003;361:13–20.

6- Chalikias G, Drosos I, Tziakas DN. Contrast-Induced Acute Kidney Injury: An Update.*Cardiovasc Drugs Ther. 2*016;30(2): 215-28.

7- Gruberg L, Mehran R, Dangas G, et al. Acute renal failure requiring dialysis after percutaneous coronary interventions. *Catheter Cardiovasc Interv*. 2001;52(4):409-16.

8- Rihal CS, Textor SC, Grill DE, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. *Circulation.* 2002(14);105(19):2259-64.

9- Marenzi G, Lauri G, Assanelli E, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol.* 2004;44:1780–85.

10- Chong E, Poh KK, Liang S, et al. Comparion of risks and clinical predictors of contrast-induced nephropathy in patients undergoing emergency versus nonemergency percutaneous coronary interventions. *J Interv Cardiol.* 2010;23(5):451-9.

11-Yuan Y, Qiu H, Hu XY, et al. Risk Factors of Contrast-induced Acute Kidney Injury in Patients Undergoing Emergency Percutaneous Coronary Intervention. *Chin Med J (Engl)*. 2017;130(1):45-50.

12- Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol.*. 2004(6);44(7):1393-9.

13 Sun G, Chen P, Wang K, et al. Contrast-Induced Nephropathy and Long-Term Mortality After Percutaneous Coronary Intervention in Patients with Acute Myocardial Infarction. Angiology. 2019 Aug;70(7):621-626. doi: 10.1177/0003319718803677.

14- Liu YH, Jiang L, Chen J, et al. Does N-terminal pro-brain natriuretic peptide add prognostic value to the Mehran risk score for contrast-induced nephropathy and long-term outcomes after primary percutaneous coronary intervention? *Int Urol Nephrol.*2016;48(10):1675-82.

15- Lucreziotti S, Centola M, Salerno-Uriarte D, et al. Female gender and contrast-induced nephropathy in primary percutaneous intervention for ST-segment elevation myocardial infarction. *Int J Cardiol*. 2014;1;174(1):37-42.

16- Iakovou I, Dangas G, Mehran R, et al. Impact of gender on the incidence and outcome of contrast induced nephropathy after percutaneous coronary intervention. *J Invasive Cardiol.* 2003;15(1):18-22.

17- Kurtul A, Duran M, Yarlioglues M, et al. Association Between N-Terminal Pro-Brain Natriuretic Peptide Levels and Contrast-Induced Nephropathy in Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndrome. *Clin Cardiol.* 2014;37(8):485-92.