Ann Med Res 2021;28(7):1273-6

Annals of Medical Research

DOI: 10.5455/annalsmedres.2020.04.403

Original Article

Contrast nephropathy ıncidence after coronary angiography among patients with acute renal dysfunction

Bilal Katipoglu[1], Ihsan Ates[2], Fatih Acehan[2], Fatih Dede[3]

[1]Division of Geriatrics, University of Health Sciences, Gulhane Faculty of Medicine and Gulhane Training and Research Hospital, Ankara, Turkey
[2]Department of Internal Medicine, Ankara City Hospital, Ankara, Turkey
[3]Department of Nephrology, Ankara City Hospital, Ankara, Turkey

Abstract

Aim: Contrast-induced acute kidney injury (AKI) may be seen after contrast agent exposure which is known as nephrotoxic. It is fact that, patients with AKI have an increased risk after contrast exposure compared to patients with normal renal function values. However, the incidence of contrast nephropathy after contrast exposure with AKI is not clear. In this study, we planned to investigate the incidence of contrast nephropathy in patients undergoing coronary angiography with AKI and whether there is an increased risk compared to the control group.

Materials and Methods: The study was planned as a retrospective study and we investigated the clinical outcome of patients who underwent isoosmolar contrast angiography in the arterial phase with acute kidney injury between January 2014 and December 2017.

Results: The number of AKI patients included in the study was 280 and the control group consisted of 478 participants. There was no statistically significant difference between two groups. (3.2% (n: 9) of patients with AKI contrast nephropathy vs 6.5% (n: 31) of patients with normal kidney function and contrast nephropathy) (p> 0.05). On the other hand, there was statistically significant difference between two groups according to diabetes mellitus (patients with contrast nephropathy 26 (65%) had diabetes vs 14 (35%) participants had no diabetes). This difference was statistically significant (p <0.05).

Conclusion: In this study, in patients who underwent emergency angiography with a preliminary diagnosis of acute coronary syndrome in the emergency department, we did not find an increased risk of acute renal injury and contrast-enhanced nephropathy compared to the patient population with normal kidney function.

Keywords: Acute kidney injury; contrast nephropathy; coronary anjiography

INTRODUCTION

Acute kidney injury (AKI) is an important cause of mortality and morbidity in the community. Contrast-related AKI can also be seen after contrast agent which is known nephrotoxic. Although the pathophysiology is not clearly elucidated, it is thought that contrast nephropathy may occur due to hemodynamic changes such as direct tubular toxicity effect of the contrast agent, oxidative stress and renal vasoconstriction. Although the incidence of contrast-related AKI is less than 2% in the general population, the incidence is higher in patients with diabetes and chronic kidney disease (CKD).

Patients with AKI are known to have an increased risk factor for devolopment of contrast induced nephropathy after contrast exposure when compared to patients with normal kidney function (1-3). However, the incidence of contrast nephropathy after contrast exposure with AKI is not clear (4). In this study, we aimed to investigate the incidence of contrast nephropathy and changes in renal function in patients undergoing coronary angiography with AKI.

MATERIALS and METHODS

Patient Population

The study was planned as a retrospective study and we evaluated the clinical outcome of patients who underwent isoosmolar contrast angiography in the arterial phase with AKI between January 2014 and December 2017.

The study was approved in accordance with the patient rights regulation and approved by the Ankara Numune Education and Research Hospital Scientific Research Evaluation Commission on the date of 21/03/2018 with the decision number 1856-2018.

Definitions

Contrast nephropathy was defined as a consulted to the nephrology clinic and exact diagnosed with contrast

Received: 28.04.2020 **Accepted:** 29.07.2020 **Available online:** 08.07.2021
Corresponding Author: Bilal Katipoglu, Division of Geriatrics, University of Health Sciences, Gulhane Faculty of Medicine and Gulhane Training and Research Hospital, Ankara, Turkey **E-mail:** drbilal07@gmail.com

nephropathy were included and following two definitions are used increase from baseline in serum creatinine (sCr) ≥0.3mg/dl or increase from baseline of 25% within 72 hours (5).

AKI was defined as an absolute increase in sCr, at least 0.3 mg/dL (26.5 µmol/L) within 48 hours or by a 50% increase in sCr from baseline within 7 days, or a urine volume of less than 0.5 mL/kg/h for at least 6 hours before contrast exposure (6).

Control group was defined as patients who have kidney function test normal before contrast exposure.

Diabetes Mellitus was defined as medical history of patients who used anti diabetic agents or HbA1c ≥6.5% (7).

Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) for Windows 20 (IBM SPSS Inc., Chicago, IL) program. The normal distribution of the data was evaluated by Kolmogorov-Smirnov test. Numerical variables with normal distribution were shown as mean ± standard deviation and numerical variables with not normal distribution were shown as median (min-max). Categorical variables were shown as numbers and percentages. Continuous variables were compared with independent sample t-test, ANOVA or Mann Whitney U test, Kruskall Wallis H test where appropriate. Comparison of categorical variables was made with Chi-square test. P<0.05 values were considered statistically significant.

RESULTS

The number of AKI patients included in the study was 280 and the control group consisted of 478 participants. 116 (41.4%) patients in the AKI group were female and 166 (34.7%) patients of the control group were female. The mean eGFR of the AKI group was 43.6ml/min, while the eGFR of the control group was 60.4ml/min. eGFR difference was statistically significant when compared with patients with AKI and normal renal function (p <0.001). The median length of hospitalization was 5 day in patients with AKI and 4 day in the control group. This difference was not statistically significant (p> 0.05) (Table 1).

Table 1. Demographic and clinical data of patients with AKI and normal kidney function

	AKI (n=280)	Normal (n=478)	P value
Age (year)	73 (35-95)	74 (21-92)	0.182
Gender (female), n (%)	116 (41.4)	166 (34.7)	0.06
GFR(ml/min)	43.6±10.2	63.4 ±2.8	**0.001***
Hospitilization(day)	5 (0-114)	4 (0-76)	0.088
Contrast Nephropathy n (%)	9 (3.2)	31 (6.5)	0.052

AKI: Acute kidney injury GFR: Glomerular filtration rate
Numerical variables with normal distribution were shown as mean ± standard deviation.Numerical variables with not normal distribution were shown as median (min-max). Categorical variables were shown as numbers (%). * p <0.05 was statistically significant

There was no statistically significant difference between 9 (3.2%) of patients with AKI and contrast nephropathy and 31 (6.5%) of patients with normal kidney function and contrast nephropathy (p=0.052) (Table 1).

Diabetes Mellitus was detected in 26 (65%) patients who developed contrast nephropathy, while 14 (35%) participants had no Diabetes Mellitus. This difference was statistically significant (p=0.004) (Table 2).

Table 2. The relationship between Contrast Nephropathy and Diabetes Mellitus

	DM (+)	DM (-)	P value
Contrast Nephropathy (+) (n=40)	26 (65%)	14 (35%)	*0.004
Contrast Nephropathy (-) (n=718)	301 (41.9%)	417 (58.1%)	

DM:Diabetes Mellitus
* p <0.05 was statistically significant

DISCUSSION

Contrast nephropathy is one of the most common causes of AKI in clinical practice (8). This kidney injury can be irreversible and may progress CKD (9). The most important mechanism in the pathophysiology of contrast nephropathy is medullary hypoxia and direct renal tubular toxicity due to medullary vasoconstriction (10,11). Oxidative stress also plays an important role in the development of contrast nephropathy (12,13). In addition, contrast exposure causes increased viscosity in the circulation, which further exacerbate the medullary hypoxia (14).

The most important risk factor for the development of contrast nephropathy is known to be CKD and diabetes mellitus (15). In one study, the incidence of contrast nephropathy was higher in patients with CKD and diabetes than in patients with CKD (16). Similar to the studies in the literature, in our study, a significantly higher detection of diabetes patients in the development of contrast nephropathy indicates that diabetes is an important risk factor in the development of contrast nephropathy.

In addition, contrast exposure was found to be intra-arterial and the risk of contrast nephropathy was higher compared to intra-venous exposure (17). One of the reason for this may be the higher density of the contrast agent in the kidney in the arterial phase (18). In our study although percutaneous transluminal coronary angioplasty (PTCA) was performed in the arterial phase, we found similar contrast nephropathy in patients with AKI when compared to the group with normal kidney function. Although the development of contrast nephropathy in patients with AKI is of clinical importance, we have shown in this study, it may not be worry about the development of contrast nephropathy in patients with AKI.

In observational study, the incidence of contrast nephropathy patients with CKD and PTCA and was found to be 12% and progression was defined in 18% of these

patients (9). In another report, 16% persistent renal damage was detected in patients who underwent PTCA and a significantly higher 5-year mortality rate was found (19). In another study, the incidence of new renal failure was found 0.9% within 6 months after PTCA. In this study, also a lower incidence of CKD was found in PTCAs with trans-radial catheter than femoral catheters (20). In another study, the incidence of contrast nephropathy was found to be less than 2%, but in patients with CKD and diabetes, the incidence can be as high as 50% (21). In our study, factors such as the presence of trans-radial catheters, use of isoosmolar contrast media, research population may be the reason for lower incidence of contrast nephropathy.

Another study showed that , the risk of contrast induced nephropathy has been exaggerated. Clinical adverse outcomes from cardiac catheterization and intervention is lower than expected side effects such as contrast nephropathy (22). Fluctuations in creatinine levels can be seen especially in hospitalized patients. Contrast nephropathy can be overdiagnosis due to many confounding factors (23). In our study, we included cases that were diagnosed with contrast nephropathy and were confirmed with nephrologist. As a result, the confounding factors were eliminated and the actual incidence of the contrast nephropathy was found similar with normal kidney function in the litrature (24). We can speculated that, patient with AKI may not increase risk for contrast nephropathy.

CONCLUSION

Patients with risk factors such as diabetes and chronic kidney damage should be closely monitored for the development of contrast nephropathy. The incidence of contrast nephropathy with AKI was found to be lower than expected.

This report has some limitations. This study was performed retrospectively. Before hospitalization, the current treatments for patients are unknown. The etiology of AKI has not been clarified by prerenal, renal and postrenal diseases. However, studies have shown that kidney damage is an independent risk factor despite of etiology (25). This study will help to clarify this issue because it is difficult to make a prospective study of incidence of contrast nephropathy in patients with AKI in the literature.

Competing Interests: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical Approval: The study was approved in accordance with the patient rights regulation and approved by the Ankara Numune Education and Research Hospital Scientific Research Evaluation Commission on the date of 21/03/2018 with the decision number 1856-2018.

REFERENCES

1. Babuin L, Scarpa D, Jaffe AS. Renal Dysfunction: How to Think About That in Acute Coronary Syndromes. Curr Cardiol Rep 2017;19:91.
2. Simone DB, Ansaloni L, Sartelli M, et al. Is the risk of contrast-induced nephropathy a real contraindication to perform intravenous contrast enhanced Computed Tomography for non-traumatic acute abdomen in Emergency Surgery Department? Acta Biomed 2018; 89:158-72.
3. Sedhai YR, Golamari R , Timalsina S, et al. Contrast-Induced Nephropathy After Cardiac Catheterization: Culprits, Consequences and Predictors.Am J Med Sci 2017;354:462-6.
4. Bruce RJ, Djamali A, Shinki K, et al. Background fluctuation of kidney function versus contrast-induced nephrotoxicity. AJR Am J Roentgenol 2009;192:711-8.
5. Guitterez NV, Diaz A, Timmis GC, et al. Determinants of serum creatinine trajectory in acute contrast nephropathy. J Interv Cardiol 2002;15:349-54.
6. Kellum JA, Lameire N, Aspelin P, et al. Kidney disease: improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int 2012;2:1-138.
7. Association AD. 2. Classification and diagnosis of diabetes. Diabetes Care 2017;40:11-24.
8. Rudnick M, Feldman H. Contrast-induced nephropathy: what are the true clinical consequences? Clin J Am Soc Nephrol 2008;3:263-72.
9. Maioli M, Toso A, Leoncini M, et al. Persistent renal damage after contrast-induced acute kidney injury: incidence, evolution, risk factors, and prognosis. Circulation 2012;125:3099-107.
10. Scharnweber T, Alhilali L, Fakhran S. Contrast-induced acute kidney injury: pathophysiology, manifestations, prevention, and management. Magn Reson Imaging Clin N Am 2017;25:743-53.
11. Heyman SN, Brezis M, Epstein FH, et al. Early renal medullary hypoxic injury from radiocontrast and indomethacin. Kidney Int 1991;40:632-42.
12. Naziroglu M, Yoldas N, Uzgur EN, Kayan M. Role of contrast media on oxidative stress, Ca 2+ signaling and apoptosis in kidney. J Membr Biol 2013;246:91-100.
13. Liu ZZ, Schmerbach K, Lu Y, et al. Iodinated contrast media cause direct tubular cell damage, leading to oxidative stress, low nitric oxide, and impairment of tubuloglomerular feedback. Am J Physiol Renal Physiol 2014;306:864-72.
14. Zhang Y, Wang J, Yang X, et al. The serial effect of iodinated contrast media on renal hemodynamics and oxygenation as evaluated by ASL and BOLD MRI. Contrast Media Mol Imaging 2012;7:418-25.
15. Ozkok S, Ozkok A. Contrast-induced acute kidney injury: A review of practical points. World J Nephrol 2017;6:86.
16. Parfrey PS, Griffiths SM, Barrett BJ, et al. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. New England J Med 1989;320:143-9.
17. Li J, Solomon RJ. Creatinine increases after intravenous contrast administration: incidence and impact. Invest Radiol 2010;45:471-6.

18. Dong M, Jiao Z, Liu T, et al. Effect of administration route on the renal safety of contrast agents: a meta-analysis of randomized controlled trials. J Nephrol 2012;25:290.
19. Nemoto N, Iwasaki M, Nakanishi M, et al. Impact of continuous deterioration of kidney function 6 to 8 months after percutaneous coronary intervention for acute coronary syndrome. Am J Cardiol 2014;113:1647-51.
20. Vuurmans T, Byrne J, Fretz E, et al. Chronic kidney injury in patients after cardiac catheterisation or percutaneous coronary intervention: a comparison of radial and femoral approaches (from the British Columbia Cardiac and Renal Registries). Heart 2010;96:1538-42.
21. Manske CL, Sprafka JM, Strony JT, Wang Y. Contrast nephropathy in azotemic diabetic patients undergoing coronary angiography. Am J Med 1990;89:615-20.
22. Katzberg RW, Newhouse JH. Intravenous contrast medium–induced nephrotoxicity: is the medical risk really as great as we have come to believe? : Radiology 2010;256:21-8.
23. Newhouse JH, Kho D, Rao QA, Starren J. Frequency of serum creatinine changes in the absence of iodinated contrast material: implications for studies of contrast nephrotoxicity. AJR Am J Roentgenol 2008;191:376-82.
24. Rihal CS, Textor SC, Grill DE, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation 2002;105:2259-64.
25. McCullough PA, Wolyn R, Rocher LL, et al. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. Am J Med 1997;103:368-75.