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Original Article

The correlation between the increase rate of serum creatinine levels and long-term adverse clinical outcomes in patients with non st-segment elevation myocardial infarction

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Abstract

Aim: We purposed to evaluate the correlation between the rate of increase in SCrea levels and major adverse cardiac and cerebrovascular events(MACCE) in non ST-segment elevation myocardial infarction(NSTEMI) patients who was made coronary angiography(CAG) in this study. According to studies on especially stable coronary artery disease (SCAD); contrast-induced acute kidney injury CI-AKI) is described as an rising in serum creatinine (SCrea) levels more than 0.5 mg / dl or more than 25% within 48-72 hours after the contrast agent implementation. However, data on the increase rates of SCrea levels in patients with acute coronary syndrome (ACS) are insufficient..

Materials and Methods: 884 NSTEMI patients were admitted to our study. We classified the patients into 3 groups according to the increase rates in SCrea values; first group(ΔSCrea <10%), second group(10%≤ ΔSCrea <25%) and third group(ΔSCrea ≥25%). Results: MACCE were defined as all-cause mortality, myocardial infarction (MI) and cerebrovascular accident (CVA) at one year follow-up. MACCE occurred in 123(13.9%) of the 884 patients. Patients in group three had a meaningfully higher rate of MACCE than in the other groups (P < 0.001). This difference was primarily sourced from all-causes mortality; the all-causes mortality ratio was 3-4 times higher than the other groups. There was no meaningful difference in MACCE among first and second groups.

Conclusion: Using an increase rate of ≥25% creatinine as the definition for CI-AKI is more reliable for primary end points in patients with NSTEMI than the increase rate of creatine in lower levels.

Keywords: Contrast induced nephropathy; serum creatinine increase ratio; acute coronary syndrome; adverse clinical outcomes

INTRODUCTION

In patients who develop contrast-induced acute kidney injury (CI-AKI), prolonged hospitalization increase in morbidity and mortality was reported (1,2). It is described as an acute reduction in glomerular filtration rate (GFR), manifested by an increase in serum creatinine (SCrea) that occurs in the first 24 hours after contrast media exposure, and peaking up to five days afterwards(2,3). To our knowledge, there is no consensus on the optimal description of CI-AKI. According to the literature data, CI-AKI is described as an increase of SCrea levels more than 0.5 mg / dl or more than 25% within 48-72 hours after the contrast agent implementation. In most of these clinical trials, patients with stable coronary artery diseases (SCAD) are included (2-5). This description has also consistently predicted major advers cardiac and cerebrovascular events (MACCE) after contrast agent encountered because of coronary angiography (CAG) in SCAD patients (6). Acute coronary syndrome patients (ACS) have a three times higher risk for CI-AKI (7). In one study, authors have shown that inflammatory cells accumulate in various organs during ACS, including the kidney (8). This inflammatory state may explain the frequency of CI-AKI in ACS patients. Studies have demonstrated that the CI-AKI incidence after coronary interventions is more frequent in ACS patients than in those with stable patients (9-11). For this reason, patients with ACS should be very carefully monitored for CI-AKI. In addition, by the recommendations from Kidney Disease: Improving Global Outcomes (KGIDO) and the acute kidney injury network (AKIN), the AKI definition is an increase of 0.3 or 50% in the SCrea level (12,13). In studies on CI-AKI definition, the majority of patient populations have SCAD, so that it is important to reevaluation CI-AKI

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definitions for those with ACS. We sought to investigate the correlation between the ratio of enhancement in SCrea and MACCE in NSTEMI patients who have been CAG or percutaneous coronary intervention (PCI).

MATERIALS and METHODS

Patient groups

Our study was a prospective single center study. 1,052 ACS patients who applied to our center between 2011 and 2015 were included in the study to evaluate. ACS patients who refused additional therapy and included in the risky group (ie, patients with mechanical complications occurring in the course of myocardial infarction; patients who have been life-threatening arrhythmias or cardiac arrest in the course of MI, patients who have been hemodynamical uncertainty or cardiogenic shock) were excluded from the study. In addition; patients with heavy impairment of systolic left ventricular function, killip score III-IV, acute renal injury, chronic renal disease at terminal stage, gravidity and a history of contrast-related allergies were excluded. After exclusion criteria, 916 patients were analyzed in the study. Besides, the SCrea value of 19 patients was absent, and 13 patients did not requirement CAG, and these patients were excluded in the study. A flowchart of the patients selection was demonstrated that Figure -1. We received written informed consent from all patients who enrolled in the study, and this study was approved by the local ethics council.

Study Protocol

NSTEMI was described as elevated cardiac enzyme values showing myocardial destruction (troponin T/I and creatinine kinase-MB) with at one and/or more of the following situations: (1) Having ischemia complaints longer than 20 minutes; (2) high risk criteria for ECG findings (T wave changes, ST segment decline or temporary ST segment elevation (<30 minutes); (3) new abnormal local wall movement (14). Charging dosages of adenosine diphosphate (ADP) receptor antagonist (300 mg clopidogrel or 180 mg ticagrelor) and acetylsalicylic acid (300 mg) were every time administered to NSTEMI patients during their acceptance. Estimated glomerular filtration rate (eGFR) rates were calculating by the Levey-modified Modification of Diet in Renal Disease formula (MDRD): (186.3*SCrea[mg/dL]-1.154)*(age[years]-0.203)*(0.742 patients with female)(15). If the patients we included in our study were at high risk for CI-AKI, hydration treatment was applied with intravenous (IV) isotonic saline for 12 hours before the procedure and 24 hours after the procedure (16]. In patients whose left ventricular ejection fraction (LVEF) was calculated under 40% by echocardiography, hydration treatment was applied by decreasing IV isotonic saline rate to half dose(0.5 ml/kg/h).

The risk levels of the patients included in the study for the development of CI-AKI were calculated using the Mehran risk score (4). The PCI of the patients were performed by experienced interventional cardiologists, who performed more than 200 cases annually via the radial or femoral artery. Low osmolar non-ionic contrast media was applied in all treatments. IV unfractionated heparin (UFH) was administered in all patients when PCI was planned (100 IU/ kg; dose reduced to 50IU / kg in patients requiring gp IIb / IIIa inhibitor during the procedure). Selection of stent type and size; thrombus aspiration catheter usage; GP IIb/IIIa inhibitors therapy were released to the doctor who made PCI. The biochemical parameters (SCrea, blood urea nitrogen, sodium, potassium etc) of the patients were analyzed during their acceptance and 48-72 hours after the procedure. Patients with detected CI-AKI in the follow-up were examined again in terms of biochemical parameters on the 5th and 10th days. All biochemical tests for CI-AKI were performed in the same laboratory with the same method. In our study, we classified the patients into 3 groups according to the increase rates in SCrea values; first group(ΔSCrea <10%), second group(10%≤ ΔSCrea <25%) and third group(ΔSCrea ≥25%). Follow up of all patients were performed every 3 months by clinical or telephone interviews.

Statistical Analysis

Statistical evaluation of our trial was made by the SPSS 21 (IBM Corp., Armonk, NY, USA) package program. Normal distribution analyzes of continuous variables were done with the Kolmogorov-Smirnov test. If continuous variables showed normal distribution among three groups, we analysed by one-way ANOVA tests and if they didn't normally distributed, we analysed by Kruskal-Wallis tests. Categorical parameters were evaluated using the chi-square test. Predictors of MACCE were evaluated using a multivariate analysis model on parameters that had P values < 0,1. If P values were found to <0,05 (2 tailed), it was accepted statistically significant.

RESULTS

Demographical and Biochemical Parameters

Overall, 512 (57.9%) patients met the criteria for inclusion in group 1, 210 (23.7%) patients in group 2, and 162 (18.3%) patients in group 3. The clinical, demographic, angiographic characteristics and biochemical features of the three groups are demonstrated in Table-1 and 2.

Some parameters were significantly different between the three groups. Especially, there was a lower incidence of male sex, frequency of diabetes mellitus, ratio of active smoking, rate of ejection fraction (EF), and lower hemoglobin values in group 3. Whereas, systolic blood pressure, length of hospital stay and ratio of multivessel disease were higher in group 3. Serum platelets, triglycerides, baseline glucose, HbA1c, and baseline creatinine values were also higher in group 3. Amount of contrast media was similar among the 3 groups.

Clinical Adverse Events

In our study, patients were followed up for a median of 22 (min: 12-max: 48) months. Clinical adverse events at long-term follow-up for all groups are shown in Table 3. Major adverse events happened in 123 (13.9%) of the 884

patients. In group-3 patients had a meaningfully high percentage of MACCE than in the groups (P < 0.01). This difference was mostly caused by all causes mortality; the mortality rate was 3-4 times higher than the other groups. There was no difference in MACCE between groups 1 and 2.

The demographic and biochemical features of the groups with and without MACCE are given in Table 4. The mean age of MACCE (+) group was higher (p<0.01). DM and prior MI ratio were higher in MACCE (+) group (p:0.03; p<0.01, respectively). EF ratios and Hb levels were lower in the MACCE (+) group. (p<0.01; p<0.01, respectively). In addition, baseline creatinine and delta creatinine levels were higher in the MACCE(+) group. (p<0.01; p<0.01, respectively).

Figure 2 shows the ratio of MACCE throughout long-term follow-up in all the groups.

The Cox-proportional regression model's results are demonstrated in Table 5. A multivariate analysis model was made in those who developed MACCE using age, hypertension, male gender, baseline glucose values, systolic blood pressure, active smoking, serum hemoglobin levels, ejection fraction and Δ cre ratio. Age (HR: 1,03,CI 95% [1,02-1,05], P < 0.01) Ejection fraction (HR: 0.96,CI 95% [0,94-0,98], P < 0.01) , serum hemoglobin levels (HR: 0.87,CI 95% [0,79-0,96], P < 0.01) and Δ cre ratio (HR: 1.01,CI 95% [1,01-1,02], P < 0.01) were independent predictors of the MACCE.

Table 1. Baseline clinical, demographic and angiographic characteristics of study population

	Δ cre < %10 (n=512)	%10 ≤Δcre < %25 (=210)	%25 ≤ Δ cre (n=162)	p value
Age (year)	60.2±12.0	60.5±11.4	62.4±11.5	0.10
Male	374(73.0)	163(77.6)	106(65.4)	**0.03**
Hypertension	290(56.6)	119(56.7)	107(66.0)	0.09
Diabetes Mellitus	166(32.4)	74(35.2)	83(51.2)	< 0.01
Hyperlipidemia	221(43.2)	88(41.9)	70(43.2)	0.94
Active smoking	253(49.4)	114(54.3)	60(37.0)	**<0.01**
Previous MI	131(25.6)	55(26.2)	43(26.5)	0.94
Previous PCI	140(27.3)	66(31.4)	49(30.2)	0.49
Previous CABG	74(14.5)	24(11.4)	27(16.7)	0.33
Ejection fraction (%)	52.2±9.2	52.1±9.3	49.6±10.7	**0.01**
Length of hospital stay (day)	6.68±4.8	6.91±4.8	8.25±6.2	**<0.01**
Heart rate (bpm)	79.1±17.8	76.42±13.5	80.1±14.6	0.07
Systolic blood pressure (mmHg)	134.1±21.7	137.0±22.8	140.0±25.1	**<0.01**
ST depression	223(43.6)	85(40.5)	74(45.7)	0.58
GRACE score>140	138.6±35.4	133.7±33.6	141.6±35.8	0.09
No. of involved coronaries				
Non significant CAD	60(11.7)	14(6.7)	10(6.2)	
1 vessel	195(38.1)	86(41.0)	52(32.1)	**0.04**
2 vessel	124(24.2)	60(28.6)	48(29.6)	
3 vessel	133(26.0)	50(23.8)	52(32.1)	
Contrast volume (ml)	207.8±117.3	200.7±112.5	210.6±137.3	0.69
Tirofiban usage	17(4.2)	5(3.1)	6(4.5)	0.77

CABG, coronary artery bypass grafting; MI, myocardial infarction; PCI, percutaneous coronary intervention; CAD; coronary artery disease

Table 2. Baseline biochemical characteristics of study population

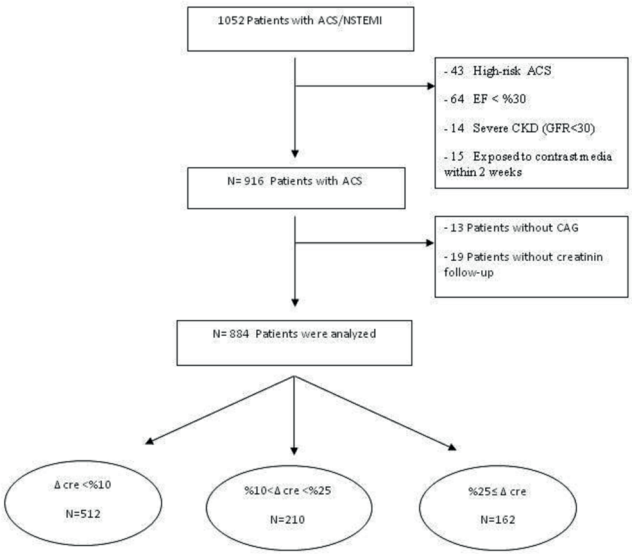
	Δ cre < %10 (n=512)	%10 ≤Δcre < %25 (=210)	%25 ≤ Δ cre (n=162)	p value
Haemoglobin (mg/dl)	13.4±1.9	13.5±1.8	12.9±1.9	**<0.01**
Leukocytes (/mm^3)	9618±3269	9252±2655	9686±3074	0.28
Platelets (x10^3/mm^3)	243.9±72.5	240.1±63.5	261.5±88.4	**0.01**
Total cholesterol (mg/dl)	187.6±46.8	182.5±40.2	190.9±56.7	0.21
LDL cholesterol (mg/dl)	123.5±40.5	120.1±34.9	123.4±44.7	0.58
HDL cholesterol (mg/dl)	37.6±10.6	39.2±11.4	38.1±10.8	0.18
Triglycerides (mg/dl)	182.8±128.3	165.7±107.5	199.9±179.6	**0.05**
Baseline glucose (mg/dL)	126.5±54.5	123.7±49.1	140.4±60.5	**<0.01**
HbA1c (%)	6.8±1.6	6.9±1.7	7.4±1.9	**0.01**
Baseline creatinine (mg/dl)	1.0±0.5	0.9±0.3	1.0±0.8	**0.02**
Max. creatinine (mg/dl)	1.0±0.6	1.1±0.3	1.6±1.3	**< 0.01**
Baseline eGFR (ml/min/m^2)	81.7±24.6	92.8±25.6	88.3±29.9	**< 0.01**
Max. eGFR (ml/min/m^2)	80.6±24.1	78.4±21.8	59.1±24.6	**< 0.01**
Δ creatinine (mg/dl)	0.01±0.06	0.15±0.05	0.55±0.72	**< 0.01**

eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein;

Table 3. Primary clinical end-points at long-term follow-up

	Δ cre < %10 (n=512)	%10 ≤Δcre < %25 (=210)	%25 ≤ Δ cre (n=162)	p value
Primary end point (Death/MI/CVA)	67(13.1)	19(9.0)	37(22.8)	**< 0.01**
Death	22(4.3)	6(2.9)	21(13.0)	**< 0.01**
MI	50(9.8)	14(6.7)	19(11.7)	0.23
CVA	0	0	2	**0.02**

CVA, cerebrovascular accident; MI, myocardial infarction



ACS; acute coronary syndrome, NSTEMI; non–ST-segment elevation myocardial infarction , EF;ejection fraction, CKD; chronic kidney disease, CAG; coronary angiography, GFR; glomerular filtration rate

Figure 1. Trial Flow Chart

Table 4. Baseline Clinical and Biochemical Characteristics of Patients According to The Development of MACCE

	MACCE(-) (n=761)	MACCE(+) (N=123)	p value
Age (year)	60.6±10	67.5±11	**<0.01**
Male, n(%)	558 (73.3)	80 (65.1)	0.09
Hypertension, n(%)	430 (56.5)	79 (64.2)	0.10
Diabetes Mellitus, n(%)	267 (35.1)	56 (45.5)	0.03
Hyperlipidemia, n(%)	326 (42.8)	53 (43.1)	0.96
Active smoking, n(%)	375 (49.3)	50 (40.6)	0.09
Previous MI, n(%)	184 (24.2)	45 (36.6)	**<0.01**
Previous PCI, n(%)	213 (28.0)	42 (34.1)	0.16
Ejection fraction (%)	52.9±8	45.3±11	**<0.01**
Systolic blood pressure (mmHg)	138.8±24	136.7±20	0.10
GRACE score>140, n(%)	325(42.9)	79(64.2)	**<0.01**
Contrast volume (ml)	208.1±108	204.6±109	0.30
Haemoglobin (mg/dl)	13.4±1.8	12.4±2.1	**<0.01**
Leukocytes (/mm^3)	9719±3068	9182±2842.5	0.96
Platelets (x10^3/mm^3)	246.9±74.3	265.6±97.1	0.63
Total cholesterol (mg/dl)	189.4±50.1	182.7±40.1	0.20
LDL cholesterol (mg/dl)	124.5±41.7	120.7±38.6	0.06
Baseline glucose (mg/dL)	134.1±58.9	146.7±69.6	0.06
HbA1c (%)	6.89±1.6	7.36±1.9	0.06
Baseline creatinine (mg/dl)	0.96±0.4	1.16±0.7	**<0.01**
Max. creatinine (mg/dl)	1.06±0.5	1.51±1.2	**< 0.01**
Δ creatinine (mg/dl)	0.11±0.2	0.34±0.8	**< 0.01**

eGFR: estimated glomerular filtration rate, HbA1c: hemoglobin A1c, HDL: high-density lipoprotein, LDL: low-density lipoprotein, MI: myocardial infarction, PCI: percutaneous coronary intervention

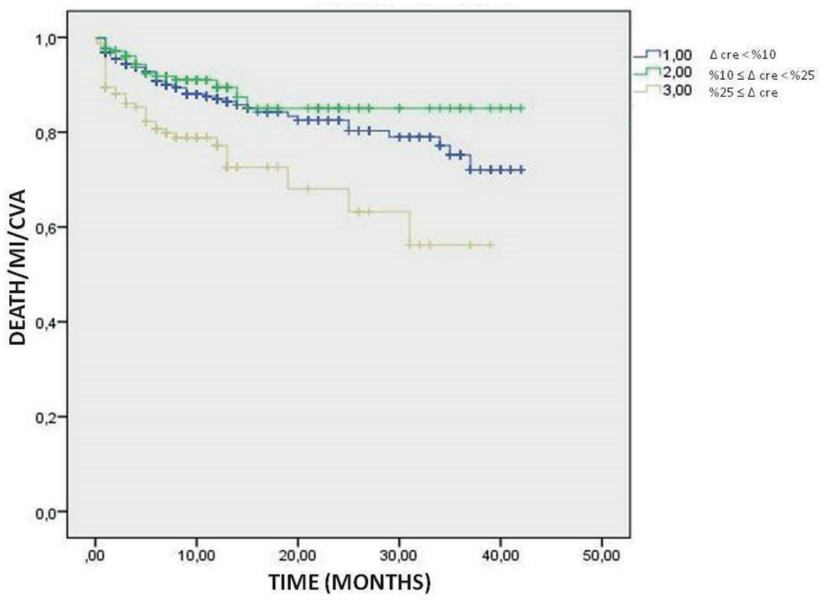


Figure 2. Kaplan-Maier curve for MACCE at long term follow up in all the groups

Table 5. Multivariable predictors of MACCE

	HR	%95 CI	p value
Age	1.03	1.02-1.05	**<0.01**
Hypertension	0.76	0.50-1.14	0.18
Male	1.12	0.71-1.75	0.64
Baseline glucose	1.01	0.99-1.01	0.09
Systolic blood pressure	1.01	0.99-1.01	0.62
Active smoking	0.95	0.63-1.43	0.81
Hemoglobin	0.87	0.79-0.96	**<0.01**
Ejection fraction (%)	0.96	0.94-0.98	**<0.01**
Δ SCrea % group	1.01	1.01-1.02	**<0.01**

CI: Confidence interval, HR: Hazard ratio, SCrea: serum creatinine

DISCUSSION

We found that patients with increase of creatinine ≥ 25% had significantly higher primary endpoints, compared with patients in the other groups. There was no meaningful difference in MACCE among the groups in which the increase rate below 25%. In addition, age, ratio of increase in SCrea, baseline HgB level and EF ratio were detached predictors of the MACCE.

Different mechanisms play a role to impairment of kidney functions after contrast media exposure. Especially; vasoconstrictor agents such as angiotensin, anti-diuretic hormone(ADH), adenosine and endotheline; increased inflammation of the kidney interstitial area (tubular blockage and complement system activation); renal medullary ischemia owing to decreased nitric oxide(NO) synthesis; increased release of proinflammatory cytokines due to increased oxidative stress (17-20). In animal models of ACS have demonstrated that inflammation process is not only limited to the regional myocardium but includes the all over the organism (21,22). So, these situations could be clarify the elevated rate of CI-AKI development in ACS patients. In their study (n=860), Watanabe et al. (23) reported that even a 0.1 mg / dL rise in SCrea values predicted CI-AKI development with 73% sensitivity and 86% specificity in post-PCI daily follow-ups. However, approximately 90% of the study participants in had stable coronary artery disease. Kishore et al (6) compared the 4 definitions previously used for the CI-AKI in a study of 985 patients, and found that increases in creatinine ≥ 0.5mg/dl and ≥ 25% indicated concurrently the most accurate primary end points. In that study, approximately 87% of the participants had stable coronary artery disease. Nicklaus et al.(24] compared two different groups of PCI patients with conventional (SCrea increase ≥ 0.5mg/dl) and new CI-AKI (SCrea increase ≥ 0.5mg/dl or increase ratio ≥ 25%) definitions, and reported that the conventional CI-AKI definition was better than the primary end points. Approximately 50% of the patients included in this study were ACS patients. One of the striking results of the study was that the amount of increase in creatinine between 0.35-0.45mg/dl in the sensitivity and specificity optimization (calculated using the Youden index [j]) was similar. As a result of this work, it was recommended that the current CI-AKI definition should be worked on to the extent that the values are further reduced. In our prospective study, all patients had NSTEMI. Similar to data reported in the literature, our results showed that ≥ 25% rise of SCrea values ratio was related with a significant increase in MACCE relative to lower rates of SCrea increase.

In a follow-up study of ACS patients, Uzunhasan et al. (10) found that CI-AKI development and MEHRAN scores were independent predictors of the development of MACCE. In another single-centered study of ACS patients, Farhan and colleagues (25) reported that heart failure, increase in creatinine level, and hypotension were predictors of in-hospital mortality. Similarly, increases in creatinine, low ejection fraction (EF), age and anemia significantly predicted MACCE in our study.

Altogether, there are three fundamental outcomes from our work. First, we found that the current CI-AKI definition can still be reliably used in ACS patients and it is related with an increase in mortality. Second, our study that may help researchers to assess future increases in lower levels and further question the CI-AKI definition. Third, we hope that our research will raise awareness about the need to be more cautious in terms of CI-AKI in ACS patients who are already alone in high mortality.

Our study also has some limitations. It was a restricted usage of new generation drug eluting stent and new anti-aggregants.

CONCLUSION

using an increase rate of ≥25% creatinine as the definition for CI-AKI is more reliable for primary end points in patients with NSTEMI than the increase rate of creatinine in lower levels. We also found that higher rates of SCrea are independent predictors of mortality. Progressive deleterious in renal function in ACS patients has serious adverse consequences. Therefore, we think that studies seeking to update CI-AKI definition in ACS patients will continue.

Conflict of interest : The authors declare that they have no competing interest.

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Ethical approval: Ethics committee approval was received for this study from the ethics committee of Cerrahpaşa Medical Faculty. Protocol no: 59491012-604.01.02

REFERENCES

1. 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;44:1393-9.

2. McCullough PA, Adam A, Becker CR, et al. CIN Consensus Working Panel. Epidemiology and prognostic implications of contrast-induced nephropathy. Am J Cardiol 200618;98:5-13.
3. Kidney Disease: Improving Global Outcomes (KDIGO) AKI Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Inter Suppl 2012;2:1–138.
4. Morcos SK. Contrast medium-induced nephrotoxicity. In: Dawson P, Cosgrove DO, Grainger RG, editors. Textbook of Contrast Media. Oxford, England: Isis Medical Media Ltd 1999:135-48.
5. Nikolsky E, Aymong ED, Dangas G, et al. Radio contrast nephropathy: identifying the high-risk patient and the implications of exacerbating renal function. Rev Cardiovasc Med 2003;4(suppl 1):7-14.
6. Harjai KJ, Raizada A, Shenoy C, et al. A comparison of contemporary definitions of contrast nephropathy in patients undergoing percutaneous coronary intervention and a proposal for a novel nephropathy grading system. Am J Cardiol 2008;101:812-9.
7. Leoncini M, Toso A, Maioli A, et al. Early high-dose rosuvastatin and cardioprotection in the protective effect of rosuvastatin and anti- platelet therapy on contrast-induced acute kidney injury and myocardial damage in patients with acute coronary syndrome (PRATO-ACS) study. Am Heart J 2014;168:792–7.
8. Ruparelia N, Digby JE, Jefferson A, et al. Myocardial infarction causes inflammation and leukocyte recruitment at remote sites in the myocardium and in the renal glomerulus. Inflamm Res 2013;62:515–25.
9. Narula A, Mehran R, Weisz G, et al. Contrast-induced acute kidney injury after primary percutaneous coronary intervention: results from the HORIZONS-AMI substudy. Eur Heart J 2014;35:1533-40.
10. Uzunhasan I, Yildiz A, Arslan S, et al. Contrast-Induced Acute Kidney Injury Is Associated With Long-Term Adverse Events in Patients With Acute Coronary syndrome. Angiology 2017;68:621-6.
11. Abaci O, Arat Ozkan A, Kocas C, et al. Impact of Rosuvastatin on contrast-induced acute kidney injury in patients at high risk for nephropathy undergoing elective angiography. Am J Cardiol 2015;115:867-71.
12. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract 2012;120:c179-84.
13. Mehta RL, Kellum JA, Shah SV, et al. Acute Kidney Injury Network. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007;11:R31.
14. Thygesen K, Alpert JS, Jaffe AS, et al. Joint ESC/ ACCF/AHA/WHF task force for third universal definition of myocardial infarction. J Am Coll Cardiol 2012;60:1581-98
15. Levey AS, Coresh J, Greene T, et al. Chronic kidney disease epidemiology collaboration. expressing the modification of diet in renal disease study equation for estimating glomerular filtration rate with standardized serum creatinine values. Clin Chem 2007;53:766-72.
16. Levine GN, Bates ER, Blankenship JC, et al. ACCF; AHA; SCAI. 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention: executive summary: a report of the American college of cardiology foundation/american heart association task force on practice guidelines and the society for cardiovascular angiography and interventions. Catheter Cardiovasc Interv 2012;79:453-95.
17. McCullough PA. Contrast-induced acute kidney injury. J Am Coll Cardiol. 2008;51:1419-28.
18. Tumlin J, Stacul F, Adam A, et al. CIN Consensus working panel. Pathophysiology of contrast-induced nephropathy. Am J Cardiol 2006;98:14-20.
19. James MT, Ghali WA, Knudtson ML, et al. Alberta provincial project for outcome assessment in coronary heart disease (APPROACH) Investigators. Associations between acute kidney injury and cardiovascular and renal outcomes after coronary angiography. Circulation 2011;123:409-16.
20. Abaci O, Harmankaya O, Kocas B, et al. Long-term follow-up of patients at high risk for nephropathy after contrast exposure. Angiology 2015;66:514-8.
21. Brunetti ND, Correale M, De Gennaro L, et al. Blunted inflammatory response in STEMI patients timely reperfused. J Cardiovasc Med (Hagerstown). 2014;15:48-52.
22. Ruparelia N, Digby JE, Jefferson A, et al. Myocardial infarction causes inflammation and leukocyte recruitment at remote sites in the myocardium and in the renal glomerulus. Inflamm Res 2013;62:515-25.
23. Watanabe M, Saito Y, Aonuma K, et al. CINC-J study investigators. Prediction of contrast-induced nephropathy by the serum creatinine level on the day following cardiac catheterization. J Cardiol 2016;68:412-418.
24. Slocum NK, Grossman PM, Moscucci M, et al. The changing definition of contrast-induced nephropathy and its clinical implications: insights from the Blue Cross Blue Shield of Michigan Cardiovascular Consortium (BMC2). Am Heart J 2012;163:829-34.
25. Farhan S, Vogel B, Tentzeris I, et al. Contrast induced acute kidney injury in acute coronary syndrome patients: A single centre experience. Eur Heart J Acute Cardiovasc Care 2016;5:55-61.