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Original article

Is atrial fibrillation a risk factor for contrast-induced nephropathy in patients with ST-elevation myocardial infarction?

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ABSTRACT

Background: Contrast-induced nephropathy (CIN) is an iatrogenic problem in patients with ST-elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI). Atrial fibrillation (AF) may also contribute to impaired kidney function. Several factors may contribute to the development of CIN. In patients with STEMI, concomitant AF is associated with higher in-hospital/follow-up mortality and morbidity. Therefore, we aimed to investigate the relationship between AF and CIN developments.

Methods: In this study, 650 consecutive STEMI patients treated with PPCI were enrolled. Patients with AF at admission who did not achieve a sinus rhythm during 48 h after hospitalization were defined as AF patients. CIN was defined by an increase in serum creatinine by >25% or 0.5 mg/dL within 72 h following contrast media exposure.

Results: Our patients were divided into two groups based on whether they had AF, and although warfarin usage was different, the other parameters were similar between the groups. When our patients were grouped according to CIN development [group 1: CIN (+), group 2: CIN (−)], creatinine levels prior to PPCI ($p = 0.020$), estimated glomerular filtration rate (eGFR) prior to PPCI ($p < 0.001$), left ventricular ejection fraction (LVEF) ($p = 0.011$), AF ($p < 0.001$), and warfarin usage ($p = 0.016$) were different between the two groups. We also performed multivariate logistic regression analyses and found that AF [odds ratio (OR), 6.945; 95% confidence interval (CI), 2.789–17.293; $p < 0.001$], eGFR (OR, 0.973; 95% CI, 0.957–0.989; $p = 0.001$), and LVEF (OR, 0.963; 95% CI, 0.935–0.991; $p = 0.010$) independently predicted CIN development in patients with STEMI.

Conclusions: The risk factors for CIN are multifactorial and identifying high-risk patients is the most important step for prevention. In addition to traditional risk factors, AF can contribute to CIN development in patients with STEMI.

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Introduction

Acute kidney injury is an important renal problem that may have various causes, including radio-contrast agents. Contrast-induced nephropathy (CIN) is the third most common cause of renal insufficiency in hospitalized patients and a major cause of morbidity and mortality [1]. As a preventable and treatable disease, CIN may also occur in patients with ST-elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI) [2]. Several factors may contribute to the development of CIN, including hypovolemia, the volume of contrast medium given, an impaired baseline estimated glomerular filtration rate (eGFR), and advanced age [3]. To identify high-risk patients in terms of CIN development is one of the main goals of preventive measures.

Atrial fibrillation (AF), the most prevalent type of sustained cardiac arrhythmia, is increasing in incidence. In addition to increasing treatment costs, many epidemiological studies have shown that AF is related to higher morbidity and mortality [4,5]. In patients with acute coronary syndrome (ACS), especially STEMI, concomitant AF is also associated with higher in-hospital and/or follow-up mortality and morbidity. Paradoxically, ACS is an important trigger for AF development [6,7]. Although many contributing factors have been examined so far, whether AF is a risk factor of CIN development has not been evaluated clearly. Therefore, we aimed to evaluate the relationship between AF and CIN development in patients with STEMI undergoing PPCI.

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Methods

In this study, we included 650 consecutive STEMI patients treated with PPCI between March 2013 and January 2015. STEMI was defined according to current guidelines [8]. We excluded patients with a known history of obstructive coronary artery disease, severe valvular heart disease, severe heart failure [left ventricular ejection fraction (LVEF) < 30%], serum creatinine levels of ≥1.5 mg/dL, and a recent history of acute myocardial infarction and/or PCI. Patients defined as Killip class 2–4, those who required an intra-aortic balloon pump, those who did not achieve thrombolysis in myocardial infarction grade 3 flow after PPCI, and stable patients presenting more than 12 h after symptom onset were excluded from the study. Our study was approved by our local ethics committee, and informed consent was obtained from all patients.

The medical history and current medications of the patients were recorded. Hypertension was defined as a blood pressure reading ≥140/90 mmHg or being on treatment with antihypertensive medications. Diabetes mellitus (DM) was defined as a fasting glucose level ≥126 mg/dL or being treated with oral anti-diabetic drugs or insulin. Hyperlipidemia (HPL) was defined according to current guidelines [9]. Anemia was defined as a baseline hematocrit <39% for males and <36% for females. Smokers were defined as current tobacco users or patients who had quit smoking less than 1 month prior to the procedure. Severe heart failure was identified by transthoracic echocardiography and defined as an LVEF < 30% using the modified Simpson's method.

Venous blood was sampled from a peripheral vein prior to PPCI and then daily during hospitalization. Blood samples were drawn into standardized tubes and assayed within 30 min using routine laboratory techniques. Hematological measurements were performed using the aid of an XT-2000i analyzer (Sysmex America Inc., Long Grove, IL, USA). Routine biochemical measurements [e.g. creatinine (Cr)] were made using an automated biochemistry analyzer (Abbott Aeroset, Minneapolis, MN, USA).

Patients with no P wave and irregular R–R intervals on surface electrocardiogram were defined as having AF. Patients with AF at admission and who could not achieve a sinus rhythm during 48 h after hospitalization were defined as AF patients [10].

CIN was defined by the increase in serum creatinine by >25% or 0.5 mg/dL within 72 h following contrast media exposure [11,12]. The eGFR was determined prior to each procedure using the Modification of Diet in Renal Disease formula, but in this calculation we used body surface area instead of the assumed value of 1.73 m^2 to properly evaluate influence of contrast medium on renal function [13].

PPCI was performed by experienced interventional cardiologists using a femoral or radial approach according to current guidelines [8] with a 6- or 7-Fr sheath and a 6- or 7-Fr guiding catheter. Before PPCI, 300 mg oral acetylsalicylic acid, 600 mg oral clopidogrel, and 100 IU/kg intravenous unfractionated heparin (to achieve an activated clotting time >250 s) were administered to all patients. During PPCI, the stent diameter, stent length, stent type, tirofiban use, and pre- and post-dilatation decisions were left to the physician's discretion.

All patients received low-osmolarity contrast medium (containing iohexol) during PPCI, and the volume of given contrast medium was recorded. After PPCI, transthoracic echocardiography was performed at the earliest possible time (within 12 h). All echocardiographic measurements were performed using a Vivid 7 GE Medical System (Horten, Norway) with a 3.5-MHz transducer.

Statistical analysis

All analyses were performed using SPSS version 19 for Windows (SPSS Inc., Chicago, IL, USA). Numerical variables are presented as means ± standard deviations, and nominal variables as percentages. All variables were subjected to Kolmogorov–Smirnov testing to determine whether they were normally distributed. The independent samples *t*-test was used to compare the values of continuous variables between the two groups. To evaluate the effects of various factors on CIN development, we performed multivariate regression analyses using backward logistic regression method. The model was adjusted using various candidate factors, including height, weight, number of diseased arteries, white blood cell counts, platelet counts, eGFR, baseline creatinine level, LVEF, atrial fibrillation, warfarin usage, age, DM, statin usage, angiotensin-converting enzyme inhibitor/angiotensin receptor blockers usage, oral anti-diabetics usage, and beta-blocker usage. Coefficients with 95% confidence intervals (CIs) were presented. A *p*-value <0.05 was considered significant.

Results

In the present study, we evaluated 650 patients with STEMI who underwent PPCI. Firstly, our study patients were divided into two groups based on presence of AF (patients with AF and without AF). Although warfarin usage was different, the other characteristics were similar between the groups (Table 1).

Table 1
Baseline demographic, angiographic, and clinical characteristics of patient groups based on presence of atrial fibrillation.

Variables	Patients with AF $n=26$	Patients without AF $n=624$	*p*-Value
Age (years)	59.9 ± 11.9	61.2 ± 10.8	0.560
Sex (female), *n* (%)	8 (30.8)	236 (37.8)	0.468
DM, *n* (%)	3 (11.5)	59 (9.5)	0.724
HT, *n* (%)	3 (11.5)	86 (13.8)	0.745
HPL, *n* (%)	20 (76.9)	398 (63.8)	0.171
Smoking, *n* (%)	8 (30.8)	191 (30.6)	0.986
Previous medications			
ASA, *n* (%)	3 (11.5)	50 (8.0)	0.521
Statins, *n* (%)	4 (15.4)	137 (22.0)	0.427
ACEI/ARB, *n* (%)	2 (7.7)	67 (10.7)	0.620
OAD, *n* (%)	2 (7.7)	40 (6.4)	0.795
Insulin, *n* (%)	1 (3.8)	12 (1.9)	0.493
CCB, *n* (%)	2 (7.7)	33 (5.3)	0.597
BB, *n* (%)	1 (3.8)	21 (3.4)	0.895
Clopidogrel, *n* (%)	0 (0.0)	27 (4.3)	0.279
Warfarin, *n* (%)	15 (57.7)	0 (0.0)	**<0.001**
LVEF (%)	46.3 ± 10.3	46.0 ± 9.0	0.867
Infarct localization (anterior), *n* (%)	9 (34.6)	196 (31.4)	0.731
Creatinine level (mg/dL) prior to PPCI	0.9 ± 0.2	0.9 ± 0.2	0.249
GFR (mL/min/1.73 m^2) prior to PPCI	95.6 ± 25.6	92.5 ± 17.0	0.375
Glucose, mg/dL	141.7 ± 71.5	142.3 ± 64.0	0.964
HDL, mg/dL	35.7 ± 9.1	38.7 ± 9.2	0.104
LDL, mg/dL	136.1 ± 30.2	128.0 ± 31.3	0.201
TG, mg/dL	149.4 ± 69.7	169.4 ± 103.7	0.331
PLT count ($\times 10^3/mm^3$)	221.1 ± 68.7	209.0 ± 74.1	0.415
WBC ($\times 10^3/mm^3$)	12.8 ± 3.2	11.4 ± 4.7	0.137
Hematocrit (%)	42.0 ± 5.6	40.4 ± 5.4	0.137
Single-vessel disease, *n* (%)	9 (34.6)	298 (47.8)	0.189
Two-vessel disease, *n* (%)	14 (53.8)	234 (37.5)	0.093
Three-vessel disease, *n* (%)	4 (15.4)	92 (14.7)	0.928
Contrast volume (mL)	86.3 ± 14.8	84.0 ± 20.3	0.564
Weight (kg)	84.8 ± 15.4	84.2 ± 14.4	0.840
Height (cm)	165.2 ± 10.0	163.1 ± 9.7	0.269

AF, atrial fibrillation; DM, diabetes mellitus; HT, hypertension, HPL, hyperlipidemia; ASA, acetylsalicylic acid; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; OAD, oral anti-diabetics; CCB, calcium channel blockers; BB, beta-blockers; LVEF, left ventricular ejection fraction; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; PPCI, primary percutaneous coronary intervention; GFR, glomerular filtration rate; PLT, platelets; WBC, white blood cell.

The baseline clinic, angiographic, and laboratory characteristics of the patient groups in terms of CIN development are presented in Table 2. The creatinine levels prior to PPCI ($p = 0.020$), eGFRs prior to PPCI ($p < 0.001$), LVEF ($p = 0.011$), AF ($p < 0.001$), and warfarin usage ($p = 0.016$) were different between the two groups. The other parameters did not differ significantly between the groups ($p > 0.05$ for all).

Predictors of CIN development are shown in Table 3. According to our multivariate logistic regression analyses, presence of AF (OR = 6.801; 95% CI = 2.771–16.697; $p < 0.001$), eGFR (OR = 0.975; 95% CI = 0.958–0.991; $p = 0.003$), and LVEF (OR = 0.963; 95% CI = 0.936–0.992; $p = 0.011$) were found to be independent predictors of CIN development in patients with STEMI who underwent PPCI.

Table 2
Baseline demographic, angiographic, and clinical characteristics of patient groups based on CIN development.

Variables	Group 1 CIN (+) $n = 59$	Group 2 CIN (−) $n = 591$	p-Value
Age (years)	63.3 ± 9.2	60.9 ± 10.9	0.106
Sex (female), n (%)	25 (42.4)	219 (37.1)	0.422
DM, n (%)	9 (15.3)	53 (9.0)	0.117
HT, n (%)	10 (16.9)	79 (13.4)	0.446
HPL, n (%)	41 (69.5)	377 (63.8)	0.384
Smoking, n (%)	21 (35.6)	178 (30.1)	0.385
Previous medications			
ASA, n (%)	7 (11.9)	46 (7.8)	0.275
Statins, n (%)	9 (15.3)	132 (22.3)	0.209
ACEI/ARB, n (%)	9 (15.3)	60 (10.2)	0.228
OAD, n (%)	6 (10.2)	36 (6.1)	0.225
Insulin, n (%)	1 (1.7)	12 (2.0)	0.861
CCB, n (%)	4 (6.8)	31 (5.2)	0.622
BB, n (%)	4 (6.8)	18 (3.0)	0.131
Clopidogrel, n (%)	3 (5.1)	24 (4.1)	0.708
Warfarin, n (%)	4 (6.8)	11 (1.9)	**0.016**
Atrial fibrillation	9 (15.3)	17 (2.9)	**<0.001**
Infarct localization (anterior), n (%)	20 (33.9)	185 (31.3)	0.683
LVEF (%)	43.2 ± 10.9	46.3 ± 8.8	**0.011**
Creatinine level (mg/dL) prior to PPCI	0.9 ± 0.2	0.9 ± 0.2	**0.020**
GFR (mL/min/m^2) prior to PPCI	76.8 ± 19.1	83.6 ± 17.0	**<0.001**
Glucose, mg/dL	138.9 ± 59.4	142.6 ± 64.7	0.673
HDL, mg/dL	38.1 ± 8.4	38.6 ± 9.3	0.687
LDL, mg/dL	130.6 ± 32.9	128.1 ± 31.1	0.566
TG, mg/dL	178.7 ± 93.4	167.6 ± 103.5	0.429
PLT ($\times 10^3$/mm^3)	225.6 ± 66.7	207.9 ± 74.4	0.079
WBC ($\times 10^3$/mm^3)	12.6 ± 8.8	11.3 ± 4.0	0.053
Hematocrit (%)	40.1 ± 6.6	40.5 ± 5.2	0.626
Single-vessel disease, n (%)	23 (39.0)	284 (48.1)	0.184
Two-vessel disease, n (%)	26 (44.1)	222 (37.6)	0.327
Three-vessel disease, n (%)	11 (18.6)	85 (14.4)	0.380
Contrast medium volume (mL)	86.4 ± 15.9	83.8 ± 20.4	0.341
Weight (kg)	83.8 ± 14.4	84.3 ± 14.5	0.800
Height (cm)	163.9 ± 10.2	163.1 ± 9.7	0.542

CIN, contrast-induced nephropathy; DM, diabetes mellitus; HT, hypertension; HPL, hyperlipidemia; ASA, acetylsalicylic acid; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; OAD, oral anti-diabetics; CCB, calcium channel blockers; BB, beta-blockers; LVEF, left ventricular ejection fraction; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; PPCI, primary percutaneous coronary intervention; GFR, glomerular filtration rate; PLT, platelets; WBC, white blood cell.

Table 3
The predictors of the development of CIN in the multivariable logistic regression analyses.

Parameters	B	OR	95% CI	p-value
Atrial fibrillation	1.917	6.801	2.771–16.697	<0.001
GFR (mL/min/m^2)	−0.026	0.975	0.958–0.991	0.003
LVEF (%)	−0.037	0.963	0.936–0.992	0.011

CIN, contrast-induced nephropathy; LVEF, left ventricular ejection fraction; GFR, glomerular filtration rate.

Discussion

Our present study is the first evaluating the relationship between AF and CIN development in patients with STEMI who underwent PPCI. We found that the presence of AF was an independent risk factor for the development of CIN in patients with STEMI. We also found that LVEF and baseline eGFR were other independent predictors of CIN development in STEMI patients.

CIN generally develops within 48–72 h after contrast media exposure and the incidence of CIN varies from 6.4% to 27.7% depending on the definition criteria [14,15]. Marenzi et al. observed CIN in 19% of their study patients [16], but our study patients had lower CIN incidence (9% of all groups). Patients who had high risk (such as patients with Killip class 2–4, required an intra-aortic balloon pump, and creatinine levels ≥1.5 mg/dL) in terms of CIN development were excluded; we believe that the lower CIN incidence in our study may be related to these exclusion criteria.

CIN can be considered as a multifactorial disease and many risk factors have been proposed to explain the development of CIN, including baseline renal insufficiency, heart failure, DM, and myocardial infarction [17]. Our study results showed that DM and baseline creatinine levels did not predict CIN development. We think that the relatively small size of our patient population, lack of data about DM duration/glycemic control status, and lack of data about creatinine levels after 72 h following PPCI may be related to these conflicting results. In accordance with previous studies [16–18], eGFR, which is a more sensitive parameter than serum creatinine levels in terms of baseline renal function, and LVEF were shown to be the independent predictors of CIN development.

It is well known that some patients with related risk factors including heart failure and acute myocardial infarction have increased incidence of CIN development via vasoconstriction, renal medullar hypoxia tubular obstruction, and higher inflammatory status [14,17,18]. Although many risk factors have been identified in the development of CIN, the relationship between CIN development and presence of AF has not been clearly evaluated. However, it is the most prevalent type of sustained arrhythmia; AF has an increasing incidence due to aging and may also complicate ACS management [19]. In patients with STEMI, AF is associated not only with an increased risk of stroke but also with left ventricular dysfunction and other negative outcomes [20]. AF and renal function have bi-directional relations. It is well known that chronic kidney disease increases the risk of developing AF; however, some studies have shown that the presence of AF may contribute to impaired kidney function (e.g. proteinuria) [21–23].

Some pathophysiological mechanisms may explain the contribution of AF to contrast-induced acute kidney injury development. AF is related to some adverse hemodynamic effects due to rapid ventricular rates, irregular heart beat, loss of atrio-ventricular synchrony, and atrial contraction [24]. These factors may lead to improved cardiac output even if LVEF is normal and decreased cardiac output can contribute to CIN development in patients with AF who underwent PPCI. Patients with AF have increased atrial expression of angiotensin-converting enzyme inhibitor receptor levels in the atrium and activation of the renin-angiotensin-aldosterone (RAS) cascade is related to progression of renal dysfunction. We think that activated RAS cascade may also contribute to CIN development in patients with AF [25,26]. Patients with AF have higher inflammatory status related to fibrotic changes in myocardium and kidney. These fibrotic changes could be associated with increased sensitivity to contrast agent toxicity

[26–28]. In addition to these mechanisms, patients with AF might have renal micro-emboli, which may contribute to renal impairment. To clarify these pathogenic mechanisms, further studies are needed.

Although our study is the first to evaluate the relationship between AF and CIN development, it has a number of limitations. First, we did not have data about the type and duration of AF such as permanent, persistent, and paroxysmal. Second, we could not evaluate any inflammatory markers which reflect the inflammatory status of study groups. Third, our present study is a single-center study and we do not have follow-up data.

Conclusions

The pathogenesis of and risk factors for CIN are multifactorial and remain poorly understood. Nevertheless, the determination of an individual's risk profile is the most important step in protection against the development of CIN, even if the patient's baseline renal function is normal. In addition to traditional risk factors, AF can contribute to CIN development in patients with STEMI undergoing PPCI. We believe that additional prospective studies are required to investigate this issue.

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Conflict of interest

The authors declare that there is no conflict of interest.

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