Association Between N-Terminal Pro-Brain Natriuretic Peptide Levels and Contrast-Induced Nephropathy in Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndrome

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ABSTRACT

Background: Contrast-induced nephropathy (CIN) is associated with significantly increased morbidity and mortality after percutaneous coronary intervention (PCI). Patients with acute coronary syndrome (ACS) are at higher risk for CIN. N-terminal pro-brain natriuretic peptide (NT-proBNP) is closely linked to the prognosis as a strong predictor of both short- and long-term mortality in patients with ACS.

Hypothesis: We hypothesized that NT-proBNP levels on admission can predict the development of CIN after PCI for ACS.

Methods: A total of 436 patients (age 62.27 $\pm$ 13.01 years; 64.2% male) with ACS undergoing PCI enrolled in this study. Admission NT-proBNP levels were measured before PCI. Serum creatinine values were measured before and within 72 hours after the administration of contrast agents. Patients were divided into 2 groups: CIN group and no-CIN group. CIN was defined as an increase in serum creatinine level of $\geq$0.5 mg/dL or $\geq$25% above baseline within 72 hours after contrast administration.

Results: CIN developed in 63 patients (14.4%). Baseline NT-proBNP levels were significantly higher in patients who developed CIN compared to those who did not develop CIN (median 774 pg/mL, interquartile range 177.4–2184 vs median 5159 pg/mL, interquartile range 2282–9677, respectively; $P < 0.001$). Multivariate analysis found that NT-proBNP (odds ratio [OR]: 3.448, 95% confidence interval [CI]: 1.394-8.474, $P = 0.007$) and baseline creatinine (OR: 6.052, 95% CI: 1.860-19.686, $P = 0.003$) were independent predictors of CIN.

Conclusions: Admission NT-proBNP level is an independent predictor of the development of CIN after PCI in ACS.

Introduction

Contrast-induced nephropathy (CIN) is a serious complication of invasive cardiovascular procedures. The incidence of CIN is 2% for the general population. However, patients undergoing percutaneous coronary intervention (PCI) are at greater risk, and patients with diabetes or previous renal impairment have a risk of almost 50%.[1,2] Development of CIN after PCI is associated with poor clinical outcomes including prolonged hospitalization, increased costs, increased rates of end-stage renal failure, myocardial infarction, repeat revascularization, and short- and long-term mortality.[3–6] Furthermore, patients with acute coronary syndrome (ACS) have a 3-fold higher risk of developing CIN.[7,8] Because CIN occurs more frequently after urgent PCI in patients with ST-segment elevation myocardial infarction (STEMI) and non-STEMI,[9] objective and rapidly available and reliable markers may be useful for identification of patients at risk of development of CIN.

N-terminal pro-brain natriuretic peptide (NT-proBNP) is synthesized and secreted from the cardiac ventricles in response to increased ventricular wall stress,[10] but myocardial ischemia and infarction may also stimulate its release.[11,12] This marker is closely linked to the prognosis as a strong predictor of both short- and long-term mortality in patients with ACS.[13–16] NT-proBNP is associated with poor hemodynamics, neurohormonal responses, and inflammation in ACS

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patients, all of which play a role in the development of CIN.[17,18]

In the present study, we sought to investigate whether NT-proBNP level on admission is an independent risk factor that predicts the development of CIN in patients with ACS with ST-segment elevation (STE-ACS) and unstable angina/non–ST-segment elevation (NSTE-ACS) to undergo interventional therapy

Methods

Study Population

Between January 2013 and December 2013, a total of 530 consecutive patients (mean age, 62.27 ± 13.01 years; 64.2% male) were identified with acute STE-ACS or NSTE-ACS undergoing emergency PCI. After an evaluation according to inclusion and exclusion criteria, 436 patients were enrolled in our study (Figure 1). Patients with STE at the J point in 2 or more consecutive leads (with the cutoff point being >0.2 mV in leads V1, V2, or V3, and >0.1 mV in the other leads) and elevation of cardiac troponin T level greater than the upper limit of normal were defined as having STE-ACS. Patients with ST-segment depression, T-wave inversion, or no electrocardiographic abnormalities and/or elevation of cardiac troponin T level greater than the upper limit of normal were defined as having NSTE-ACS. We excluded patients receiving long-term peritoneal or hemodialysis treatment, or those who underwent a renal transplantation or received administration of metformin, nonsteroidal anti-inflamamatory drugs, aminoglycosides, or acetylcysteine 1 week before or after PCI. Patients were also excluded if they had intra-aortic balloon pump support before PCI because of cardiogenic shock, cardiac surgery for coronary revascularization, severe chronic heart failure (New York Heart Association class ≥3), and contrast exposure 2 weeks before PCI or died during PCI.

The study protocol was approved by the local ethics committee, and written informed consent was obtained from all participants.

Study Protocol and Definitions

Baseline serum creatinine and NT-proBNP levels were measured before angiography. NT-proBNP measurements were performed in plasma on an Elecsys 2010 analyzer, a commercially available electrochemiluminescent sandwich immunoassay (Elecsys proBNP; Roche Diagnostics, Mannheim, Germany). The lowest and highest detection limits of the assay were at 5 to 35.000 pg/mL. High-sensitivity C-reactive protein (hs-CRP) levels were also measured. Immediately after intervention, all patients underwent hydration with intravenous isotonic saline (0.9%) at a rate of 1 mL/kg/h for 12 hours (or 0.5 mL/kg/h for 12 hours in cases of overt heart failure). Any nephrotoxic medications (ie, metformin, nonsteroidal anti-inflammatory drugs) were suspended on admission. Serum creatinine was also measured at 24, 48, and 72 hours after contrast medium administration. Patients were divided into 2 groups: CIN group and no-CIN group. CIN was defined as an increase in serum creatinine level of ≥0.5 mg/dL or ≥25% above baseline within 72 hours after contrast administration.[19]

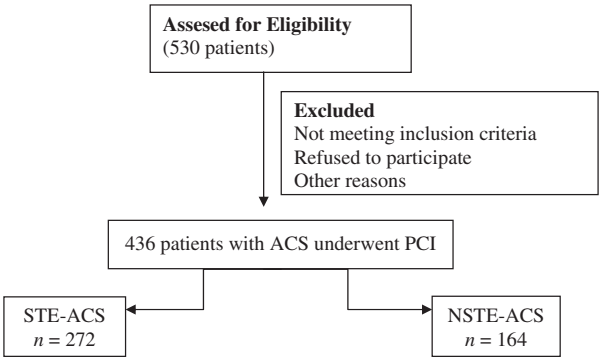

Figure 1. Diagram of the 436 patients who were enrolled in the study after inclusion and exclusion criteria evaluation. Abbreviations: ACS, acute coronary syndrome; NSTE, non–ST-segment elevation; PCI, percutaneous coronary intervention; STE, ST-segment elevation.

High-contrast volume was defined as the administration of a contrast volume of >140 mL.[20] The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation.[21] Left ventricular ejection fraction (LVEF) was measured using the Simpson method according to the recommendations of the American Society of Echocardiography.[22]

Coronary Interventions and Medications

Coronary angiography (CA) (Siemens Axiom Artis zee 2011; Siemens Healthcare, Erlangen, Germany) was performed using the femoral approach according to standard clinical practice. On admission, all patients in the emergency department received a bolus of 5,000 U of intravenous unfractionated heparin, followed by additional intraprocedural boluses to maintain an activated clotting time of 200 to 250 seconds (>300 seconds when tirofiban was not used), acetylsalicylic acid (300 mg orally), and clopidogrel loading dose of 600 mg orally. Nonionic, low-osmolar contrast medium (iohexol, Omnipaque 350 mg/mL; GE Healthcare, Cork, Ireland) was used to visualize the coronary arteries. PCI was performed immediately after diagnostic CA when appropriate. Coronary stenting was performed using standard techniques.[23] Thrombolysis in Myocardial Infarction grade 3 coronary flow in the treated vessel with a residual stenosis <50% was considered successful PCI. The use of glycoprotein IIb/IIIa inhibitor as well as bare-metal or drug-eluting stents was left to the discretion of the interventional cardiologist. Additional use of thrombectomy was recommended depending on thrombus in the infarct-related artery. After CA, all patients continued to take aspirin (100 mg/d orally) indefinitely and clopidogrel (75 mg/d) for at least 12 months.

Statistical Analysis

We decided the sample size of the study by using a program named Power & Sample Size Calculator (Statistical Solutions, Cottage Grove, WI; www.statisticalsolutions.net/pss_calc.php). Statistical analysis was performed with the Statistical Package for the Social Sciences (SPSS) for Windows, version 18.0 (SPSS Inc., Chicago, IL).

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Table 1. Baseline Characteristics of the Study Patients

Variable	No-CIN Group, n = 373, 85.6%	CIN Group, n = 63, 14.4%	*P* Value
Age, y	60.59 ± 12.29	72.22 ± 12.79	<0.001
Male gender, n (%)	241 (64.6)	39 (61.9)	0.679
BMI, kg/m²	27.85 ± 4.53	27.13 ± 4.47	0.354
Systolic blood pressure, mm Hg	129 ± 25	133 ± 32	0.257
Diastolic blood pressure, mm Hg	78 ± 14	78 ± 16	0.907
Hypertension, n (%)	161 (43.2)	35 (55.6)	0.067
Diabetes mellitus, n (%)	119 (31.9)	23 (36.5)	0.471
Smoking, n (%)	176 (47.2)	10 (15.9)	<0.001
Hyperlipidemia, n (%)	110 (29.5)	11 (17.5)	0.128
Prior CABG, n (%)	20 (5.4)	3 (4.8)	0.844
Prior myocardial infarction, n (%)	36 (9.7)	5 (7.9)	0.666
LVEF, %	47 ± 10	40 ± 10	<0.001
LVEF <40%, %	20.7	38.6	0.003
Type of ACS, n (%)			
STE-ACS	234 (62.7)	38 (60.3)	0.714
NSTE-ACS	139 (37.3)	25 (39.7)	
Treatment before admission, %			
ACEI or ARB	28.1	31.3	0.794
Statins	28.3	27.2	0.814

Abbreviations: ACEI, angiotensin converting enzyme inhibitor; ACS, acute coronary syndrome; ARB, angiotensin receptor blocker; BMI, body mass index; CABG, coronary artery bypass graft; CIN, contrast-induced nephropathy; LVEF, left ventricular ejection fraction; NSTE, non–ST-segment elevation; STE, ST-segment elevation.

Table 2. Baseline Biochemical and Hematologic Measurements of Patients

Variable	No-CIN Group, n = 373	CIN Group, n = 63	*P* Value
Serum glucose on admission	158 ± 87	158 ± 78	0.962
HbA1c, %	7.00 ± 1.98	6.97 ± 1.90	0.916
Serum creatinine, mg/dL	1.06 ± 0.27	1.37 ± 0.38	<0.001
eGFR, mL/min per 1.73 m²	71.9 ± 19.8	50.1 ± 17.6	<0.001
White blood cell count, × 10^3/mm³	10.99 ± 3.51	11.28 ± 3.86	0.557
Hemoglobin, g/L	14.11 ± 1.87	12.72 ± 2.09	<0.001
Platelet count, × 10^3/mm³	241 ± 66	247 ± 92	0.492
Mean platelet volume, fL	8.70 ± 1.03	8.89 ± 1.31	0.189
NT-proBNP, pg/mL	774 (177.4–2184)	5159 (2282–9677)	<0.001
Total cholesterol, mg/dL	191 ± 49	176 ± 51	0.030
Triglyceride, mg/dL	131 (35–815)	112 (39–624)	0.454
Low-density lipoprotein, mg/dL	121 ± 42	106 ± 40	0.010
High-density lipoprotein, mg/dL	41 ± 10	42 ± 11	0.765
hs-CRP, mg/L	7.15 ± 3.78	8.48 ± 3.88	0.014
Peak CK-MB, ng/mL	35.6 (0.78–425)	50.4 (2.25–419)	0.318
Peak troponin T, ng/mL	1060 (4.13–10000)	1778 (43.9–10000)	0.029
Serum uric acid, mg/dL	5.49 ± 1.53	6.53 ± 1.76	<0.001

Abbreviations: CIN, contrast-induced nephropathy; CK-MB, creatine kinase-myocardial band; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c; hs-CRP, high-sensitivity C-reactive protein; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Continuous variables were compared using the Student *t* test. In case of non-normal distribution, nonparametric methods were used (Mann–Whitney *U* test). All values are expressed as mean ± standard deviation or median and interquartile range. Models were developed with stepwise techniques and by consideration of potential confounding factors, and of variables that are shown to be statistically significant at univariate analysis. Results of this model were presented as odds ratio (OR) and 95% confidence interval (CI). To identify independent parameters associated with CIN, multivariable logistic regression analysis was used. According to the results of univariate analysis, age, smoking, LVEF, hemoglobin, creatinine, NT-proBNP ≥2149 pg/mL, total cholesterol, low-density lipoprotein (LDL) cholesterol, hs-CRP, uric acid, troponin T, SYNTAX (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) score, presence of multivessel disease (MVD), and presence of chronic total occlusion (CTO) were analyzed with multivariate logistic regression model. The receiver operating characteristic (ROC) analysis was performed to determine the best cutoff value of NT-proBNP, and the sensitivity and specificity at that point were obtained for predicting the development of CIN. *P* values <0.05 were considered statistically significant.

Results

The study population consisted of 436 patients (mean age, 62.27 ± 13.01 years and 64.2% male) who had measurements of baseline serum NT-proBNP and creatinine, followed by additional daily measures of creatinine up to 72 hours. A total of 63 patients (14.4%) developed CIN.

The baseline clinical characteristics of the patient population stratified by CIN are summarized in Table 1. Patients in the CIN group were significantly older than those in the no-CIN group (72.59 ± 12.29 years vs 60.59 ± 12.79

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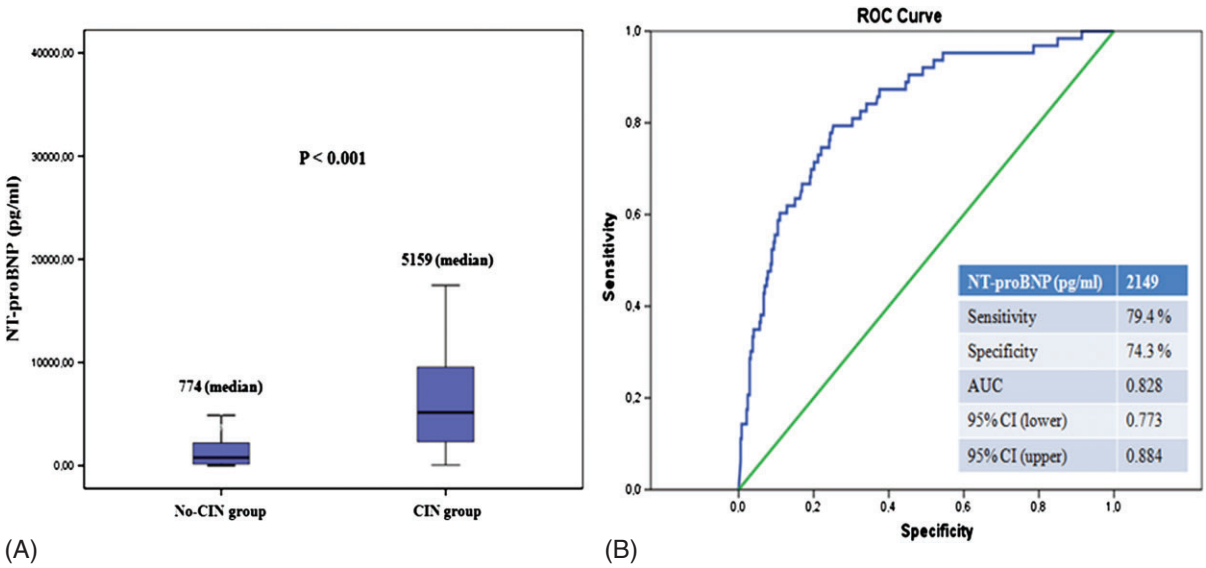

Figure 2. (A) Comparison of serum NT-proBNP levels between groups. (B) The receiver operating characteristic (ROC) curve analysis for serum NT-proBNP levels in predicting of postprocedural development of CIN. AUC = 0.828 (0.773-0.884). Abbreviations: AUC, area under the curve; CI, confidence interval; CIN, contrast-induced nephropathy; NT-proBNP, N-terminal pro-brain natriuretic peptide.

years, respectively, $P < 0.001$) and had a significantly lower proportion of smoking ($P < 0.001$) compared with patients in the no-CIN group. LVEF was also lower in the CIN group ($P < 0.001$). There were no significant differences between the groups regarding gender, hypertension, diabetes mellitus, hyperlipidemia, prior myocardial infarction, prior stroke, prior medications, in-hospital medications, and type of ACS.

The baseline laboratory measurements of the study patients are shown in Table 2. NT-proBNP levels at baseline were significantly higher in patients who developed CIN compared to those who did not (median, 774 pg/mL [interquartile range, 177.4–2184] vs median, 5159 pg/mL [interquartile range, 2282–9677], respectively; $P < 0.001$). NT-proBNP levels according to risk of developing CIN are shown in Table 2 and Figure 2A.

In addition to having elevated NT-proBNP, patients who developed CIN had significantly higher baseline serum creatinine, peak troponin T, and uric acid levels, but significantly lower baseline hemoglobin, total cholesterol, LDL, and eGFR than those in whom CIN did not occur.

Patients with CIN had a higher prevalence of MVD, presence of CTO in nonculprit vessels, and higher SYNTAX score, and lower stent diameter compared with those without CIN. However, the location of the culprit lesion, the contrast volume, procedure duration, and procedure success rate did not significantly differ between patients who developed CIN and those who did not. Angiographic and procedural characteristics of the study patients are listed in Table 3.

The ROC curve analysis of NT-proBNP for predicting CIN is shown in Figure 2B. NT-proBNP ≥2149 pg/mL measured on admission had a 79.4% sensitivity and 74.3% specificity in predicting CIN.

The effects of multiple variables were analyzed with univariate and multivariate logistic regression analyses. According to the results of univariate analysis, age, smoking, LVEF, hemoglobin, creatinine, NT-proBNP ≥2149 pg/mL, total cholesterol, LDL cholesterol, hs-CRP, uric acid, troponin T, SYNTAX score, presence of MVD, and presence of CTO were analyzed with multivariate logistic regression model. At multivariate analyses, NT-proBNP (OR: 3.448, 95% CI: 1.394-8.474, $P = 0.007$) and admission creatinine (OR: 6.052, 95% CI: 1.860-19.686, $P = 0.003$) were still significant independent predictors of the development of CIN after PCI in patients with ACS (Table 4).

Discussion

In the present study, we demonstrated that admission NT-proBNP level is an independent predictor of the development of CIN in ACS patients undergoing PCI. Baseline creatinine level was the other independent predictor of CIN.

CIN after PCI is strongly associated with prolonged hospitalization, increased costs, increased rates of end-stage renal failure, myocardial infarction, repeat revascularization, and early and late mortality.[3–5] Patients who develop CIN and require dialysis after PCI have a 40% in-hospital mortality and 80% 2-year mortality rates.[24] On the other hand, patients with ACS have a 3-fold higher risk of CIN,[3,7,8] and development of CIN is a sign of poor short- and long-term prognosis after STE-ACS and NSTE-ACS despite successful early coronary revascularization.[9,25] Preexisting renal impairment, diabetes, congestive heart failure, advanced age, anemia, use of high-contrast media volume, and reduced intravascular volume are risk factors for the development of CIN.[7,26,27]

Brain natriuretic peptides (BNPs) have a variety of effects, such as diuretic, natriuretic, and hypotensive action,

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Table 3. Angiographic and Procedural Characteristics and Medications of Patients

Variable	No-CIN Group, n = 373	CIN Group, n = 63	*P* Value
Total time of procedure, min	37.49 ± 16.58	40.98 ± 16.09	0.162
Total amount of contrast, mL	166 ± 66	177 ± 78	0.280
High-contrast volume, n (%)	194 (61.0)	32 (62.7)	0.813
Multivessel disease, n (%)	207 (55.5)	48 (76.2)	0.002
Chronic total occlusion, n (%)	65 (17.4)	20 (31.7)	0.008
Syntax score	15.29 ± 8.29	21.49 ± 10.69	<0.001
Culprit vessel, n (%)			
Left main coronary artery	1 (0.3)	0 (0)	0.487
Left anterior descending artery	178 (47.7)	31 (49.2)	
Left circumflex artery	74 (19.8)	12 (19.0)	
Right coronary artery	112 (30)	19 (30.2)	
Saphenous vein graft	8 (2.1)	0 (0)	
Stent implantation, n (%)	349 (93.6)	53 (84.1)	0.010
Total length of stent, mm	24.00 ± 11.15	25.17 ± 11.36	0.479
Stent diameter, mm	3.17 ± 0.43	3.04 ± 0.35	0.037
Procedural success, n (%)	299 (80.2)	44 (69.8)	0.064
In-hospital medications, %			
ACE inhibitor or ARB	71	68.4	0.771
Statin	77.7	72.6	0.371
Tirofiban	36.5	26.2	0.120
β-blocker	86.9	77.4	0.052
Diuretic	22.1	25.1	0.873
Clopidogrel	97.9	95.2	0.211

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; CIN, contrast-induced nephropathy.

along with inhibition of both the sympathetic nervous system and renin-angiotensin-aldosterone system (RAAS) as wells as endothelin secretion.[28,29] They also increases glomerular filtration rate by selectively dilating renal afferent arterioles and constructing renal efferent arterioles.[30] Jarai et al[31] demonstrated a relationship between (BNP) level on admission and development of CIN after primary PCI in STE-ACS patients. Our study includes the whole spectrum of ACS, not only STE-ACS, and we used NT-proBNP, which is a more specific marker than BNP.

The N-terminal portion of pro-BNP appears more stable than BNP and is used as a marker instead of BNP.[32] NT-proBNP is synthesized and secreted by the left and right ventricles in response to increased left ventricular wall stretch[10] and neurohormonal activation,[33] and myocardial ischemia and infarction stimulate its release.[11,12,34] Although natriuretic peptides primarily reflect central hemodynamics, their levels may also reflect a variety of processes including ischemia, inflammation, and oxidative stress.[29,35] Neurohormonal activation is known to increase oxidative stress and inflammation.[36,37] It was shown that NT-proBNP is closely linked to the prognosis as a powerful predictor of both short- and long-term mortality in patients with STEMI and ACS.[13–15,38]

The pathophysiologic mechanisms of CIN is complex, multifactorial, and incompletely understood. Possible mechanisms include intrarenal vasoconstriction, reduced renal blood flow, medullary hypoxia, oxidative stress, inflammation, endothelial dysfunction, and direct tubular epithelial cell injury by contrast media.[39] CIN is a specific type of cardio-renal syndrome.[40] Renal injury starts with hemodynamic effects of myocardial infarction. Abnormal hemodynamic states lead to a decrease in renal blood flow, and activation of immune and neurohormonal systems, such as sympathetic nervous system and RAAS, contribute to medullary hypoxia.[41,42] Direct cytotoxicity to renal tubular cells caused by the contrast agent then occurs. The results of the injury include renal vasoconstriction, impaired vasodilation, medullary hypoxia leading to oxidative stress, and direct tubular injury.[43]

Both hemodynamic impairment and neurohormonal activation involved in the development of CIN are known to stimulate NT-proBNP.[17] Therefore, NT-proBNP is a part of neurohumoral signaling between the heart and kidney,[18] which indicates the presence of pathologic processes underlying the cardio-renal syndrome. As a diuretic, vasodilator, and negative inotrope, NT-proBNP may also have a direct precipitating effect on the development of CIN. NT-proBNP inhibits myocardial contractility by inhibiting sarcoplasmic reticulum Ca+2 ATPase, increasing matrix metalloproteinases, reducing the effects of catecholamines, and increasing the effect of nitric oxide[44,45] Thus, high NT-proBNP levels in ACS may be responsible for systemic vasodilatation and renal hipoperfusion, which in turn potentiates CIN. Furthermore, plasma NT-proBNP is found to be increased in a model of systemic inflammation in healthy men with normal heart function.[46] Therefore, NT-proBNP is accepted as an acute-phase reactant,[47] and some inflammatory cytokines, such as tumor necrosis factor, is known to stimulate NT-proBNP.[48] Thus, NT-proBNP may be an indicator of increased immune response and inflammation in ACS, which play an important role in the development of CIN.

The hyperosmolar extracellular environment caused by radiocontrast agents induces oxidative stress via reactive oxygen species and causes renal tubular cell apoptosis.[49,50] Other factors associated with increased oxidative stress include activation of RAAS and matrix metalloproteinases,

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Table 4. Univariate and Multivariate Logistic Regression Analysis of the Association Between Contrast-Induced Nephropathy and Multiple Parameters

	Univariate Analysis		Multivariate Analysis	
Variable	Odds Ratio (95% CI)	*P* Value	Odds Ratio (95% CI)	*P* Value
Age	1.079 (1.053-1.106)	<0.001	1.018 (0.982-1.056)	0.327
Smoking	0.211 (0.104-0.427)	<0.001	0.473 (0.160-1.333)	0.157
NT-proBNP >2149 pg/mL	11.111 (5.780-21.276)	<0.001	3.448 (1.394-8.474)	0.007
LVEF	0.940 (0.914-0.967)	<0.001	0.972 (0.934-1.013)	0.178
Hemoglobin	0.705 (0.614-0.809)	<0.001	0.911 (0.759-1.095)	0.321
Creatinine	14.017 (6.071-32.363)	<0.001	6.052 (1.860-19.686)	0.003
Uric acid	1.453 (1.238-1.706)	<0.001	1.125 (0.895-1.414)	0.312
hs-CRP	1.105 (1.019-1.198)	0.015	0.933 (0.832-1.046)	0.235
Troponin T	1.066 (1.037-1.106)	0.002	1.022(0.092-1.036)	0.490
Total cholesterol	0.993 (0.987-0.999)	0.030	1.017 (0.995-1.040)	0.130
Low-density lipoprotein	0.990 (0.983-0.998)	0.010	0.980 (0.954-1.006)	0.135
Syntax score	1.077 (1.045-1.110)	<0.001	1.030 (0.981-1.081)	0.233
Multivessel disease	2.564 (1.923-4.793)	0.003	0.665 (0.269-1.645)	0.377
Chronic total occlusion	2.204 (1.217-3.992)	0.009	1.251 (0.504-3.104)	0.629

Abbreviations: CI, confidence interval; hs-CRP, high-sensitivity C-reactive protein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide.

which are associated with increased NT-proBNP levels.[36] Matrix metalloproteinase-9 activity is associated with oxidative stress in patients with ACS.[51] Therefore, NT-proBNP is an indicator of oxidative stress, which participates in the development of CIN.

In our study, MVD, presence of CTO in nonculprit vessels, high SYNTAX scores, and low stent diameters were associated with a higher incidence of CIN. In our opinion, high SYNTAX scores, MVD, and presence of CTO in nonculprit vessels are related to more serious presentations (eg, shock, arrhythmia), longer procedural duration, more contrast media use, higher incidence of no-reflow, and lower LVEF. Thus, the renal vasoconstrictive response and the resulting cortico-medullar hypoxia[42] may be aggravated in patients with high scores. It appears (although not statistically significant) that procedural success was considerably lower in the CIN group as compared to the no-CIN group. We think it may be due to the amount of contrast agent and total time of the procedure in the CIN group compared to the no-CIN group (although not statistically significant).

Greater contrast volume use is associated with greater rates of CIN.[52] However, it was shown that contrast media volume do not have an effect on CIN if iso-osmolar contrast agents and adequate hydration are used.[53] In our study, we did not find any effect of contrast agent volume on CIN. Two factors might play a role in this result. First, we use iso-osmolar agents and adequate hydration for our patients. Second, amount of contrast volume used during the procedure was similar in our study population.

Study Limitations

This study has several limitations. First, we measured NT-proBNP level only once at admission and without correction for potential variability in levels. Second, the follow-up assessment of renal function in our study was 1 to 3 days after PCI; therefore, we might have missed a later increase in serum creatinine in some patients who did not have renal function deterioration within 72 hours of their procedure. This might have resulted in a slight underestimation of CIN. Finally, we did not measure inflammatory indices except for hs-CRP.

Conclusion

Admission NT-proBNP levels may predict the development of CIN after PCI in patients with ACS. Thus, NT-proBNP can be a quick and useful marker for the early estimation of CIN.

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