Effect of Renal Function on Ultrasonic Coronary Plaque Characteristics in Patients With Acute Myocardial Infarction

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We used intravascular ultrasonography to assess plaque morphology and morphometry in 310 patients with acute myocardial infarction (125 with ST-segment elevation and 185 with non–ST-segment elevation myocardial infarction) with varying degrees of renal dysfunction according to the creatinine clearance (CrCl): CrCl >70 ml/min in 153, CrCl of 30 to 69 ml/min in 103, and CrCl of <30 ml/min in 54 patients, including 20 patients requiring dialysis). The lesion site plaque burden was greatest (77.4 ± 11.0% vs 79.8 ± 12.5% vs 82.0 ± 10.3%, p = 0.031) and the lesion was longest (20.9 ± 9.1 vs 23.1 ± 9.5 vs 26.3 ± 9.6 mm, p = 0.038) in the lowest CrCl group. Infarct-related artery plaque rupture (31.4% vs 34.0% vs 53.7%, p = 0.011) and multiple plaque ruptures (11.1% vs 12.6% vs 33.3%, p <0.001) were the most common, the ruptured plaque cavities were the largest (1.98 ± 0.89 vs 2.20 ± 1.45 vs 3.06 ± 1.70 mm^2, p = 0.002), and the ruptured plaque was longest (2.33 ± 0.93 vs 2.59 ± 1.50 vs 3.33 ± 1.76 mm, p = 0.008) in the lowest CrCl group (<30 ml/min). Intravascular ultrasound-detected thrombus was observed most frequently in the lowest CrCl group (22.9% vs 23.3% vs 40.7%, p = 0.027). CrCl was the one of the independent predictors of culprit lesion plaque rupture (odds ratio 0.979, 95% confidence interval 0.963 to 0.994, p = 0.008). During 1 year of follow-up, the incidence of nonfatal myocardial infarction (2.6% vs 4.9% vs 11.1%, p = 0.044) and cardiac death (3.9% vs 6.8% vs 14.8%, p = 0.024) was greatest in the lowest CrCl group. Also, a strong trend was found toward the greatest incidence of stent thrombosis (2.0% vs 3.9% vs 9.3%, p = 0.057) in the lowest CrCl group. In conclusion, patients with acute myocardial infarction and significant renal dysfunction had more plaque vulnerability compared to those with normal renal function. This might be associated with poor clinical outcomes in patients with acute myocardial infarction and renal dysfunction. **(Am J Cardiol 2010;105:936–942)**

Patients with acute myocardial infarction (AMI) have various vulnerable plaque characteristics on intravascular ultrasound (IVUS) examination.[1–8] One IVUS study demonstrated that patients with end-stage renal disease had larger reference segment arterial and lumen areas; larger lesion site arterial, lumen, and plaque areas; larger arcs of calcium; and progressive calcific atherosclerosis.[9] To date, no study has examined the plaque characteristics using IVUS studies in patients with AMI according to renal function. Therefore, the purpose of the present study was to assess plaque morphology and plaque morphometry using IVUS examination in patients with AMI and varying degrees of renal dysfunction.

Methods

The present study was a retrospective, single-center study. A total of 2,995 patients with a first AMI were admitted to our institute from August 2004 to July 2008. We performed preintervention IVUS examination of coronary culprit lesions in infarct-related arteries within 24 hours of symptom onset in 380 patients. Of these 380 patients, we excluded 5 patients with subacute or late stent thrombosis, 3 with coronary artery bypass graft failure, 32 with cardiogenic shock, 20 with important systemic disease, and 10 patients in whom adequate IVUS images could not be obtained. Thus, the study population consisted of 310 patients with AMI (125 with ST-segment elevation and 185 with non–ST-segment elevation myocardial infarction). The diagnosis of AMI was according to a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocar-

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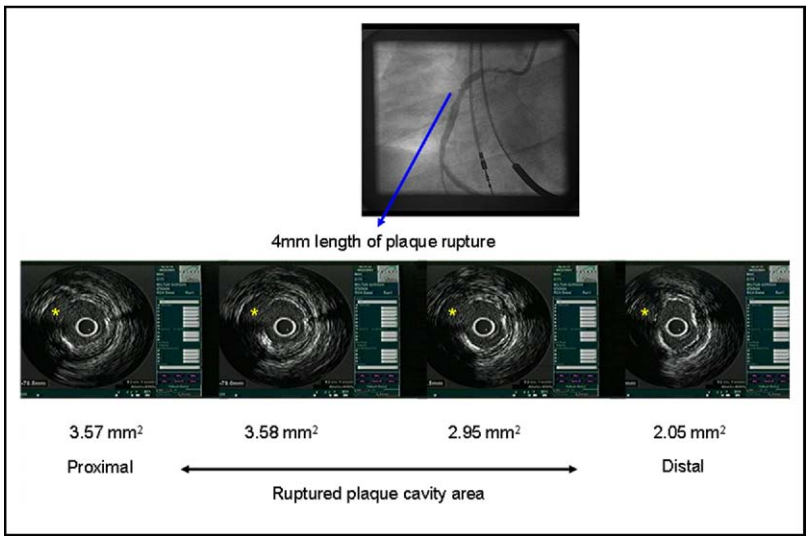

Figure 1. Example of plaque rupture in 71-year-old patient who presented with non–ST-segment elevation myocardial infarction and chronic renal failure with a history of diabetes mellitus, hypertension, and dyslipidemia. Her creatinine level was 5.7 mg/dl and CrCl was 8.0 ml/min.

dial Infarction.[10] The patients were divided into 3 groups according to the degree of renal function as determined by the calculated creatinine clearance (CrCl): CrCl >70 ml/min (n = 153); CrCl 30 to 69 ml/min (n = 103); and CrCl <30 ml/min (n = 54, including 20 patients requiring dialysis). The CrCl was calculated by applying the Cockcroft-Gault formula using the baseline serum creatinine level: CrCl = [(140 − age) × weight/serum creatinine × 72], with female gender adjustment ($CrCl_{female}$ = CrCl × 0.85).[11] All 310 infarct lesions were treated with stent implantation: 138 with sirolimus-eluting stents (Cypher stent, Cordis, Johnson & Johnson, Miami Lakes, Florida), 49 with paclitaxel-eluting stents (Taxus stent, Boston Scientific, Boston, Massachusetts), and 123 with bare metal stents. The institutional review board approved the protocol.

The absolute creatine kinase-MB levels were determined by radioimmunoassay (Dade Behring, Miami, Florida). Cardiac-specific troponin I levels were measured by a paramagnetic particle, chemiluminescent immunoenzymatic assay (Beckman-Coulter, Fullerton, California). High-sensitivity C-reactive protein was assessed using the immunoturbidimetric C-reactive protein-Latex (II) high-sensitivity assay using an Olympus 5431 AutoAnalyzer (Denka Seiken, Tokyo, Japan). The assay was performed according to the manufacturer's protocol and has been validated against the Dade-Behring method.[12]

Coronary angiograms were analyzed using a validated quantitative coronary angiographic system (Phillips H5000 or Allura DCI program, Philips Medical Systems, Best, The Netherlands). With the outer diameter of the contrast-filled catheter as the calibration standard, the minimal lumen diameter, reference diameter, and lesion length were measured in diastolic frames from orthogonal projections. Perfusion was evaluated according to the Thrombolysis In Myocardial Infarction criteria.[13] No-reflow was defined as Thrombolysis In Myocardial Infarction grade 0, 1, or 2 flow after percutaneous coronary intervention in the absence of mechanical obstruction.[14] Normal reflow was defined as Thrombolysis In Myocardial Infarction grade 3 flow. If the Thrombolysis In Myocardial Infarction flow after percutaneous coronary intervention was 0, 1, or 2 in the absence of angiographic stenosis, a repeat IVUS examination was performed to exclude the possibility of mechanical vessel obstruction.

All IVUS examinations were performed before stenting using a commercially available IVUS system (Boston Scientific/SCIMed, Minneapolis, Minnesota). Qualitative analysis was performed according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies.[15] Using planimetry software (Tape Measure, INDEC Systems, Mountain View, California), we measured the external elastic membrane (EEM) and lumen cross-sectional area (CSA). The plaque plus media CSA was calculated as the EEM CSA minus the lumen CSA, and the plaque burden was calculated as the plaque plus media CSA divided by the EEM CSA. The lesion was the site with the smallest lumen CSA. If multiple image slices showed the same minimum lumen CSA, the image slice with the largest EEM and plaque plus media was measured. A culprit lesion with ruptured plaque was defined as that containing a cavity that communicated with the lumen with an overlying residual fibrous cap fragment (Figure 1). Rupture sites separated by a length of artery containing smooth lumen contours without cavities were considered to represent different plaque ruptures (infarct-related artery with multiple plaque ruptures).[1–4,16] The identification of thrombus required ≥2 of the following: a distinct hypoechoic mass, brightly speckled plaque, channeling within the plaque, evacuated plaque cavity, or detached mobile mass.[4] Soft plaque was less bright compared to the reference adventitia. Fibrotic plaque was as bright as, or brighter than, the reference adventitia without acoustic shadowing. Calcific plaque was hyperechoic with shadowing. A calcified lesion contained >90° of circumferential lesion calcium. When no dominant plaque composition was seen, the plaque was classified as mixed. Coronary artery remodeling was assessed by comparing the lesion site to the reference EEM CSA. The remodeling index was the lesion

Table 1
Baseline characteristics

Variable	CrCl (ml/min) Group			p Value
	≥70 (n = 153)	30–69 (n = 103)	<30 (n = 53)	
Age (years)	57 ± 12	62 ± 15	69 ± 11	<0.001
Men	94 (61.4%)	60 (58.3%)	25 (46.3%)	0.152
Clinical presentation				0.026
ST-segment elevation myocardial infarction	73 (47.7%)	36 (35.0%)	16 (29.6%)	
Non–ST-segment elevation myocardial infarction	80 (52.3%)	67 (65.0%)	38 (70.4%)	
Diabetes mellitus	36 (23.5%)	38 (36.9%)	37 (68.5%)	<0.001
Hypertension	97 (63.4%)	78 (75.7%)	43 (79.6%)	0.027
Smoking	53 (34.6%)	38 (36.9%)	14 (25.9%)	0.371
Family history of coronary artery disease	22 (14.4%)	23 (22.3%)	7 (13.0%)	0.177
Ejection fraction (%)	47.2 ± 11.9	41.2 ± 13.4	40.9 ± 11.7	<0.001
Creatinine (mg/dl)	0.92 ± 0.19	1.26 ± 0.30	4.86 ± 2.14	<0.001
Creatinine clearance (ml/min)	92.0 ± 21.1	47.9 ± 8.7	16.1 ± 9.5	<0.001
High-sensitivity C-reactive protein (mg/L)	17.5 ± 14.5	16.7 ± 11.2	43.4 ± 34.4	0.016
Creatine kinase-MB (U/L)	17.9 ± 24.8	24.8 ± 30.4	35.9 ± 40.0	0.182
Cardiac-specific troponin I (ng/ml)	11.5 ± 23.0	13.8 ± 24.5	28.1 ± 31.7	0.047
White blood cell count (×1,000/mm^3)	9.3 ± 3.5	8.3 ± 3.0	10.0 ± 4.0	0.023
Hemoglobin (mg/dl)	13.0 ± 1.6	12.3 ± 2.1	10.4 ± 2.4	<0.001
Platelet count (×1,000/mm^3)	236 ± 72	220 ± 99	228 ± 93	0.506
Total cholesterol (mg/dl)	176 ± 44	180 ± 39	183 ± 56	0.635
Triglycerides (mg/dl)	130 ± 67	127 ± 63	125 ± 65	0.928
Low-density lipoprotein cholesterol (mg/dl)	109 ± 41	115 ± 29	122 ± 50	0.466
High-density lipoprotein cholesterol (mg/dl)	43.6 ± 13.1	40.3 ± 12.1	38.1 ± 14.4	0.316

Data are presented as n (%) of patients or mean ± SD.

Table 2
Coronary angiographic findings and procedural results

Variable	CrCl (ml/min) Group			p Value
	≥70 (n = 153)	30–69 (n = 103)	<30 (n = 53)	
Infarct-related coronary artery				0.237
Left main	1 (0.7%)	3 (2.9%)	1 (1.9%)	
Left anterior descending	81 (52.9%)	54 (52.4%)	32 (59.3%)	
Left circumflex	18 (11.8%)	13 (12.6%)	11 (20.4%)	
Right	53 (34.6%)	33 (32.0%)	10 (18.5%)	
Coronary lesion location				0.829
Ostium	3 (2.0%)	1 (1.0%)	0 (0.0%)	
Proximal	57 (37.3%)	40 (38.8%)	24 (44.4%)	
Middle	80 (52.3%)	50 (48.5%)	25 (46.3%)	
Distal	13 (8.5%)	12 (11.7%)	5 (9.3%)	
No. of diseased coronary arteries				0.027
1	88 (57.5%)	48 (46.6%)	20 (37.0%)	
2	38 (24.8%)	25 (24.3%)	23 (42.6%)	
3	27 (17.6%)	30 (29.1%)	11 (20.4%)	
Multivessel coronary disease	65 (42.5%)	55 (53.4%)	34 (63.0%)	0.019
Reference diameter (mm)	3.09 ± 0.77	2.95 ± 0.72	2.86 ± 0.61	0.065
Minimal lumen diameter (mm)	0.94 ± 0.62	0.87 ± 0.45	0.76 ± 0.48	0.024
Diameter stenosis (%)	69.6 ± 16.7	70.5 ± 17.4	73.4 ± 20.3	0.028
Lesion length (mm)	18.0 ± 6.4	20.6 ± 7.5	22.0 ± 10.9	0.130
Stent type				0.269
Sirolimus-eluting stent	68 (44.4%)	47 (45.6%)	23 (42.6%)	
Paclitaxel-eluting stent	21 (13.7%)	22 (21.4%)	6 (11.1%)	
Bare metal stent	64 (41.8%)	34 (33.0%)	25 (46.3%)	
Stent diameter (mm)	3.29 ± 0.46	3.23 ± 0.41	3.16 ± 0.46	0.148
Stent length (mm)	23.1 ± 9.8	25.1 ± 12.3	28.8 ± 11.2	0.024
No. of deployed stents	1.18 ± 0.45	1.15 ± 0.35	1.33 ± 0.55	0.032

Data are presented as n (%) of patients or mean ± SD.

Table 3
Intravascular ultrasound (IVUS) findings

Variable	CrCl (ml/min) Group			p Value
	≥70 (n = 153)	30–69 (n = 103)	<30 (n = 54)	
Referent				
External elastic membrane cross-sectional area (mm^2)	12.8 ± 4.5	12.2 ± 4.8	11.9 ± 4.5	0.034
Lumen cross-sectional area (mm^2)	8.5 ± 3.0	7.7 ± 3.0	7.0 ± 3.1	0.008
Plaque plus media cross-sectional area (mm^2)	4.3 ± 2.5	4.5 ± 2.8	4.9 ± 2.2	0.041
Plaque burden (%)	30.1 ± 12.3	36.9 ± 11.4	41.2 ± 10.8	0.003
Lesion site				
External elastic membrane cross-sectional area (mm^2)	12.7 ± 4.3	12.4 ± 5.1	11.1 ± 4.4	0.047
Lumen cross-sectional area (mm^2)	2.6 ± 1.2	2.5 ± 1.5	2.0 ± 1.1	0.017
Plaque plus media cross-sectional area (mm^2)	10.0 ± 4.0	9.9 ± 4.7	9.1 ± 4.1	0.024
Plaque burden (%)	77.4 ± 11.0	79.8 ± 12.5	82.0 ± 10.3	0.031
Lesion length (mm)	20.9 ± 9.1	23.1 ± 9.5	26.3 ± 9.6	0.038
Plaque morphology				0.065
Soft	82 (53.6%)	44 (42.7%)	20 (37.0%)	
Fibrotic	26 (17.0%)	16 (15.5%)	10 (18.5%)	
Calcific	31 (20.3%)	34 (33.0%)	19 (35.2%)	
Mixed	14 (9.2%)	9 (8.7%)	5 (9.3%)	
Arc of calcium (°)	103 ± 96	142 ± 110	180 ± 114	<0.001
Remodeling index	0.99 ± 0.23	1.02 ± 0.22	0.93 ± 0.19	0.031
Plaque rupture	48 (31.4%)	35 (34.0%)	29 (53.7%)	0.011
Multiple plaque rupture	17 (11.1%)	13 (12.6%)	18 (33.3%)	<0.001
Plaque cavity area (mm^2)	1.98 ± 0.89	2.20 ± 1.45	3.06 ± 1.70	0.002
Ruptured plaque length (mm)	2.33 ± 0.93	2.59 ± 1.50	3.33 ± 1.76	0.008
Intravascular ultrasound-detected thrombus	35 (22.9%)	24 (23.3%)	22 (40.7%)	0.027

Data are presented as n (%) of patients or mean ± SD.

site EEM CSA divided by the average of the proximal and distal reference EEM CSA.[8]

The hospital records of all the patients were reviewed to obtain information on the clinical demographics and medical history. Follow-up information was obtained through review of the hospital charts, telephone interviews, and the interventional database of the Heart Center of Chonnam National University Hospital (Gwangju, Korea). All deaths were considered of cardiac origin unless a noncardiac origin had been established clinically or at autopsy. Target lesion revascularization was defined as any intervention to treat in-stent restenosis, including stent edges within 5 mm proximal or distal to the stent. Nonfatal myocardial infarction was defined as ischemic symptoms associated with cardiac enzyme elevation of ≥3 times the upper limit of normal. Stent thrombosis included definite, probable, or possible stent thrombosis, classified according to the Academic Research Consortium definition.

The Statistical Package for Social Sciences for Windows, version 15.0 (SPSS, Chicago, Illinois) was used for all analyses. Continuous data are expressed as the mean ± SD and categorical data as frequencies and percentages. Differences in continuous variables were tested by analysis of variance for normal or log-normal distributed variables (ie, age, ejection fraction, and CrCl). The Kruskal-Wallis test was used for other variables (ie, creatine kinase-MB, cardiac-specific troponin I, and high-sensitivity C-reactive protein). Differences in the categorical variables were tested using the chi-square test or Fisher's exact test. Pearson's correlation coefficient was used to evaluate the associations between CrCl and various clinical and IVUS parameters. Multivariate analysis was performed to identify independent predictors of culprit lesion plaque rupture. A p value <0.05 was considered statistically significant.

Results

The baseline characteristics are summarized in Table 1. The CrCl was 92.0 ± 21.1 mg/min in the highest CrCl group, 47.9 ± 8.7 mg/min in the middle CrCl group, and 16.1 ± 9.5 mg/min in lowest CrCl group. Patients with lowest CrCl were the oldest and had the greatest incidence of diabetes mellitus and hypertension. The left ventricular ejection fraction was lowest in the lowest CrCl group. The high-sensitivity C-reactive protein, troponin I, and white blood cell count levels were the greatest and the hemoglobin was the lowest in the lowest CrCl group.

The angiographic findings are summarized in Table 2. No significant differences were found in the infarct-related arteries, lesion location, or lesion length. However, multivessel disease was most common in the lowest CrCl group. The minimal lumen diameter was the smallest and the diameter stenosis was the greatest in the lowest CrCl group. No significant differences were found in the stent type used or the stent diameter; however, the stent length was longest and the number of deployed stent was the greatest in the lowest CrCl group. A trend was seen toward post-stenting no-reflow observed most frequently in the lowest CrCl group (8.5% [13 of 153] vs 11.7% [12 of 103] vs 20.4% [11 of 53]; p = 0.064).

The IVUS findings are summarized in Table 3. The reference segment EEM and lumen CSAs were the smallest and the plaque plus media CSA and plaque burden were the

Table 4
Multivariate analysis for culprit lesion plaque rupture

Variable	OR	95% CI	p Value
Soft plaque	8.913	3.170–25.066	<0.001
Creatinine clearance	0.979	0.963–0.994	0.008
Diabetes mellitus	2.895	1.138–7.365	0.026
High-sensitivity C-reactive protein	1.009	1.001–1.018	0.035
Smoking	1.836	0.786–4.284	0.160
Positive remodeling	1.505	0.657–3.449	0.334
Ejection fraction	0.987	0.955–1.020	0.425
Cardiac-specific troponin I	1.001	0.989–1.012	0.919

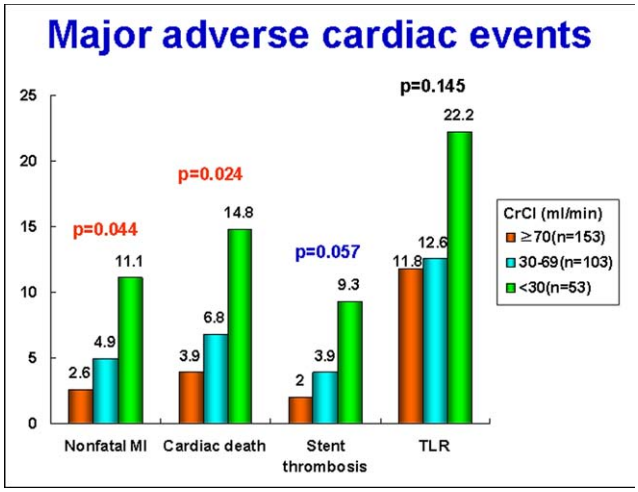

Figure 2. Incidence of nonfatal myocardial infarction, cardiac death, stent thrombosis, and target lesion revascularization according to CrCl at 1 year of follow-up.

greatest in the lowest CrCl group. The lesion site EEM and lumen CSAs were the smallest, the plaque plus media CSA and plaque burden were the greatest, and the IVUS lesion length was the longest in the lowest CrCl group. The arc of calcium was the greatest and the remodeling index was the smallest in the lowest CrCl group. Infarct-related artery plaque rupture and multiple plaque ruptures were the most common, the ruptured plaque cavities were the largest, and the plaque rupture length was the longest in the lowest CrCl group. IVUS-detected thrombus was observed most frequently in the lowest CrCl group.

The CrCl correlated with the ejection fraction ($r = 0.260$, $p < 0.001$), C-reactive protein ($r = -0.173$, $p = 0.027$), hemoglobin ($r = 0.387$, $p < 0.001$), reference segment plaque burden ($r = -0.203$, $p < 0.001$), arc of calcium ($r = -0.311$, $p < 0.001$), plaque cavity area ($r = -0.249$, $p = 0.008$), and ruptured plaque length ($r = -0.212$, $p = 0.025$).

The cardiac enzymes were elevated most significantly after stenting in the lowest CrCl group (change in creatine kinase-MB $+2.3 \pm 2.0$ U/L vs $+3.2 \pm 6.1$ U/L vs $+13.5 \pm 6.1$ U/L, $p < 0.002$, and change in cardiac-specific troponin I $+1.3 \pm 3.5$ ng/ml vs $+2.2 \pm 3.4$ ng/ml vs $+18.0 \pm 23.4$ ng/ml, $p < 0.001$).

We performed multivariate analysis to determine the independent predictors of culprit lesion plaque rupture. The following variables were tested (all with $p < 0.2$ on univariate analysis): diabetes mellitus, smoking, ejection fraction, high-sensitivity C-reactive protein, baseline cardiac-specific troponin I, CrCl, positive remodeling, and soft plaque. Soft plaque (odds ratio [OR] 8.913, 95% confidence interval [CI] 3.170 to 25.066, $p < 0.001$), CrCl (OR 0.979, 95% CI 0.963 to 0.994, $p = 0.008$), diabetes mellitus (OR 2.895, 95% CI 1.138 to 7.365, $p = 0.026$), and high-sensitivity C-reactive protein (OR 1.009, 95% CI 1.001 to 1.018, $p = 0.035$) were the independent predictors of culprit lesion plaque rupture (Table 4).

During 1 year of follow-up, although no significant difference was found in the incidence of target lesion revascularization among the 3 CrCl groups, the incidence of nonfatal myocardial infarction and cardiac death was greatest in the lowest CrCl group, with a strong trend toward the greatest incidence of stent thrombosis (subacute and late stent thrombosis) in the lowest CrCl group (Figure 2).

Discussion

The present IVUS study has demonstrated that a significant decrease in renal function (CrCl <30 ml/min) is associated with more severe and diffuse atherosclerosis (longer lesions with larger lesion site and reference segment plaque burdens) and more unstable plaque morphology (more frequent plaque ruptures and thrombus). Also, CrCl was the independent predictor of culprit lesion plaque rupture, with the incidence of nonfatal myocardial infarction and cardiac death the greatest in the lowest CrCl group. Also, a strong trend was found toward the greatest incidence of subacute and late stent thrombosis in the lowest CrCl group at 1 year of clinical follow-up in patients with AMI.

Previous studies have shown that renal insufficiency is an independent predictor of significant coronary artery disease and is associated with subclinical atherosclerosis, vascular dysfunction, brachial artery endothelial dysfunction, and carotid artery intima-media thickening.[17,18] Gruberg et al[9] reported that chronic renal insufficiency in the absence of dialysis was not associated with increased plaque burden and calcium. However, the transition to the need for dialysis was associated with progressive calcific atherosclerosis (larger lesion plaque area and calcium).[9] In the present study, patients with AMI and severe renal dysfunction had severe and diffuse calcific atherosclerosis. This might be associated with the increasing prevalence of hypertension and diabetes, especially among an aging population, in patients with renal dysfunction.

The typical IVUS features of AMI include plaque rupture, thrombus formation, positive remodeling, and only spotty or deep calcium within the minimum lumen site.[1–8] In the present study, patients with AMI and severe renal dysfunction had unstable plaque morphology (a greater frequency of single and multiple plaque ruptures and thrombus). Several studies have reported that the rupture of a vulnerable plaque and subsequent thrombus formation is the most important mechanism leading to AMI.[19,20] The results of the present study are consistent with those of previous IVUS studies, and renal dysfunction might play a part in the plaque vulnerability in patients with AMI.

Plaque ruptures tend to occur at a point at which the fibrous cap is thinnest and heavily infiltrated by macrophages, indicating ongoing inflammation at the site of

plaque disruption.[20] The inflammatory response during AMI is strong, and the severity of the inflammatory response might be an important determinant of the clinical outcome.[21] Several studies have shown that C-reactive protein elevation is observed in patients with renal dysfunction and has been associated with a high incidence of clinical events.[22–28] In the present study, renal function was associated with an inflammatory response, which was reflected by C-reactive protein elevation. Our results suggest that poor clinical outcomes in patients with renal dysfunction might be associated with the development of vulnerable plaque and an increased inflammatory response.

Lambert et al[29] reported that patients with AMI and chronic kidney disease and dipstick proteinuria had an increased cumulative incidence of stent thrombosis (hazard ratio 3.69), all-cause mortality (hazard ratio 2.68), and nonfatal myocardial infarction or death (hazard ratio 3.20) at 1 year of follow-up. Latif et al[30] evaluated the outcomes of patients with chronic kidney disease undergoing percutaneous coronary intervention with drug-eluting stents from 4,791 patients enrolled in the Evaluation of Drug Eluting Stents and Ischemic Events (EVENT) Registry. They reported that lower CrCl was associated with more frequent death or myocardial infarction during the initial hospital stay ($p = 0.001$) and at 1 year ($p < 0.001$).[30] In the present study, the cardiac enzymes were elevated most significantly after stenting in the lowest CrCl group, with a trend toward post-stenting no-reflow most frequently in the lowest CrCl group. Also, the incidence of nonfatal myocardial infarction and cardiac death was greatest in the lowest CrCl group, with a strong trend toward the greatest incidence of stent thrombosis in lowest the CrCl group at 1 year. The results of the present study are consistent with those from previous studies. The poor clinical outcomes might be associated with more severe and diffuse atherosclerosis and more unstable plaque morphology with an increased inflammatory response in patients with AMI and renal dysfunction.

The present study had several limitations. First, the present study included a small sample of patients, increasing the possibility of selection bias. Second, this was a retrospective, single-center study. The results of our study should be verified by additional prospective investigations. Third, the serum creatinine level for the calculation of CrCl is a less precise measure of renal function than the CrCl obtained from a 24-hour urine collection. Fourth, IVUS examination is limited in the detection of thrombi compared to other imaging modalities such as optical coherence tomography. Finally, we did not perform 3-vessel IVUS examinations. Therefore, we could not assess the frequency of noninfarct-related artery plaque ruptures or thrombus.

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