Contrast Volume and Decline in Kidney Function in Optical Coherence Tomography-Guided Percutaneous Coronary Intervention

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Summary

Optical coherence tomography (OCT)-guided percutaneous coronary intervention (PCI) may increase contrast volume. However, the impact of OCT-guided PCI on the decline in kidney function (DKF) in actual clinical practice remains unclear.

Among 1,003 consecutive patients who underwent either OCT-guided or intravascular ultrasound (IVUS)-guided PCI in our institute, we identified 202 propensity score-matched pairs adjusted by baseline factors. The incidence of DKF was compared between the OCT-guided PCI group and the IVUS-guided PCI group. DKF was defined as an increase in serum creatinine level of ≥0.5 mg/dL or a relative increase of ≥25% over baseline within 48 hours (acute DKF) or 1 month (sustained DKF) after PCI.

Baseline characteristics, including the prevalence of chronic kidney disease (54% versus 46%, \(P = 0.09\)), were comparable between the OCT- and IVUS-guided PCI groups except for the age. The contrast volume was comparable between the two groups (153 ± 56 versus 144 ± 60 mL, \(P = 0.09\)), although it was significantly greater in the OCT-guided PCI group in patients with acute coronary syndrome (ACS; 175 ± 55 versus 159 ± 43 mL, \(P = 0.04\)). The incidence of acute DKF (0.5% versus 2.5%, \(P = 0.22\)) and sustained DKF (5.0% versus 10.4%, \(P = 0.31\)) was comparable between the two groups. Multivariate analysis demonstrated that ACS (odds ratio 4.74, 95% confidence interval 2.72-8.25, \(P < 0.001\)) was a predictor of sustained DKF.

Compared with IVUS-guided PCI, OCT-guided PCI did not increase the incidence of DKF in actual clinical practice, although the increased contrast volume was observed in ACS cases.

Key words: Contrast-induced nephropathy, Renal dysfunction, Intravascular ultrasound

Intravascular imaging modalities, including optical coherence tomography (OCT) and intravascular ultrasound (IVUS), during percutaneous coronary intervention (PCI) provide detailed quantitative and qualitative information. A recent meta-analysis demonstrated the superiority of imaging-guided PCI to angi-guided PCI regarding clinical outcomes, including all cause death. This is partly owing to greater accuracy in measurement of vessel/luminal diameter and subsequent device selection with appropriate size. In particular, the greater accuracy of OCT measurement in both cross-sectional and longitudinal images compared with IVUS measurement has been demonstrated. In addition, a recent randomized controlled study showed that the incidence of stent edge dissection was lower in OCT-guided PCI than in IVUS-guided PCI. Thus, the importance of OCT-guided PCI in daily clinical practice has been highlighted, although the clinical advantage of OCT over IVUS has not been proven.

In contrast to IVUS, OCT requires removing blood flow from the imaging field by the injection of contrast or dextran during pullback. Because this may increase the total amount of contrast during PCI, some physicians dislike performing OCT-guided PCI despite the advantage. However, it remains uncertain if the larger amount of contrast and subsequent greater incidence of nephropathy is observed in OCT-guided PCI in actual clinical practice. Thus, the aim of the present study was to evaluate 1) the contrast volume required in OCT-guided PCI compared with that in IVUS-guided PCI and 2) the incidence of both acute and sustained decline in kidney function (DKF) after OCT-guided PCI.

Methods

Study population: This was a retrospective, observational study in a single center. Among 1,375 patients who underwent PCI in our institute between January 2013 and March 2017, a total of 1,003 patients who underwent...
either OCT-guided ($n = 537$) or IVUS-guided ($n = 466$) PCI were identified (Figure 1). Exclusion criteria included angio-guided PCI, hemodialysis, patients with in-hospital death, and cases without data of kidney function at baseline. To adjust potential selection bias for the total contrast volume and the incidence of DKF between the OCT-guided group and the IVUS-guided group, propensity score-matching analysis was performed. Finally, we analyzed a total of 202 matched patients in the present study. All procedures were performed in accordance with the ethical standard of the Ethics Committee of the Kitasato University Hospital, and all patients provided written informed consent before the procedure.

**Percutaneous coronary intervention:** Patients with stable angina pectoris (SAP) received dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg) at least 5 days before PCI. Patients with acute coronary syndrome (ACS) received a loading dose of aspirin (200 mg) and clopidogrel (300 mg) before the procedure. Per protocol in our institute, prophylaxis with saline infusion was performed in SAP patients with chronic kidney disease (CKD) before PCI (total 500 mL, 160 mL/hour) and after PCI (total 1,000 mL, 80 mL/hour). All procedural strategies including the selection of imaging modality, were decided by the operators. OCT imaging was performed using a frequency-domain OCT system (C7-XR OCT Intravascular Imaging System; St. Jude Medical, St. Paul, MN, USA) during injection of contrast media or dextran to clear blood from the imaging field.

**Study definition:** Renal function was routinely assessed within 48 hours and 1 month after PCI in all the patients in the present study. Acute DKF and sustained DKF were defined as an increase in serum creatinine level ≥ 0.5 mg/dL or a relative increase of ≥ 25% over baseline value within 48 hours and 1 month after PCI, respectively. ACS consisted of ST-segment elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), and unstable angina pectoris. STEMI was defined as continuous chest pain lasting > 30 minutes, arrival at the hospital within 12 hours from the onset of chest pain, ST-segment elevation > 0.1 mV in ≥ 2 contiguous leads, or new left bundle-branch block on a 12-lead electrocardiogram, and elevated cardiac markers (creatinine kinase-MB or troponin T/I). NSTEMI was defined as ischemic symptoms with elevated cardiac markers in the absence of ST-elevation on electrocardiogram. Unstable angina was defined as angina at rest, accelerated angina, or new-onset angina, without the elevation of cardiac markers.

**Statistical methods:** Categorical variables were summarized as counts (%), and between-group comparisons were performed using Fisher’s exact test or the chi-squared test, as appropriate. Continuous variables were summarized as mean ± standard deviation or median (25th-75th percentile), as appropriate, depending on the normality of distribution using the Kolmogorov-Smirnov test. Between-group comparisons were performed using independent-sample $t$-tests or Mann-Whitney $U$ test. Propensity score-matching analysis was performed to control selection bias for the total contrast volume and the incidence of DKF using the nearest neighbor one-to-one pair matching based on clinical presentation, CKD, diabetes, low left ventricular ejection fraction, lesion characteristics. These variables were selected based on clinical knowledge or previous reports which had shown its prognostic significance. A caliper width of 0.2 of the standard deviation of the logit of the propensity score was applied for the developed propensity score. Multivariate analyses were performed to adjust potential confounders to detect independent predictors for DKF, using factors that had been reported as the potential factor.
Baseline characteristics: The baseline characteristics of a cohort before and after adjustment using propensity score-matching are shown in Tables I, II, respectively. After adjustment, there was no significant difference between the OCT-guided and IVUS-guided groups, other than the age, the percentage of patients with PCI history, and the prevalence of patients with medications for renin-angiotensin system.

**Table I. Baseline Characteristics Before Adjustment**

|                  | OCT-guided PCI | IVUS-guided PCI | P value |
|------------------|----------------|-----------------|---------|
|                  | n = 537        | n = 466         |         |
| Age, years       | 69.9 ± 9.7     | 67.4 ± 11.7     | < 0.001 |
| Male gender, n (%)| 437 (81)       | 368 (80)        | 0.341   |
| BMI, kg/m²       | 23.9 ± 3.7     | 24.2 ± 3.4      | 0.276   |
| Risk factor, n (%)|               |                 |         |
| Hypertension     | 444 (83)       | 337 (72)        | < 0.001 |
| Dyslipidemia     | 432 (80)       | 313 (67)        | < 0.001 |
| Diabetes         | 195 (36)       | 182 (39)        | 0.361   |
| Smoking          | 355 (66)       | 289 (62)        | 0.184   |
| Chronic kidney disease | 250 (47) | 226 (49) | 0.518   |
| Family history of IHD | 121 (23) | 101 (22) | 0.879   |
| History of MI    | 158 (29)       | 73 (16)         | < 0.001 |
| History of PCI   | 241 (45)       | 93 (20)         | < 0.001 |
| History of CABG  | 8 (1.5)        | 11 (2.4)        | 0.311   |
| Clinical presentation, n (%) | 81 (15) | 338 (73) | < 0.001 |
| ACS              | 456 (85)       | 128 (27)        |         |
| Stable Angina    | 58 ± 11        | 53 ± 12         | < 0.001 |
| Medication, n (%)|               |                 |         |
| Aspirin          | 470 (88)       | 199 (43)        | < 0.001 |
| Thienopyridine   | 445 (83)       | 164 (36)        | < 0.001 |
| ACEI/ARB         | 431 (80)       | 217 (47)        | < 0.001 |
| β-blocker        | 264 (49)       | 152 (33)        | < 0.001 |
| Statin           | 473 (88)       | 226 (49)        | < 0.001 |
| NOAC             | 25 (4.7)       | 18 (3.9)        | 0.528   |
| Laboratory findings |           |                 |         |
| HbA1c, %         | 6.5 ± 1.1      | 6.5 ± 1.2       | 0.528   |
| LDL-C, mg/dL     | 95 ± 29        | 114 ± 39        | < 0.001 |
| Serum creatinine, mg/dL | 0.93 ± 0.22 | 1.00 ± 0.33 | < 0.001 |
| eGFR, mL/minute/1.73 m² | 62.6 ± 16.5 | 60.9 ± 19.4 | 0.165   |
| BNP, pg/mL       | 87.0 [42.4, 162.4] | 61.7 [21.9, 167.4] | 0.253   |
| Lesion characteristics |           |                 |         |
| Target lesion, n (%) |            | < 0.001         |         |
| LAD              | 334 (62)       | 227 (49)        |         |
| LCX              | 111 (21)       | 77 (17)         |         |
| RCA              | 148 (28)       | 199 (43)        |         |
| LMT              | 18 (3.6)       | 19 (4.1)        |         |
| De novo lesion, n (%) | 305 (57) | 408 (88) | < 0.001 |

Continuous variables are presented as mean ± standard deviation or median [25th-75th percentile]. ACEI indicates angiotensin converting enzyme inhibitor; ACS, acute coronary syndrome; ARB, angiotensin II receptor blocker; BMI, body mass index; BNP, brain natriuretic peptide; CABG, coronary artery bypass grafting; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c; IHD, ischemic heart disease; IVUS, intravascular ultrasound; LAD, left anterior descending artery; LCX, left circumflex artery; LDL-C, low-density lipoprotein cholesterol; LMT, left main trunk; LVEF, left ventricular ejection fraction on echocardiography; MI, myocardial infarction; NOAC, novel oral anticoagulant; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; and RCA, right coronary artery.

Results

Baseline characteristics: The baseline characteristics of a cohort before and after adjustment using propensity score-matching are shown in Tables I, II, respectively. After adjustment, there was no significant difference between the OCT-guided and IVUS-guided groups, other than the age, the percentage of patients with PCI history, and the prevalence of patients with medications for renin-angiotensin system.

Contrast volume in OCT-guided and IVUS-guided PCI: Comparisons of the total contrast volume between the OCT-guided and IVUS-guided groups after adjustment are shown in Figure 2. The total contrast volume was comparable between the two groups in overall (153 ± 56 versus 144 ± 60 mL, P = 0.09) and SAP patients (139 ± 52 versus 132 ± 67 mL, P = 0.35), although a greater amount was observed in the OCT-guided PCI group in patients with ACS (175 ± 55 versus 159 ± 43 mL, P = 0.04). Among the cases in the OCT-guided PCI group, the dextran injection was performed (50 [35-90] mL) in a total of eight cases. A trend toward smaller contrast volume was observed in cases with dextran compared with cases without dextran (n = 195; 115 ± 35 versus 149 ± 57 mL).
Table II. Baseline Characteristics of the Matched Cohort

|                          | OCT-guided PCI n = 202 | IVUS-guided PCI n = 202 | P value |
|--------------------------|------------------------|-------------------------|---------|
| Age, years               | 70.3 ± 10.3            | 67.8 ± 11.5             | 0.021   |
| Male gender, n (%)       | 159 (79)               | 173 (86)                | 0.091   |
| BMI, kg/m²               | 24.1 ± 4.0             | 24.3 ± 3.1              | 0.607   |
| Risk factor, n (%)       |                        |                         |         |
| Hypertension             | 156 (77)               | 153 (76)                | 0.815   |
| Dyslipidemia             | 146 (72)               | 147 (73)                | 1.000   |
| Diabetes                 | 92 (46)                | 85 (42)                 | 0.548   |
| Smoking                  | 115 (58)               | 135 (67)                | 0.063   |
| Chronic kidney disease   | 110 (54)               | 92 (46)                 | 0.091   |
| Family history of IHD    | 48 (24)                | 47 (24)                 | 0.907   |
| History of MI            | 58 (29)                | 48 (24)                 | 0.309   |
| History of PCI           | 83 (41)                | 63 (31)                 | 0.049   |
| History of CABG          | 6 (3.0)                | 7 (3.5)                 | 1.000   |
| Clinical presentation, n (%) |              |                         | 0.544   |
| ACS                      | 80 (40)                | 87 (43)                 |         |
| Stable Angina            | 122 (60)               | 115 (57)                |         |
| LV EF, %                 | 58.1 ± 12.4            | 55.1 ± 11.8             | 0.014   |
| Medication, n (%)        |                        |                         |         |
| Aspirin                  | 151 (75)               | 133 (66)                | 0.064   |
| Thienopyridine           | 136 (67)               | 118 (58)                | 0.080   |
| ACEI/ARB                 | 143 (71)               | 116 (58)                | 0.009   |
| β-blocker                | 110 (54)               | 100 (50)                | 0.424   |
| Statin                   | 155 (77)               | 148 (73)                | 0.491   |
| NOAC                     | 13 (6.4)               | 9 (4.5)                 | 0.512   |
| Laboratory findings      |                        |                         |         |
| HbA1c, %                 | 6.3 [5.8, 7.0]         | 6.1 [5.8, 6.8]          | 0.146   |
| LDL-C, mg/dL             | 95 [78, 118]           | 96 [75, 125]            | 0.868   |
| Serum creatinine, mg/dL  | 0.96 [0.80, 1.09]      | 0.92 [0.79, 1.18]       | 0.396   |
| eGFR, mL/minute/1.73 m²  | 58.1 [48.4, 69.2]      | 61.1 [47.8, 72.4]       | 0.239   |
| BNP, pg/mL               | 87.2 [40.9, 203.7]     | 70.2 [27.3, 152.8]      | 0.273   |
| Lesion characteristics   |                        |                         | 0.370   |
| Target lesion, n (%)     | 102 (51)               | 112 (55)                |         |
| LAD                      | 49 (24)                | 40 (20)                 |         |
| RCA                      | 78 (39)                | 73 (36)                 |         |
| LMT                      | 10 (5.0)               | 10 (5.0)                |         |
| Type B2/C, n (%)         | 154 (76)               | 151 (75)                | 0.817   |
| De novo lesion, n (%)    | 162 (80)               | 157 (78)                | 0.626   |

Abbreviations are as in Table I.

\( P = 0.10 \).

**Incidence of acute DKF:** Comparisons of the incidence of acute DKF between the OCT-guided and IVUS-guided groups after adjustment are shown in Figure 3. The incidence of acute DKF (0.5% versus 2.5%, \( P = 0.22 \)) was not significantly different between the two groups. In both the CKD and non-CKD subgroups, the incidence of acute DKF (0% versus 3.3%, \( P = 0.09 \); 1.0% versus 1.8%, \( P = 1.00 \), respectively) was not significantly different between the two groups.

**Incidence of sustained DKF:** Comparisons of the incidence of sustained DKF between the OCT-guided and IVUS-guided groups after adjustment are shown in Figure 4. The incidence of sustained DKF (5.0% versus 10.4%, \( P = 0.06 \)) was not significantly different between the two groups. In both the CKD and non-CKD subgroups, the incidence of sustained DKF (1.8% versus 5.4%, \( P = 0.25 \); 8.7% versus 14.6%, \( P = 0.28 \), respectively) was not significantly different between the two groups. The incidence of both acute and sustained DKF is shown in Table III.

**Predictors of sustained DKF:** Multivariate models showed that ACS was an independent predictor for the incidence of sustained DKF (Table IV).

**Discussion**

The main findings of the present study were as follows: (1) the contrast volume was comparable between OCT- and IVUS-guided PCI, although a greater amount in OCT-guided PCI was observed in cases with ACS. (2) The incidence of both acute DKF and sustained DKF was not significantly different between OCT- and IVUS-guided PCI.

**Contrast volume in OCT-guided PCI:** Previous randomized studies reported that the total contrast volume in OCT-guided PCI was greater than that in IVUS-guided PCI. In the ILUMIEN III: OPTIMIZE PCI trial, which demonstrated the non-inferiority of minimal stent area in
Figure 2. Contrast volume. The total contrast volume was comparable between OCT- and IVUS-guided PCI, although a greater amount was observed in OCT-guided PCI in patients with ACS. ACS indicates acute coronary syndrome; IVUS, intravascular ultrasound; NS, not significant; OCT, optical coherence tomography; and SAP, stable angina pectoris.

Figure 3. Incidence of acute DKF. The incidence of acute DKF was comparable between OCT- and IVUS-guided PCI irrespective of the presence of CKD. CKD indicates chronic kidney disease; DKF, decline in kidney function; IVUS, intravascular ultrasound; NS, not significant; and OCT, optical coherence tomography.

Figure 4. Incidence of sustained DKF. The incidence of sustained DKF was comparable between OCT- and IVUS-guided PCI irrespective of the presence of CKD. CKD indicates chronic kidney disease; DKF, decline in kidney function; IVUS, intravascular ultrasound; NS, not significant; and OCT, optical coherence tomography.
Table III. Incidence of DKF according to CKD Stage

| OCT guided PCI (n = 202) | IVUS guided PCI (n = 202) | P value |
|-------------------------|--------------------------|---------|
| **Acute DKF, n (%)**    |                          |         |
| G1 eGFR ≥ 90            | 1/11 (9.1)               | 1/18 (5.6) | 0.767  |
| G2 60-89                | 0/81 (0)                 | 1/92 (1.1) | 0.260  |
| G3a 45-59               | 0/67 (0)                 | 2/55 (3.6) | 0.201  |
| G3b 30-44               | 0/37 (0)                 | 0/28 (0)  | -      |
| G4 15-29                | 0/6 (0)                  | 1/9 (11)  | 0.301  |
| G5 < 15                 | -                       | -        | -      |

| **Sustained DKF, n (%)**|                          |         |
| G1 eGFR ≥ 90            | 1/11 (9.1)               | 6/18 (33)| 0.202  |
| G2 60-89                | 7/11 (8.6)               | 10/92 (11)| 0.260  |
| G3a 45-59               | 1/67 (1.5)               | 1/55 (1.8)| 0.888  |
| G3b 30-44               | 0/37 (0)                 | 1/28 (3.6)| 0.431  |
| G4 15-29                | 1/6 (17)                 | 3/9 (33)  | 0.604  |
| G5 < 15                 | -                       | -        | -      |

CKD indicates chronic kidney disease; DKF, decline in kidney function; and eGFR, estimated glomerular filtration rate.

Table IV. Factors Associated with Sustained DKF

|                | Odds ratio | 95% CI    | P value |
|----------------|------------|-----------|---------|
| **Non-adjusted cohort** |            |           |         |
| ACS            | 4.741      | 2.724-8.252 | < 0.001 |
| Diabetes       | 2.256      | 1.367-3.722 | 0.002   |
| eGFR < 45 mL/minute/1.73 m² | 0.561      | 0.252-1.251 | 0.158   |
| Age ≥ 70       | 1.413      | 0.848-2.353 | 0.185   |
| Contrast volume ≥ 200 mL | 0.645      | 0.324-1.283 | 0.211   |
| Low LVEF (LVEF < 40%) | 2.043      | 0.995-4.192 | 0.052   |
| **Adjusted cohort** |            |           |         |
| ACS            | 4.498      | 1.951-11.386 | < 0.001 |
| Diabetes       | 2.088      | 0.985-4.550 | 0.055   |
| eGFR < 45 mL/minute/1.73 m² | 1.436      | 0.441-4.055 | 0.324   |

ACS indicates acute coronary syndrome; CI, confidence interval; CKD, chronic kidney disease; DKF, decline in kidney function; eGFR, estimated glomerular filtration rate; and LVEF, left ventricular ejection fraction.

OCT-guided PCI compared with IVUS-guided PCI, the contrast volume in OCT-guided PCI was significantly greater than that in IVUS-guided PCI (222 versus 190 mL, P = 0.004). The greater contrast volume in optical frequency domain imaging (OFDI)-guided PCI than in IVUS-guided PCI (164 versus 138 mL, P < 0.001) was also reported in the OPINION trial. In contrast, the contrast volume was comparable between the two groups in the present study. Although the exact reason for this discrepancy is unclear, the difference in procedural steps between the protocol-based PCI in randomized trials and daily practice might be a potential reason. In our daily practice of OCT-guided PCI, we do not usually perform angiography in addition to the OCT pullback in each procedural step, except for the initial and final angiogram, because angiograms can be simultaneously obtained during the OCT pullback. Thus, the total number of contrast injections in OCT-guided PCI might be similar to that in IVUS-guided PCI. In ACS cases, a greater contrast volume in OCT-guided PCI was observed in the present study. This might be due to the need for repeat OCT pullbacks to obtain clear images. The presence of thrombus in addition to tight stenosis may prevent deep penetration of light needed for clear images in ACS cases. Repetitive thrombectomy and predilatation before OCT pullback would help to ensure clearer images in ACS cases with large amounts of thrombus. Irrespective of clinical presentation, the use of dextran can negate the concern regarding the increased amount of contrast used in OCT-guided PCI, particularly in patients with ACS and/or CKD.

DKF in OCT-guided PCI: The low incidence of acute kidney injury in OCT-guided PCI was reported in previous randomized trials. Meneveau et al. showed that the incidence of acute kidney injury, defined as an absolute increase in serum creatinine of ≥ 0.5 mg/dL from baseline, was only 1.6% in both OCT- and angio-guided PCI in cases of ACS, although greater contrast was observed in OCT-guided PCI (190 versus 120 mL, P < 0.0001). In the OPINION trial, no contrast-induced nephropathy, defined as an increase in serum creatinine level of ≥ 0.5 mg/ dL or a relative increase of ≥ 25% over baseline value within 72 hours, was observed in both OFDI- and IVUS-guided PCI. Because these studies excluded patients with
CKD (e.g., estimated glomerular filtration rate < 30 mL/minute/1.73 m² or serum creatinine level > 1.5 mg/dL, in the OPTINION study), the impact of OCT-guided PCI on the incidence of acute kidney injury in daily practice was unclear. In the present study, half of the patients (50%) had CKD. The incidence of acute DKF and sustained DKF in OCT-guided PCI was 1.4% and 3.5%, respectively, among patients with CKD, without a significant difference from IVUS-guided PCI. Considering the results obtained in previous randomized trials and the present findings, physicians may not have to consider the increased risk of DKF in OCT-guided PCI.

Factors associated with DKF: Previous studies identified increasing age, CKD, and larger contrast volume (e.g., ≥ 200 mL) as risk factors for DKF after PCI. Although most DKF cases after PCI are transient without progression to irreversible renal impairment, a certain percentage of patients may require subsequent life-long hemodialysis. Thus, physicians must consider prophylaxis in patients with increased risk of DKF. In particular, the effort to reduce contrast volume during PCI in addition to prophylactic saline infusion is important. In the present study, the mean contrast volume was less than 200 mL in both OCT- and IVUS-guided PCI, without significant difference between the two groups irrespective of clinical presentation. This highlighted the fact that the choice of OCT or IVUS is not the central issue in achieving PCI with smaller contrast volume. In fact, the present study did not identify OCT-guided PCI as an independent predictor of sustained DKF.

Limitations: Some limitations exist in the present study. First, this was a retrospective study performed in a single center in which both OCT and IVUS are frequently used in daily practice. Second, the potential confounders for the incidence of DKF could not be completely adjusted between the two groups, although propensity matching analysis was performed. In addition, we found a limited number of matched cases among the overall cohort. Further studies with homogeneous population may yield different results. Third, factors associated with acute DKF were not determined in the present study because of the low incidence. Future studies with larger cohorts with different backgrounds may further clarify the impact of imaging-guided PCI on the incidence of acute DKF. Fourth, the causal relationship between the use of contrast and the incidence of DKF was unclear. Thus, we applied “DKF” instead of “contrast induced nephropathy” as a term for worsening of renal function after PCI in the present study. In particular, the cause of sustained DKF might be multifactorial in addition to ACS presentation, which was shown to be an independent predictor in multivariate analysis in the present study.

Conclusions

OCT-guided PCI did not increase the amount of contrast and incidence of DKF as compared with IVUS-guided PCI in actual clinical practice. Multicenter studies with a larger cohort may further clarify the impact of OCT-guided PCI on the incidence of DKF.

Disclosure

Conflicts of interest: Junya Ako and Yoshiyasu Minami received lecture fees from Abbott Vascular.

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