Proteinuria and Reduced Estimated Glomerular Filtration Rate Are Independent Risk Factors for Contrast-Induced Nephropathy After Cardiac Catheterization

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Background: The aim of this study was to investigate the incidence of contrast-induced nephropathy (CIN) according to renal function in patients with or without proteinuria after cardiac catheterization in Japan.

Methods and Results: We conducted a multicenter prospective observational study involving 27 hospitals from all over Japan, which enrolled 906 patients with cardiac catheterization. CIN was defined as increase in serum creatinine ≥0.5 mg/dl or ≥25% from baseline between 48 and 72 h after exposure to contrast. The incidence of CIN in patients with estimated glomerular filtration rate (eGFR) <60 ml/min/1.73 m² was significantly higher than that in patients with eGFR ≥60 ml/min/1.73 m². In patients without proteinuria, the incidence of CIN did not increase as eGFR decreased, but such a trend was observed in patients with proteinuria. Proteinuria was highly significantly associated with CIN in patients with eGFR 30–44 ml/min/1.73 m² (OR, 12.1; 95% CI: 2.81–82.8; P=0.0006) and eGFR <30 ml/min/1.73 m² (OR, 17.4; 95% CI: 3.32–321; P=0.0001). On multivariate logistic regression analysis, proteinuria (OR, 4.09; 95% CI: 1.66–10.0), eGFR (OR, 1.02; 95% CI: 1.00–1.04), contrast volume/eGFR (OR, 1.31; 95% CI: 1.04–1.65), and Ca antagonist use (OR, 3.79; 95% CI: 1.52–10.8) were significant predictors of CIN.

Conclusions: Proteinuria and reduced eGFR are independent risk factors for CIN after cardiac catheterization. (Circ J 2015; 79: 1624–1630)

Key Words: Contrast-induced nephropathy; Estimated glomerular filtration rate; Proteinuria

Contrast-induced nephropathy (CIN), a type of acute kidney injury, has been identified as the most frequent cause of hospital-acquired acute kidney injury.1–3 This iatrogenic complication has been a subject of concern for cardiologists in recent years, because CIN is associated with increased morbidity, mortality, and health-care expenditure.4,5 The incidence of CIN varies from 2% in low-risk populations to 50% in high-risk populations.6–8 Prior studies have shown that reduced renal function at baseline and large contrast volume are strong risk factors for CIN.9 Nowadays, most cardiologists are vigilant about preventing CIN after cardiac catheterization, for example, by ensuring adequate hydration before and after the procedure, minimizing the dose of contrast used, and using iso-osmolar or various low-osmolar contrast media.10–15

Editorial p 1456
In 2012, the Japan Radiology Society, the Japanese Circulation Society, and the Japanese Society of Nephrology jointly established guidelines on the use of contrast media in order to prevent CIN and the appropriate use of iodinated contrast media in patients with chronic kidney disease. Based on criteria most commonly used in prior studies, they defined CIN as an increase in serum creatinine (SCr) ≥0.5 mg/dl or ≥25% from baseline within 72 hours after contrast media exposure. At that time, we cardiologists were aware of the paucity of precise data on the incidence of CIN after cardiac catheterization because most patients are discharged from the hospital on the day of catheterization or the next day.

In Japan, more than half a million patients undergo coronary arteriography and approximately 180,000 patients undergo elective percutaneous intervention annually. They are often at high risk for CIN because their renal function is usually reduced due to diabetic nephropathy or nephrosclerosis. Therefore, risk stratification by renal function for CIN is clinically important. There were no prospective studies of CIN according to renal function in Japan, however, at that time. In this context, we conducted a study on CIN after cardiac catheterization in Japan (the CINC-J study), a multicenter prospective observational study to investigate the real-world incidence of CIN in patients stratified according to estimated glomerular filtration rate (eGFR) in 27 hospitals from all over Japan. Given that proteinuria is another important marker of renal dysfunction, we also investigated the effect of proteinuria on the development of CIN, which has not been previously investigated.

### Methods

#### Patient Enrollment

The CINC-J study is a prospective cohort study based on a multicenter registry involving 27 hospitals from all over Japan. We enrolled patients who underwent cardiac catheterization for diagnostic purposes, as well as elective and emergency percutaneous coronary intervention between November 2011 and September 2013. Patients who were already on dialysis at the time of the procedure were excluded. The type of contrast media and hydration protocols used were based on operator preferences, which were guided by institutional policies and practices. All procedures were performed using standard cardiac catheterization techniques.

At the time of enrollment, initially we planned to enroll 40 patients at each hospital: 10 patients with eGFR ≥60 ml/min/1.73 m² (normal renal function group), 10 patients with eGFR 45–59 ml/min/1.73 m² (mild renal dysfunction group), 10 patients with eGFR 30–44 ml/min/1.73 m² (moderate renal dysfunction group), and 10 patients with eGFR <30 ml/min/1.73 m² (severe renal dysfunction group). In this set of 40 patients, 10 consecutive patients were enrolled in each renal function group. After accruing the first set of 40 patients, investigators began to enroll subsequent sets of 40 patients. At the beginning we planned to accrue a total of 1,000 patients with equal numbers of patients in each subgroup.

Given that cardiac catheterization is less frequently performed in patients with severe renal dysfunction, however, there was an uneven distribution of renal function among the study participants: there were 339 patients with normal renal function, 271 patients with mild renal dysfunction, 254 patients...
with moderate renal dysfunction, and 129 patients with severe renal dysfunction. Eighty-seven patients were excluded because they did not have SCr measurements 48–72 h after contrast use. Ultimately, 906 patients were analyzed (Figure 1).

Clinical Definitions and Data Collection

In the present study, CIN was defined as an increase in SCr of 0.5 mg/dl or 25% from baseline between 48 and 72 h after contrast medium exposure. This definition is widely used and recommended in the Japanese guideline, but it is difficult to distinguish cholesterol embolization syndrome from CIN. eGFR was determined using the following equation:

\[ eGFR = \frac{194 \times SCr - 1.094 \times \text{age} - 0.287 \times 0.739 \text{ (if female)}}{1} \]

Baseline data, including clinical characteristics, laboratory data (blood and urine tests), and medication on admission, as well as procedural data were obtained for all patients. Blood tests included SCr on admission (baseline), the next day, and 48–72 h after the procedure. Dipstick urinalysis was performed with fresh spontaneously voided urine on admission. The results of urine tests were recorded as (–), (+), (1+), (2+), (3+), (–) and (+) were defined as proteinuria (–), and the rest were defined as proteinuria (+).

This study was approved by the ethics committee of each hospital, and written informed consent was obtained from all patients.

Table 1. Baseline Characteristics vs. Renal Dysfunction

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Normal (eGFR ≥60)</th>
<th>Mild (eGFR 45–59)</th>
<th>Moderate (eGFR 30–44)</th>
<th>Severe (eGFR &lt;30)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>150</td>
<td>120</td>
<td>150</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>68 (59–75)</td>
<td>70 (69–79)**</td>
<td>72 (69–79)**</td>
<td>76 (66.8–82)**</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male</td>
<td>78.1</td>
<td>79.8</td>
<td>76.2</td>
<td>69.7</td>
<td>0.2351</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.8 (21.4–26.1)</td>
<td>24.1 (21.8–26.4)</td>
<td>23.6 (21.2–26.4)</td>
<td>23.9 (21.1–26.0)</td>
<td>0.7317</td>
</tr>
</tbody>
</table>

**Risk factors**

Diabetes mellitus 40.6 45.1 48.1* 56.2** 0.0244
Hypertension 68.9 78.5* 82.0** 81.8** 0.0010
Dyslipidemia 67.9 66.5 64.4 57.9* 0.2528

**Diagnosis**

STEMI/NSTEMI 10.0 10.2 9.2 10.0 0.9861
CHF 14.2 21.8* 36.7** 47.9* <0.0001

**Laboratory data**

HbA1c (%) 6.3 (5.9–7.0) 6.3 (5.8–7.0) 6.5 (5.9–7.2) 6.4 (5.8–7.2) 0.5660
RBC (×10⁴/μl) 441.2±51.7 416.7±57.2** 393.2±59.7** 351.4±53.3** <0.0001
Hb (g/dl) 11.8 (12.6–14.7) 12.9 (11.9–14.0)** 12.0 (10.8–13.3)** 10.9 (10.0–12.0)** <0.0001
Proteinuria 4.4 12.3** 24.7** 53.2** <0.0001
BUN (mg/dl) 14.8±4.3 18.7±5.1** 24.9±8.7** 42.2±17.2** <0.0001
SCr (mg/dl) 0.75±0.14 1.03±0.14** 1.36±0.22** 2.43±1.00** <0.0001
eGFR (ml/min/1.73 m²) 77.8±16.6 52.5±4.3** 38.3±4.3** 21.9±6.0** <0.0001

**Procedural**

Emergency procedure 9.7 9.1 8.5 6.8 0.8196
PCI 49.4 48.1 43.9 33.9** 0.0254
Contrast volume (ml) 116 (75–165) 103 (70–150) 75 (40–120)** 45 (22–80)** <0.0001
Contrast volume/eGFR 1.52 (0.94–2.21) 2.06 (1.33–2.96)** 1.95 (1.01–3.09)** 2.20 (1.04–3.79)** <0.0001
Hydration 30.8 62.7 87.5** 93.4** <0.0001

**Medication**

Statin 56.3 56.9 58.8 52.1 0.6877
Ca antagonist 40.0 48.3 45.6 55.0** 0.0294
ACEI 20.6 21.6 26.4 20.8 0.3919
ARB 37.4 49.6** 45.6 53.3** 0.0058
α-blocker 3.6 4.3 3.4 10.0** 0.0219
β-blocker 40.0 45.7 50.8* 52.5* 0.0303
Diuretic 18.0 30.2** 47.9** 70.9** <0.0001

Data given as mean±SD, median (IQR) or %. *P<0.05 and **P<0.01 vs. the normal renal function group (categorical variables, χ² test; continuous variables, Dunnett’s test). ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; BMI, body mass index; BUN, blood urea nitrogen; CHF, congestive heart failure; eGFR, estimated glomerular filtration rate; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; RBC, red blood cells; SCr, serum creatinine; STEMI, ST-segment elevation myocardial infarction.

Statistical Analysis

Statistical analysis was done using JMP version 11 (SAS Institute, Cary, NC, USA). Continuous variables are presented as mean±SD or median (IQR). Categorical variables are presented as percentages. Normally distributed continuous variables were compared using one-way analysis of variance (ANOVA) and Dunnett test. Non-normally distributed continuous variables were compared using the Kruskal-Wallis and Steel tests. Categorical data were evaluated using the Person chi-squared test. Univariate logistic regression was performed to identify significant clinical predictors of CIN after cardiac catheterization. Multivariate logistic regression was used to identify independent predictors of CIN. Multi-
The prevalence of hypertension, diabetes mellitus, and heart failure was higher in patients with more severe renal dysfunction. Patients with renal dysfunction were more frequently treated with multiple medications such as calcium channel blockers, angiotensin receptor blockers, and diuretics. Statin was equally used among groups. Decreases in renal function were associated with increasing frequency of proteinuria. In the severe and moderate renal dysfunction groups, approximately one-half and one-quarter of the patients had proteinuria, respectively. The median volume of contrast media was only 45 ml in the severe renal dysfunction group and 75 ml in the moderate renal dysfunction group. The ratio of contrast volume to eGFR, however, varied across groups, with the highest value of 2.18 in the severe renal dysfunction group. Approximately 60%, 87%, and 93% of patients received isotonic hydration before and after catheterization with continuous

**Results**

**Baseline Clinical Characteristics**

In total, 310 patients were included in the normal renal function group, 235 in the mild renal dysfunction group, 239 in the moderate renal dysfunction group, and 122 in the severe renal dysfunction group in the present study. Baseline clinical and procedural characteristics are listed in Table 1. There were no differences in gender among the groups, but patients were older in the renal dysfunction groups compared with the normal renal function group. The prevalence of emergency cardiac catheterization was similar in the 4 groups of patients.
infusion of saline or sodium bicarbonate in the mild, moderate, and severe renal dysfunction groups, respectively.

Incidence of CIN
The incidence of CIN is given in Figure 2. No patients required hemodialysis. The incidence of CIN increased with decreasing renal function in all 4 groups (P=0.0001). The incidence of CIN in the mild and moderate renal dysfunction groups was 2.6% and 4.2%, respectively, which was similar to that in the normal renal function group (4.1%). The incidence of CIN in the severe renal dysfunction group, however, was 13.1%, which was significantly higher than in the other groups. Although the incidence of CIN was similar across the 4 groups among patients without proteinuria (normal renal function group, 4.3%; mild renal dysfunction group, 2.6%; moderate renal dysfunction group, 1.2%; severe renal dysfunction group, 2.0%; P=0.2668), there were significant differences in the incidence of CIN among patients with proteinuria in the 4 groups (normal renal function group, 0%; mild renal dysfunction group, 3.7%; moderate renal dysfunction group, 12.5%; severe renal dysfunction group, 25.9%; P=0.0136). The incidence of CIN was significantly higher in patients with proteinuria than without proteinuria in the moderate and severe renal dysfunction groups.

On logistic regression analysis a significantly higher risk of CIN was identified in patients with proteinuria among patients with moderate (OR, 12.1; 95% CI: 2.81–82.8; P=0.0006) and severe renal dysfunction (OR, 17.4; 95% CI: 3.32–321; P=0.0001), with patients without proteinuria as the reference group (Table 2).

Table 3. Predictors of CIN

<table>
<thead>
<tr>
<th>Predictor</th>
<th>CIN (+) n=45</th>
<th>CIN (-) n=861</th>
<th>Univariate OR (95% CI)</th>
<th>P-value</th>
<th>Multivariate OR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
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<tr>
<td>71 (63.5–76.5)</td>
<td>72 (65–78)</td>
<td>0.99 (0.96–1.02)</td>
<td>0.4970</td>
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<tr>
<td>Male</td>
<td>64.4</td>
<td>77.2</td>
<td>0.53 (0.29–1.03)</td>
<td>0.0592</td>
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<tr>
<td>Risk factors</td>
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<td></td>
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<tr>
<td>Diabetes mellitus</td>
<td>59.1</td>
<td>45.1</td>
<td>1.76 (0.96–3.30)</td>
<td>0.0698</td>
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<tr>
<td>Hypertension</td>
<td>88.6</td>
<td>76.0</td>
<td>2.46 (1.05–7.22)</td>
<td>0.0373</td>
<td>1.38 (0.39–6.66)</td>
<td>0.6350</td>
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<tr>
<td>Dyslipidemia</td>
<td>70.5</td>
<td>65.0</td>
<td>1.28 (0.68–2.58)</td>
<td>0.4526</td>
<td></td>
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<tr>
<td>Diagnosis</td>
<td></td>
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<tr>
<td>STEMI/NSTEMI</td>
<td>16.3</td>
<td>9.5</td>
<td>1.84 (0.73–4.04)</td>
<td>0.1793</td>
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<tr>
<td>CHF</td>
<td>42.2</td>
<td>25.8</td>
<td>2.10 (1.12–3.86)</td>
<td>0.0201</td>
<td>2.26 (0.87–5.86)</td>
<td>0.0952</td>
</tr>
<tr>
<td>Laboratory data</td>
<td></td>
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<tr>
<td>HbA1c (%)</td>
<td>6.8 (6.1–7.4)</td>
<td>6.3 (5.9–7.0)</td>
<td>1.23 (0.94–1.57)</td>
<td>0.1261</td>
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<tr>
<td>RBC (x10^4/μl)</td>
<td>395.4±70.5</td>
<td>411.1±62.2</td>
<td>0.996 (0.99–1.00)</td>
<td>0.1264</td>
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<tr>
<td>Hb (g/dl)</td>
<td>12.5 (10.8–13.2)</td>
<td>12.8 (11.3–14.1)</td>
<td>0.84 (0.71–0.998)</td>
<td>0.0471</td>
<td>0.96 (0.79–1.16)</td>
<td>0.6656</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>53.5</td>
<td>16.2</td>
<td>5.93 (3.17–11.2)</td>
<td>&lt;0.0001</td>
<td>4.09 (1.66–10.0)</td>
<td>0.0025</td>
</tr>
<tr>
<td>BUN (mg/dl)</td>
<td>25.9±11.4</td>
<td>22.0±12.3</td>
<td>1.02 (0.999–1.04)</td>
<td>0.0610</td>
<td></td>
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</tr>
<tr>
<td>SCr (mg/dl)</td>
<td>1.53±1.03</td>
<td>1.19±0.64</td>
<td>1.80 (1.16–2.12)</td>
<td>0.0056</td>
<td>1.53 (0.83–2.83)</td>
<td>0.1602</td>
</tr>
<tr>
<td>eGFR (ml/min/1.73m²)</td>
<td>46.6±27.4</td>
<td>53.7±22.2</td>
<td>0.98 (0.97–0.999)</td>
<td>0.0326</td>
<td>1.00 (1.00–1.04)</td>
<td>0.0381</td>
</tr>
<tr>
<td>Procedural</td>
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<tr>
<td>Emergency procedure</td>
<td>13.3</td>
<td>8.6</td>
<td>1.63 (0.61–3.72)</td>
<td>0.3071</td>
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<tr>
<td>PCI</td>
<td>33.3</td>
<td>46.2</td>
<td>0.58 (0.30–1.08)</td>
<td>0.0882</td>
<td></td>
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<tr>
<td>Contrast volume (ml)</td>
<td>90 (51.2–150)</td>
<td>95 (55–144)</td>
<td>1.00 (0.996–1.01)</td>
<td>0.7178</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contrast volume/eGFR</td>
<td>2.19 (1.23–3.07)</td>
<td>1.73 (1.08–2.66)</td>
<td>1.28 (1.06–1.52)</td>
<td>0.0133</td>
<td>1.31 (1.04–1.65)</td>
<td>0.0249</td>
</tr>
<tr>
<td>Hydration</td>
<td>72.7</td>
<td>62.0</td>
<td>1.64 (0.85–3.35)</td>
<td>0.1414</td>
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</tr>
<tr>
<td>Medication</td>
<td></td>
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</tr>
<tr>
<td>Statin</td>
<td>48.9</td>
<td>57.0</td>
<td>0.72 (0.39–1.32)</td>
<td>0.2875</td>
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<tr>
<td>Ca antagonist</td>
<td>72.1</td>
<td>44.3</td>
<td>3.25 (1.69–6.67)</td>
<td>0.0003</td>
<td>3.79 (1.52–10.8)</td>
<td>0.0037</td>
</tr>
<tr>
<td>ACEI</td>
<td>27.9</td>
<td>22.1</td>
<td>1.36 (0.66–2.64)</td>
<td>0.3873</td>
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<tr>
<td>ARB</td>
<td>60.5</td>
<td>44.1</td>
<td>1.94 (1.05–3.89)</td>
<td>0.0355</td>
<td>1.27 (0.57–2.89)</td>
<td>0.5627</td>
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<tr>
<td>α-blocker</td>
<td>11.6</td>
<td>4.2</td>
<td>2.99 (0.99–7.44)</td>
<td>0.0528</td>
<td></td>
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</tr>
<tr>
<td>β-blocker</td>
<td>41.9</td>
<td>46.2</td>
<td>0.84 (0.44–1.55)</td>
<td>0.5773</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretic</td>
<td>60.5</td>
<td>34.8</td>
<td>2.87 (1.54–5.46)</td>
<td>0.0009</td>
<td>2.04 (0.79–5.26)</td>
<td>0.1415</td>
</tr>
</tbody>
</table>

Data given as mean±SD, median (IQR) or %. Abbreviations as in Tables 1,2.
Discussion

In the present study, the incidence of CIN was significantly higher in patients with eGFR <30 ml/min/1.73 m² compared with those with eGFR ≥60 ml/min/1.73 m². The incidence of CIN, however, in patients with eGFR 45–59 ml/min/1.73 m² or 30–44 ml/min/1.73 m² was similar to that in patients with eGFR ≥60 ml/min/1.73 m². In the majority of prior studies, mild-moderate renal dysfunction with eGFR <60 ml/min/1.73 m² was a risk for CIN.2,10–18 Although the cause of the discrepancy is not clear at this time, the smaller volume of contrast used may contribute to the prevention of CIN in patients with eGFR 30–59 ml/min/1.73 m² in the present study.

The most striking result of the present study is the effect of proteinuria on the development of CIN. In patients without proteinuria, reduced eGFR itself did not affect the incidence of CIN. The incidence of CIN increased with worsening eGFR, however, only in patients with proteinuria. Moderate (eGFR 30–44 ml/min/1.73 m²) and severe (eGFR <30 ml/min/1.73 m²) renal dysfunction were significant risk factors for CIN in patients with proteinuria. As far as we know, there was only one retrospective study showing proteinuria as a significant predictor of CIN in trauma patients who underwent contrast-enhanced imaging.19 Thus, this is the first report to show that proteinuria is a strong risk factor for CIN in patients who undergo cardiac catheterization.

Given that approximately 90% of patients with moderate-severe renal dysfunction received hydration before and after contrast use, hydration itself did not prevent development of CIN in patients with proteinuria. The present study, however, cannot resolve the question of whether hydration could reduce the incidence of CIN in patients with proteinuria or whether hydration is necessary for prevention of CIN in patients without proteinuria because there were no relevant control groups. New methods to prevent CIN should be developed for patients with proteinuria and renal dysfunction.

Regarding the predictors of CIN other than renal function and contrast volume, the present analysis suggested that the use of Ca channel blocker was significantly associated with CIN, although it had not been reported before. Other anti-hypertensive drugs such as renin angiotensin-converting enzyme inhibitor and angiotensin receptor blocker did not significantly increase the incidence of CIN, with OR of 1.36 and 1.96, respectively. The recent Japanese guidelines say that renin-angiotensin system blockers do not increase or decrease the incidence of CIN.16 The mechanism for Ca channel blockers in CIN development is not clear at present. Renal hemodynamic changes due to anti-hypertensive drugs, however, might influence blood flow to the renal medulla. As for statins, the present study did not show the beneficial effect of statin use on CIN development. Although the recent meta-analysis indicated that statin use reduces the incidence of CIN, another one did not identify a beneficial effect of statin on prevention of CIN.21 Further studies are necessary to elucidate the effect of drugs on the development of CIN.

Several other clinical factors, such as diabetes mellitus and emergency CAG, which were risks for CIN in previous studies,7,21,22 did not increase the risk of CIN significantly in the present study. Although the reason for this discrepancy is not clear at present, one possible explanation is the low number of subjects. Another one is that the present study was not conducted to investigate the effect of such factors on the development of CIN.

The mechanism underlying the development of CIN is not fully understood. It is believed, however, that contrast media use induces vasoconstriction and renal medullary ischemia, which generates radical oxygen species leading to tubular injury.19,23 The recent finding that elevation of N-acetyl-β-glucosaminidase is an early marker of CIN (which we did not measure), supports the involvement of tubular injury in the development of CIN.24 In the present study, we measured urine protein using a dipstick method, not urine albumin itself. Proteinuria, however, usually indicates albuminuria. The main mechanism of albuminuria is generally thought to be overexcretion of albumin from the glomerulus; thus, albuminuria is a marker of glomerular injury. A substantial amount of albumin, however, is excreted from the glomerulus even in the normal kidney, but it is almost completely reabsorbed by the proximal tubules.25–27 Thus, positive albuminuria indicates a high concentration of albumin in the tubules, which overcomes the capacity for reabsorption by the tubules.27 When contrast is used in such conditions, the additive effect of albumin and contrast media may result in higher osmotic pressure, which leads to more severe tubular injury. Of course, more precise experiments are needed to investigate the pathology of CIN in the presence of proteinuria.

CIN is generally transient and patients usually recover to pre-procedural levels of renal function. Sometimes kidney injury is persistent, however, and associated with poor outcome.28–30 Recently, eGFR and albuminuria were identified as independent predictors of cardiovascular events.31–35 In the present study, CIN was observed in patients with reduced eGFR and proteinuria, who are also at high risk for cardiovascular events. Thus, a long-term follow-up study is necessary.

Conclusions

Proteinuria and reduced eGFR are independent risk factors for CIN after cardiac catheterization.

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Disclosures

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