Prognostic Values of C-Reactive Protein Levels on Clinical Outcome After Implantation of Sirolimus-Eluting Stents in Patients on Hemodialysis

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Background—Percutaneous coronary intervention (PCI) using drug-eluting stents significantly reduces the risk of restenosis in the general population. However, in patients on hemodialysis, adverse cardiac events are frequently seen even if treated with drug-eluting stents. Recent studies suggest that C-reactive protein (CRP) reflects vascular wall inflammation and can predict adverse cardiac events. We evaluated possible prognostic values of CRP on outcomes in patients on hemodialysis undergoing PCI with drug-eluting stents.

Methods and Results—A total of 167 patients undergoing PCI with sirolimus-eluting stents for stable angina (322 lesions) were enrolled. They were divided into tertiles according to serum CRP levels. We analyzed the incidence of major adverse cardiovascular events including cardiovascular death, nonfatal myocardial infarction, and target lesion revascularization after PCI as well as quantitative coronary angiographic data. The mean follow-up was 31 months (SD, 14). Major adverse cardiac events occurred in 11 patients (19.6%) of the lowest tertile, in 22 patients (39.3%) of the middle tertile, and in 28 patients (50.9%) of the highest tertile during follow-up period (P=0.0009). There was a progressive increase in neointimal growth after sirolimus-eluting stent implantation during follow-up because preprocedural CRP levels were higher, despite similar angiographic data just after PCI. Angiographic restenosis at 6 to 8 months after PCI was seen in 10.6% in the lowest tertile, 17.9% in the middle tertile, and 32.0% in the highest tertile (P=0.0007).

Conclusions—Increased preprocedural serum CRP levels would predict higher major adverse cardiac events and restenosis rates after sirolimus-eluting stents implantation in patients on hemodialysis. (Circ Cardiovasc Interv. 2009;2:513-518.)

Key Words: kidney ■ follow-up studies ■ prognosis ■ stents ■ inflammation

Studies have indicated that drug-eluting stents (DES) significantly reduce the risk of restenosis after percutaneous coronary intervention (PCI) in many cases.1-5 Angiographic improvement at follow-up phase has been observed even in patients with high risk factors for coronary restenosis, including diabetes, small diameter in vessels, chronic total occlusion, and so on, which were associated with a higher restenosis rate after PCI in bare metal stent era.6-9 However, restenosis at follow-up phase is often seen even in patients treated with DES. Particularly, patients on maintenance hemodialysis (HD) are at high risk for restenosis after DES implantation.10,11 In such patients, it is attractive to find a simple clinical method to predict restenosis after DES implantation. Until now, prognostic values of angiographic restenosis after DES implantation in such patients have not been fully evaluated.

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Recent studies have suggested that C-reactive protein (CRP) reflects vascular wall inflammation and can predict adverse cardiac events.12,13 In patients with renal dysfunction, serum CRP levels that may be related to vascular inflammatory reaction are often increased.14 Therefore, we investigated a potential relationship between serum CRP levels and clinical events after DES implantation in patients on maintenance HD.
Methods

Study Population
This study consisted of 167 patients on HD who were treated with successful PCI using a sirolimus-eluting stent (SES) (Cypher, Cordis Corp, Warren, NJ) for stable angina pectoris (322 lesions) between August 2004 and October 2007. All patients had at least one of the following signs of ischemia: (i) ST depression >1 mV on a treadmill exercise test; (ii) ST depression >1 mV on a bicycle exercise test; and (iii) clinical symptoms greater than the Canadian Cardiovascular Society Classification II. Exclusion criteria included patients who were treated for bypass graft lesions, those with a contraindication for the use of aspirin or thienopyridine including ticlopidine or clopidogrel, those who had cancer or active inflammation, and those who were >85 years. Dual antiplatelet therapy (aspirin [162 mg/d] and ticlopidine [200 mg/d] or clopidogrel [75 mg/d]) was started at least 2 weeks before PCI after checking the tolerance of those drugs and followed-up for at least 1 year after PCI. All were de novo lesions of ≥50% diameter stenosis in a native coronary artery >2.5 mm in diameter based on a on-line quantitative coronary angiography (QCA) analysis. Lesions were treated with the use of standard PCI techniques after 7000 to 10 000 U heparin injection into artery. Fasting blood samples from vein were obtained on the morning of the day of the PCI procedure. Serum CRP levels were measured using a latex-enhanced high-sensitive CRP immunosassay. The primary end point was major adverse cardiac events (MACE) after PCI procedure, including cardiovascular death, nonfatal myocardial infarction, and target lesion revascularization due to stent restenosis-induced ischemia. We also evaluated angiographic data such as rates of restenosis at 6 to 8 months after PCI. The definition of myocardial infarction included new pathological Q-wave/ST-T-wave changes on electrocardiography or more than a 2-fold increase in creatine kinase above the maximum value of the normal range. Restenosis was defined as a stent stenosis >50% decrease in diameter anywhere within the stent and/or within the 5-mm borders proximal or distal to the stent. Target lesion revascularization was performed only if patients had both coronary angiographic restenosis >50% diameter stenosis by QCA and signs of coronary ischemia. The study was in agreement with the guidelines of the ethics committee of our institution, and written informed consent was obtained from each patient.

QCA
Patients received intracoronary administration of 2.5 to 5 mg isosorbide dinitrate or 2 mg nicorandil before the initial, final, and follow-up angiograms to achieve maximal vasodilatation. Image calibration using contrast-filled guiding catheters was the reference standard. We measured reference vessel diameter, minimum luminal diameter, and percent diameter stenosis from the single worst view, which were matched end-diastolic frames of angiograms before and after PCI and at the follow-up period by using a contour detection and a multivariate covariates associated with progression of all patients to the primary end point. For restenosis at follow-up, a logistic model was used. Hazard ratios and CIs were calculated for each factor by a Cox proportional hazards analysis and a logistic analysis. All the prognostic values with P<0.10 were entered into a Cox multivariable model to determine independent predictors. Differences were considered significant at P<0.05. The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Statistical Analysis
All the analyses were performed using a software program, Stat View 5.0 (SAS Institute, Cary, NC). The event-free survival rates for each end point among the groups were examined with the Kaplan–Meier method, and the differences in survival rates among the groups were compared using the log-rank test. For comparison of the baseline and outcome data among multiple groups, ANOVA and Fisher protected least significant difference test were used for quantitative variables, and \( \chi^2 \) test was used for categorical variables. We used Cox proportional hazards models to assess univariate and multivariable covariates associated with progression of all patients to the primary end point. For restenosis at follow-up, a logistic model was used. Hazard ratios and CIs were calculated for each factor by the log-rank test. For comparison of the Meier method, and the differences in survival rates among the groups each end point among the groups were examined with the Kaplan–Meier method, and the differences in survival rates among the groups.

Table 1. Baseline Patient Characteristics

<table>
<thead>
<tr>
<th>Tertile 1: &lt;1.0 mg/L (n=56)</th>
<th>Tertile 2: 1.0–4.3 mg/L (n=56)</th>
<th>Tertile 3: &gt;4.3 mg/L (n=55)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>60.7</td>
<td>71.4</td>
<td>69.1</td>
</tr>
<tr>
<td>Age, y</td>
<td>63±10</td>
<td>65±9</td>
<td>65±10</td>
</tr>
<tr>
<td>Duration of dialysis, y</td>
<td>7.1±8.1</td>
<td>6.9±7.1</td>
<td>7.6±9.1</td>
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<tr>
<td>Diabetes</td>
<td>58.9</td>
<td>53.6</td>
<td>54.5</td>
</tr>
<tr>
<td>Hypertension</td>
<td>42.9</td>
<td>51.8</td>
<td>56.4</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>19.6</td>
<td>17.9</td>
<td>12.7</td>
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<tr>
<td>Previous stroke</td>
<td>12.5</td>
<td>14.3</td>
<td>21.8</td>
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<tr>
<td>Previous peripheral artery disease</td>
<td>10.7</td>
<td>19.6</td>
<td>21.8</td>
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<tr>
<td>Previous myocardial infarction</td>
<td>3.6</td>
<td>8.9</td>
<td>7.3</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>55.3</td>
<td>51.8</td>
<td>54.5</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>10.2±1.1</td>
<td>10.4±1.1</td>
<td>10.3±1.4</td>
</tr>
<tr>
<td>Serum albumin, g/dL</td>
<td>3.5±0.3</td>
<td>3.6±0.3</td>
<td>3.4±0.3</td>
</tr>
<tr>
<td>Calcium, mg/dL</td>
<td>8.7±0.5</td>
<td>8.8±0.9</td>
<td>8.7±1.2</td>
</tr>
<tr>
<td>Phosphate, mg/dL</td>
<td>5.1±1.4</td>
<td>5.3±1.5</td>
<td>5.1±1.4</td>
</tr>
<tr>
<td>Ca×Pi, mg/dL²</td>
<td>45.2±12.5</td>
<td>47.5±14.3</td>
<td>44.7±13.1</td>
</tr>
<tr>
<td>Intact PTH, pg/mL</td>
<td>138±171</td>
<td>102±103</td>
<td>136±85</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>169±31</td>
<td>159±26</td>
<td>164±42</td>
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<tr>
<td>LDL cholesterol, mg/dL</td>
<td>94±24</td>
<td>88±26</td>
<td>90±28</td>
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<tr>
<td>HDL cholesterol, mg/dL</td>
<td>39±11</td>
<td>36±15</td>
<td>40±10</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ACE inhibitors</td>
<td>10.7</td>
<td>7.1</td>
<td>16.4</td>
</tr>
<tr>
<td>A-II receptor blockers</td>
<td>50.0</td>
<td>58.9</td>
<td>54.5</td>
</tr>
<tr>
<td>β-blockers</td>
<td>19.6</td>
<td>17.9</td>
<td>25.5</td>
</tr>
<tr>
<td>Ca antagonists</td>
<td>42.9</td>
<td>48.2</td>
<td>50.9</td>
</tr>
<tr>
<td>Statins</td>
<td>25.0</td>
<td>28.6</td>
<td>25.5</td>
</tr>
</tbody>
</table>

Data are presented as % or mean±SD. Ca×Pi indicates product of serum calcium and phosphate; iPTH, intact parathyroid hormone; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ACE, angiotensin-converting enzyme; A-II, angiotensin II.

Results
Enrolled patients were divided into tertiles according to the serum CRP levels (first tertile [T1]: <1.0 mg/L, second tertile [T2]: 1.0 to 4.3 mg/L, and third tertile [T3]: >4.3 mg/L). The follow-up was concluded on March 31, 2009. The mean duration of follow-up was 31 months (SD, 14). The baseline characteristics and lesion characteristics are shown in Tables 1 and 2, respectively. There were no differences in age, sex, duration of HD, serum variables, incidence of coronary risk factors, lesion location, and American Heart Association/ American College of Cardiology type among the 3 groups. However, there was a significant positive correlation between the serum CRP levels and calcification of coronary lesion.
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Table 2. Baseline Target Lesion Characteristics

| Tertile 1: <1.0 mg/L (n=89) | Tertile 2: 1.0–4.3 mg/L (n=125) | Tertile 3: >4.3 mg/L (n=108) | P  
|---|---|---|---|
| Lesion location  
Right coronary artery | 29.9 | 33.3 | 38.3 | 0.52  
Left anterior descending artery | 47.1 | 43.9 | 43.9 |  
Left circumflex artery | 21.8 | 21.1 | 14.1 |  
Left main trunk | 1.2 | 1.7 | 3.7 |  
| AHA/ACC type  
A | 4.5 | 2.4 | 2.8 | 0.32  
B1 | 9.0 | 13.6 | 13.9 |  
B2 | 67.4 | 52.8 | 59.3 |  
C | 19.1 | 31.2 | 21.0 |  
| Minimum lesion diameter, mm | 1.01±0.36 | 1.01±0.47 | 1.03±0.46 | 0.96  
| Reference, mm | 2.8±0.5 | 2.8±0.6 | 2.8±0.6 | 0.74  
| Lesion length, mm | 17.1±5.0 | 17.5±5.6 | 17.1±4.5 | 0.81  
| Diffuse lesion | 32.6 | 37.6 | 40.7 | 0.49  
| Bifurcation lesion | 18.3 | 22.4 | 23.1 | 0.64  
| Calcified lesion | 38.2 | 62.4 | 88.8 | <0.0001  
| Small vessel (<2.5 mm) | 24.7 | 32.0 | 30.6 | 0.49  
| Maximum inflation pressure, atm | 16.7±0.41 | 16.0±0.36 | 16.5±0.38 | 0.67  
| Stent diameter, mm | 3.0±0.4 | 3.0±0.4 | 3.1±0.4 | 0.21  
| Stent length, mm | 18.3±4.6 | 19.5±4.9 | 18.9±4.7 | 0.18  

Data are presented as % or mean±SD. AHA/ACC indicates American Heart Association/American College of Cardiology.

Clinical follow-up data were obtained from all enrolled patients. Figure 1 shows the Kaplan–Meier survival curve for composite end point of MACE. The 4-year rates for primary end point defined as cardiovascular death, nonfatal myocardial infarction, and target lesion revascularization were 22.1% in the lowest tertile, 50.3% in the middle tertile, and 61.2% in the highest tertile by Kaplan–Meier survival rate (P=0.0009; Table 3). In the multivariable model including the variable with P<0.10 by the univariate model, serum CRP levels and calcified lesion were independent predictors of MACE and cardiovascular death (Table 4). Target lesion revascularization for the PCI site was needed in 14.3% of patients in the lowest tertile, 25.0% in the middle tertile, and in 36.4% in the highest tertile (P=0.0099 by Kaplan–Meier analysis; Figure 2).

![Kaplan–Meier survival curve](https://example.com/kaplan-meier.png)

**Figure 1.** Kaplan–Meier estimates: event-free survival from composite end points of cardiovascular death, nonfatal myocardial infarction, and target lesion revascularization.

Table 3. MACE During Follow-Up Period

| Tertile 1: <1.0 mg/L (n=56) | Tertile 2: 1.0–4.3 mg/L (n=56) | Tertile 3: >4.3 mg/L (n=55) | P  
|---|---|---|---|
| Cardiovascular death | 4 (9.5) | 11 (28.7) | 14 (35.4) | 0.031  
| Nonfatal MI | 0 (0.0) | 0 (0.0) | 3 (6.1) | NA  
| TLR | 8 (15.7) | 14 (28.8) | 20 (43.0) | 0.0099  

Values in parentheses represent event rate by the Kaplan–Meier analysis. MI indicates myocardial infarction; NA, not applicable; TLR, target lesion revascularization.

A complete follow-up coronary angiography was obtained from 149 patients (89.2%) with 288 lesions (89.4%). Although there were no significant differences in pre- and postprocedural QCA data, late loss and percent diameter stenosis at follow-up phase were significantly higher in patients in 2 higher tertiles compared with those in the lowest group (Table 5). This means that neointimal hyperplasia was much more in the 2 higher tertiles than the lowest tertile. The restenosis rate at follow-up angiography was 10.1% in the lowest tertile, 17.6% in the middle tertile, and 32.4% in the highest tertile, respectively (P=0.0003).

We performed a logistic regression analysis to determine predictive risks for restenosis after SES implantation. In a univariable logistic analysis, the highest tertile of serum CRP levels and calcified lesion were significant risk factors for restenosis after SES implantation in patients on HD. Even after adjusting for other risk factors at baseline, the highest tertile of serum CRP levels was statistically significant in a multivariable logistic analysis (hazard ratio, 3.92; 95% CI, 1.69 to 9.03; P=0.0014). Other independent predictors were

Table 4. Predictive Values for Clinical Outcome by Cox Analysis

| MACE  
|---|---|---|---|
| Nonadjusted HR (95% CI) | P  | Adjusted HR (95% CI) | P  
| Serum CRP (vs T1) | 2.13 (1.03 to 4.39) | 0.040  | 2.08 (1.01 to 4.30) | 0.048  
| T2 | 3.45 (1.72 to 6.94) | 0.0005  | 3.36 (1.67 to 6.78) | 0.0027  
| T3 | 1.07 (1.01 to 1.13) | 0.021  | 1.02 (1.01 to 1.05) | 0.036  
| Albumin, g/dL | 0.40 (0.16 to 1.01) | 0.054  | 0.63 (0.19 to 2.10) | 0.45  

| Cardiovascular death  
|---|---|---|---|
| Nonadjusted HR (95% CI) | P  | Adjusted HR (95% CI) | P  
| Serum CRP (vs T1) | 2.54 (0.81 to 7.99) | 0.10  | 2.35 (0.74 to 7.38) | 0.14  
| T2 | 4.01 (1.32 to 12.2) | 0.014  | 3.61 (1.18 to 10.1) | 0.024  
| T3 | 1.05 (1.01 to 1.09) | 0.019  | 1.05 (1.01 to 1.10) | 0.026  
| Albumin, g/dL | 0.26 (0.08 to 0.88) | 0.029  | 0.37 (0.11 to 1.26) | 0.11  

| Target lesion revascularization  
|---|---|---|---|
| Nonadjusted HR (95% CI) | P  | Adjusted HR (95% CI) | P  
| Serum CRP (vs T1) | 1.79 (0.75 to 4.26) | 0.19  | 1.94 (0.81 to 4.64) | 0.13  
| T2 | 3.17 (1.40 to 7.21) | 0.0058  | 3.39 (1.48 to 7.78) | 0.0039  
| T3 | 3.58 (1.02 to 8.33) | 0.034  | 3.34 (1.03 to 10.9) | 0.045  

* T indicates tertile; HR, hazard ratio.  
*P value for trend.
enhanced with renal dysfunction. In addition, the rates of restenosis after PCI with SES is seen as renal function deterioration. Several coronary risk factors including hyperlipidemia, hypertension, diabetes, and so on are common in patients on maintenance HD. All these mechanisms might be related to a higher restenosis rate after DES implantation in patients on HD. However, it is not clear why patients on HD have such a high restenosis rate after DES implantation. We hypothesized that a worse clinical outcome and a higher restenosis might be frequently seen in patients with higher serum CRP levels, which might reflect vascular inflammation and atherosclerosis.

Studies have suggested that inflammatory processes are related to atherosclerosis progression, and that there is an association between inflammation and atherosclerotic plaque burden. CRP itself reportedly promotes atherogenesis and atherothrombosis. In the bare metal stent era, although reports showed that there was no association between CRP levels and angiographic restenosis, increased serum CRP levels predicted restenosis. In the DES era, stent implantation reduces the risk of restenosis after PCI in many cases. Therefore, in cases with higher serum CRP levels, DES has been the most expected device to use. However, this study suggests that even when treated with SES, a higher restenosis rate was seen in patients with high CRP levels. Therefore, CRP levels are strong predictors of restenosis in patients on HD. Park et al previously reported that preprocedural CRP levels were not predictive value for clinical outcomes in patients without HD (data not shown). Patients with end-stage renal disease have such a high restenosis rate after DES implantation. However, about half of the patients enrolled in their study had acute coronary syndrome. Furthermore, only limited numbers of patients had renal insufficiency. On the other hand, this study consisted of only patients with stable angina pectoris on HD. Indeed, in our institution, serum CRP levels were not predictive for clinical outcomes in patients without HD (data not shown). Patients with end-stage renal disease have characterized conditions for severe inflammatory processes. Furthermore, increased CRP concentrations have been associated with worse clinical outcomes. We showed a higher incidence of neointimal hyperplasia as CRP levels were more increased in this study. This phenomenon might explain the association between higher CRP levels and higher rates of adverse cardiac events in patients with ischemic heart disease on HD.

In this study, calcified lesion and lesion length also predicted angiographic restenosis. In agreement with a previous study, high CRP levels were associated with progressive calcified lesions in patients on HD. We also provided

Table 5. Results of QCA in Target Lesions

<table>
<thead>
<tr>
<th></th>
<th>Tertile 1:</th>
<th>Tertile 2:</th>
<th>Tertile 3:</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;1.0 mg/L</td>
<td>1.0–4.3 mg/L</td>
<td>&lt;4.3 mg/L</td>
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</tr>
<tr>
<td>Reference vessel diameter, mm</td>
<td>2.8±0.5</td>
<td>2.8±0.6</td>
<td>2.8±0.6</td>
<td>0.74</td>
</tr>
<tr>
<td>Minimum lesion diameter, mm</td>
<td>Pre 1.01±0.36</td>
<td>1.01±0.47</td>
<td>1.03±0.46</td>
<td>0.96</td>
</tr>
<tr>
<td></td>
<td>2.63±0.47</td>
<td>2.65±0.49</td>
<td>2.69±0.46</td>
<td>0.68</td>
</tr>
<tr>
<td></td>
<td>2.47±0.62</td>
<td>2.14±0.83*</td>
<td>1.99±0.85*</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>0.17±0.61</td>
<td>0.55±0.73*</td>
<td>0.69±0.89*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diameter stenosis, %</td>
<td>Pre 62.8±15.2</td>
<td>63.2±15.4</td>
<td>63.1±19.6</td>
<td>0.98</td>
</tr>
<tr>
<td></td>
<td>10.1±11.9</td>
<td>9.9±8.6</td>
<td>10.3±7.9</td>
<td>0.92</td>
</tr>
<tr>
<td></td>
<td>16.0±18.0</td>
<td>25.2±26.5*</td>
<td>30.3±28.1*</td>
<td>0.0005</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD. *P<0.05 versus tertile 1.
predictive value of calcified lesion for higher restenosis rate. Patients with long lesions sometimes have several coronary risk factors. In bare metal stent era, diffuse or long coronary lesions have been considered as high risk for percutaneous intervention. In general, PCI for diffuse or long lesions need long stent or multiple stents. These procedures are often related to stent fracture after DES implantation. Because restenosis and target lesion revascularization rates for the coronary lesions with stent fracture are higher than those for the lesions without stent fracture. Therefore, it is quite natural that lesion length is significantly related to restenosis after SES implantation.

It is well known that the potential benefit of the use of statins, 3-hydroxy-3-methylglutaryl-CoA reductase inhibitors, reduces inflammation in a nondialysis population. However, until now, there have been limited data to show beneficial effects of statins on improvement of clinical outcome data in patients on maintenance HD. Four-dimensional study and recent A Study to Evaluate the Use of Rosuvastatin in Subjects on Regular Haemodialysis: An Assessment of Survival and Cardiovascular Events (AURORA) study did not prove the beneficial effects of statins on patients on dialysis. In this study, treatment with statins was not a predictor of clinical outcomes. However, further investigation is needed in this context.

Several limitations need to be considered with respect to our study. First, this study was of single-center design, and only a limited number of patients were enrolled. Second, we enrolled only patients with stable angina on HD. Patients with acute coronary syndrome were not assessed. Third, only SES was used as DES in this study. We have no data on other DESs, such as paclitaxel-eluting stent, zotarolimus-eluting stent, tacrolimus-eluting stent, and so on, because such DESs were not available during the study period in Japan. Finally, we could not analyze angiographic follow-up for all patients but only in 89% of patients. Therefore, a selection bias might exist, although angiographic follow-up rates were higher than in previous studies regarding the relationship between CRP levels and clinical outcome after PCI.

Conclusions

Because restenosis after PCI in patients on maintenance HD remains a major clinical problem even in DES era, investigators have been attempting to develop a simple method to predict patients likely to have restenosis. We have shown that serum CRP and lesion length were independent predictors for MACE and restenosis after SES implantation in this patient population. Our results could provide additional information to detect high-risk patients on HD requiring very careful attention.

Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Accelerated atherosclerosis with coronary events is common in long-term survivors receiving hemodialysis. Patients with end-stage renal failure on hemodialysis are at high risk of restenosis after percutaneous coronary intervention (PCI), even in the drug-eluting stent era, although the initial success rate of PCI is high. Therefore, higher restenosis after PCI remains a clinical limitation in patients on hemodialysis. Recently, it is well known that C-reactive protein reflects vascular wall inflammation and can predict adverse cardiac events in general population. This study evaluated prognostic values of C-reactive protein on outcomes in patients on hemodialysis undergoing PCI with sirolimus-eluting stents and observed that increased preprocedural serum C-reactive protein levels would predict not only higher major adverse cardiac events rates but also higher restenosis rates after PCI. There was a progressive increase in neointimal growth after sirolimus-eluting stent implantation during follow-up because preprocedural C-reactive protein levels were higher, despite similar angiographic data just after PCI. The results of this study may provide additional information to detect high-risk patients on maintenance hemodialysis who require PCI for ischemic heart disease.
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