Associations Between Plasma C-reactive Protein Levels and the Severities of Coronary and Aortic Atherosclerosis

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Aim: Limited correlations between C-reactive protein (CRP) and coronary artery disease (CAD) have been reported. Recently, MRI became a useful tool for non-invasively evaluating atherosclerotic plaques in thoracic and abdominal aortas.

Methods: To elucidate the associations between plasma CRP levels and the severities of coronary and aortic atherosclerosis, we performed aortic black-blood MRI in 136 patients undergoing coronary angiography. For each patient, 9 slices of thoracic aorta and 9 slices of abdominal aorta were obtained at 12-mm intervals, and the plaque extent in each slice was scored. The degree of aortic atherosclerosis is represented as the sum of scores. The degree of coronary atherosclerosis is represented as the number of >50% stenotic vessels and >25% stenotic segments.

Results: CAD (>50% stenosis) was present in 96 patients. Patients with CAD had higher CRP levels than those without CAD (median 0.78 vs. 0.48 mg/L, p<0.02). CRP levels tended to increase depending on the number of stenotic vessels: 0.48, 0.70, 0.74, and 0.88 mg/L (p=NS). CRP correlated weakly with the number of stenotic segments (r=0.21). Regarding aortic atherosclerosis, 136 patients were divided into quartiles by plaque score. CRP levels increased stepwise in quartiles: 0.40, 0.56, 1.08, and 1.10 mg/L (p<0.001). CRP levels also correlated with the plaque score (r=0.38). In multivariate analysis, aortic atherosclerosis was an independent factor for CRP levels, but coronary atherosclerosis was not.

Conclusion: Plasma CRP levels correlated with the severities of both coronary and aortic atherosclerosis, but CRP levels are more likely to reflect the severity of aortic atherosclerosis than coronary atherosclerosis.


Key words: Aortic plaque, Coronary artery disease, C-reactive protein, MRI

Introduction

Inflammation plays an important role in both the initiation and progression of coronary artery disease (CAD)¹, ². High sensitivity C-reactive protein (hsCRP) levels have been reported to predict further cardiovascular events in patients with CAD³-⁵; however, the association between hsCRP levels and the severity of coronary atherosclerosis in patients with CAD remains controversial. Some studies have reported significant associations between hsCRP levels and the severity of coronary stenosis⁴-⁶, whereas others have not⁷-⁹. We previously reported the significant correlation between plasma hsCRP levels and the severity of coronary stenosis in 273 patients with stable CAD¹⁰; however, the correlations were weak (r=0.20–
CRP Levels and Aortic Atherosclerosis

Methods

Study Patients

Our study consisted of 136 patients (105 men; mean age 64 ± 9 years, range 40 to 85 years) undergoing elective coronary angiography for suspected or known CAD at the National Defense Medical College Hospital, of whom 102 were included in our previous study.14 Patients with acute coronary syndrome, with a history of cardiovascular surgery, with aortic disease, valvular heart disease or congenital heart disease, or with any inflammatory disease or malignancy were excluded from our study, which was approved by the institutional ethics committee. After written informed consent was obtained, aortic MRI was performed at Iruma Heart Hospital within 2 weeks of angiography. Of the 136 patients, 85 (63%) had hypertension (blood pressure ≥140/90 mmHg or on drugs), of whom 66 were on drugs, and 77 (57%) had hyperlipidemia (total cholesterol >240 mg/dL or on drugs), of whom 53 were taking statins. Diabetes mellitus (fasting plasma glucose ≥126 mg/dL or on hypoglycemic drugs or insulin) was present in 34 (25%) patients, and 55 (41%) were smokers (≥10 packs-year).

Blood samples were taken in a fasting state on the morning of the day when coronary angiography was performed. Serum lipid levels were measured by standard laboratory methods. Plasma hsCRP levels were measured using a BNII nephelometer (Dade Behring, Tokyo, Japan).

Coronary Angiography

Coronary angiograms were recorded using the Judkins technique and a cineangiogram system (Toshiba, Tokyo, Japan). Coronary artery segments were defined according to the Coronary Artery Surgery Study (CASS) classification. The degree of stenosis in each segment was evaluated by 5 grades (≤25%, 25–50%, 51–75%, 76–90%, >90% stenosis). CAD was defined as at least one coronary artery having >50% luminal diameter stenosis on angiograms. Of the 136 study patients, 96 (71%) had CAD, of whom 26 had a history of percutaneous coronary intervention (PCI) >6 months previously. In patients with a history of PCI, the degree of stenosis in the segment where PCI had been performed was defined as the degree of stenosis before PCI. All angiograms were evaluated by Y.M., who was blinded to the MRI data. Intra-observer agreement for the assessment of the grade of stenosis was 98% of segments. The severity of coronary atherosclerosis was represented as the number of >50% stenotic vessels, >50% stenotic segments, and >25% stenotic segments.

MRI of the Aorta

Aortic MRI was performed on a Signa 1.5T Cvi scanner with a phased-array body coil (GE Medical Systems, Mount Prospect, USA). The transverse proton density-weighted (PDW) and T2-weighted (T2W) images of the thoracic descending and abdominal aortas were obtained using an ECG-gated, double-inversion-recovery fast spin-echo sequence. The imaging parameters were TR = 2 RR intervals, TE = 10 ms (PDW) and 60 ms (T2W), 20-cm FOV, 4-mm slice thickness, 8-mm inter-slice gap, 256x256 acquisition matrix, and 32 echo-train. As in our previous studies,9 slices of the thoracic aorta and 9 slices of the abdominal aorta were obtained at 12-mm intervals, which each covering about a 10-cm portion of the thoracic aorta below the aortic arch and a 10-cm portion of the abdominal aorta above the bifurcation of the common iliac artery (Fig. 1). Plaque was defined as a clearly identified luminal protrusion with focal wall thickening, and the plaque extent in each slice was scored from 0 to 4 points based on the percentage of the luminal surface involved by plaque: 0 (no plaque), 1 (1–25%), 2 (26–50%), 3 (51–75%),

0.23), and there was some overlap in hsCRP levels between patients with and without CAD. Since the atherosclerotic process, which results in CAD, is recognized as a generalized process that may involve the entire vasculature,11,12 hsCRP levels may reflect not only the degree of coronary atherosclerosis but also the degree of atherosclerosis in other vascular beds.

Recently, magnetic resonance imaging (MRI) has become a useful tool for non-invasively evaluating atherosclerotic plaques in both the thoracic and abdominal aortas.13–16 We17,18 and others19 have shown good correlations regarding the aortic plaque extent between in vivo and ex vivo MRI findings and histopathology in animal models. In humans, we reported that MRI evaluation of thoracic aorta closely correlated with transesophageal echocardiography (TEE) findings. Using MRI, we previously investigated the associations of thoracic and abdominal aortic plaque with atherosclerotic risk factors and plasma hsCRP levels in 102 patients,14 and that hsCRP levels correlated with the extent of thoracic and abdominal aortic plaque. Therefore, the present study extended our previous study by showing the associations between plasma hsCRP levels and the severities of both coronary and aortic atherosclerosis in 136 patients undergoing coronary angiography and aortic MRI and by elucidating which severity of coronary or aortic atherosclerosis is more closely related to hsCRP levels.
and 4 (>75%) points. The severities of thoracic and abdominal aortic atherosclerosis are represented as the number of slices with plaque (plaque slice number) and the sum of scores of 9 slices (plaque extent score). Plaque extents were evaluated by two observers, and discrepancy was resolved by consensus. The intra-observer and inter-observer agreement for the assessment of plaque extents was 98% and 92% of slices, respectively.¹⁴

Statistical Analysis

Any differences between the 2 groups were evaluated by the unpaired t test for parametric variables, by the Mann-Whitney U test for nonparametric variables, and by the chi-square test for categorical variables. Any differences among the 3 or more groups were evaluated by analysis of variance with Scheffe's test for parametric variables, by the Kruskal-Wallis test for nonparametric variables, and by the chi-square test for categorical variables. Correlations between hsCRP levels and the severities of coronary or aortic atherosclerosis were evaluated by Spearman's rank correlation test. Forward stepwise multiple logistic regression analysis was used to elucidate the independent associations of hsCRP levels with atherosclerotic risk factors and coronary or aortic atherosclerosis. A p value of < 0.05 was considered significant. The results are presented as the mean ± SD or the median.

Results

Of the 136 study patients, CAD was found in 96 patients (71%), of whom 38 had 1-vessel disease, 34 had 2-vessel disease, and 24 had 3-vessel disease. Thoracic and abdominal aortic plaques were detected by MRI in 88 (65%) and 124 (91%) patients, respectively. Compared with 40 patients without CAD, 96 with CAD were predominantly male and had higher blood pressures and lower HDL-cholesterol levels (Table 1). Consequently, plasma hsCRP levels were higher in patients with CAD than in those without CAD (median 0.78 vs. 0.48 mg/L, p < 0.02). The hsCRP levels tended to increase depending on the number of > 50% stenotic coronary vessels: 0.48 mg/L in CAD(−), 0.70 mg/L in 1-vessel disease, 0.74 mg/L in 2-vessel disease, and 0.88 mg/L in 3-vessel disease, but the differ-

Fig. 1. MRI slices of the aortas and plaque scores

For each patient, 9 slices of the thoracic aorta and 9 slices of the abdominal aorta were obtained at 12-mm intervals, which each covered about 10-cm portions of the thoracic aorta below the arch and of the abdominal aorta above the bifurcation. In each slice, the plaque extent was scored from 0 to 4 points by the percentage of the luminal surface with plaque. Arrows indicate aortic plaques.
Clinical characteristics of patients with and without CAD

Table 1. Clinical characteristics of patients with and without CAD

<table>
<thead>
<tr>
<th></th>
<th>ALL (n = 136)</th>
<th>CAD (-) (n = 40)</th>
<th>CAD (+) (n = 96)</th>
<th>1-VD (n = 38)</th>
<th>2-VD (n = 34)</th>
<th>3-VD (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>64 ± 9</td>
<td>63 ± 10</td>
<td>NS</td>
<td>64 ± 9</td>
<td>61 ± 9</td>
<td>66 ± 7</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>105 (77%)</td>
<td>24 (60%)</td>
<td>&lt; 0.005</td>
<td>81 (84%)</td>
<td>32 (84%)</td>
<td>30 (88%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>85 (63%)</td>
<td>22 (55%)</td>
<td>NS</td>
<td>63 (66%)</td>
<td>26 (68%)</td>
<td>22 (65%)</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>132 ± 19</td>
<td>125 ± 16</td>
<td>&lt; 0.005</td>
<td>135 ± 19</td>
<td>134 ± 19</td>
<td>138 ± 21</td>
</tr>
<tr>
<td>Antihypertensive drugs</td>
<td>67 (49%)</td>
<td>19 (48%)</td>
<td>NS</td>
<td>48 (50%)</td>
<td>21 (55%)</td>
<td>16 (47%)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>77 (57%)</td>
<td>18 (45%)</td>
<td>NS</td>
<td>59 (61%)</td>
<td>23 (61%)</td>
<td>20 (59%)</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>203 ± 35</td>
<td>205 ± 30</td>
<td>&lt; 0.001</td>
<td>202 ± 36</td>
<td>205 ± 41</td>
<td>196 ± 33</td>
</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>124 ± 31</td>
<td>122 ± 27</td>
<td>NS</td>
<td>124 ± 32</td>
<td>130 ± 37</td>
<td>119 ± 29</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>52 ± 13</td>
<td>59 ± 15</td>
<td>&lt; 0.001</td>
<td>49 ± 12</td>
<td>50 ± 12</td>
<td>49 ± 12</td>
</tr>
<tr>
<td>Statin use</td>
<td>53 (39%)</td>
<td>12 (30%)</td>
<td>NS</td>
<td>41 (43%)</td>
<td>15 (39%)</td>
<td>15 (44%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>34 (25%)</td>
<td>7 (18%)</td>
<td>NS</td>
<td>27 (28%)</td>
<td>9 (24%)</td>
<td>7 (21%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>55 (41%)</td>
<td>12 (32%)</td>
<td>NS</td>
<td>43 (45%)</td>
<td>21 (55%)</td>
<td>16 (47%)</td>
</tr>
<tr>
<td>CAD</td>
<td>96 (71%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic aortic plaque</td>
<td>88 (65%)</td>
<td>15 (38%)</td>
<td>&lt; 0.001</td>
<td>73 (76%)</td>
<td>27 (71%)</td>
<td>25 (74%)</td>
</tr>
<tr>
<td>Abdominal aortic plaque</td>
<td>124 (91%)</td>
<td>32 (80%)</td>
<td>&lt; 0.001</td>
<td>92 (96%)</td>
<td>35 (92%)</td>
<td>33 (97%)</td>
</tr>
<tr>
<td>Plasma hsCRP (mg/L)</td>
<td>0.65</td>
<td>0.48</td>
<td>&lt; 0.02</td>
<td>0.78</td>
<td>0.70</td>
<td>0.74</td>
</tr>
</tbody>
</table>

Data are presented as the mean ± SD or the number (%) of patients. Plasma hsCRP levels are presented as the median. BP; blood pressure.

Fig. 2. Plasma hsCRP levels and the number of > 50% stenotic coronary vessels

Plasma hsCRP levels tended to increase depending on the number of > 50% stenotic vessels, but the differences among the 4 groups did not reach statistical significance. The central line represents the median, boxes span from the 25th to 75th percentiles, and error bars extend from the 10th to 90th percentiles. 1-VD, 1-vessel disease; 2-VD, 2-vessel disease; 3-VD, 3-vessel disease.

Fig. 3. Plasma hsCRP levels and the number of > 50% stenotic coronary vessels

Plasma hsCRP levels were found among the 4 groups and the hsCRP level was highest in patients with 3-vessel disease: 0.44 mg/L in CAD(-), 0.80 mg/L in 1-vessel disease, 0.60 mg/L in 2-vessel disease, and 1.20 mg/L in 3-vessel disease (p < 0.05). The hsCRP levels correlated, significantly but weakly, with the number of > 50% stenotic segments (r = 0.22) and the number of > 25% stenotic segments (r = 0.21) (Table 2); however, after exclusion of the 53 patients on statin, hsCRP levels correlated better with the numbers of > 50% stenotic segments (r = 0.32) and > 25% stenotic segments (r = 0.30) (Table 2).

With respect to aortic atherosclerosis, the plaque slice number and plaque extent score in the thoracic aorta correlated with plasma hsCRP levels (r = 0.32 and r = 0.31) (Table 2). The plaque slice number and plaque extent score in the abdominal aorta also correlated with hsCRP levels (r = 0.34 and r = 0.33); however, the total plaque slice number (total plaque slices in thoracic and abdominal aortas) and the total plaque extent score (total plaque extent scores in aortas) correlated best with hsCRP levels (r = 0.39 and r = 0.38). Moreover, after exclusion of the 53 patients on statins, the total plaque slice number and total plaque extent score correlated more closely with hsCRP levels (r = 0.48 and r = 0.44) (Table 2). According to the total plaque extent score, the 136 study patients were divided into the quartiles. As shown in Fig. 3, hsCRP
levels increased stepwise in the quartiles: 0.40 mg/L, 0.56 mg/L, 1.08 mg/L, and 1.10 mg/L ($p<0.001$). Even after exclusion of the 53 patients on statin, hsCRP levels were found to increase stepwise in the quartiles: 0.40 mg/L, 0.56 mg/L, 1.04 mg/L, and 1.17 mg/l ($p<0.001$).

To elucidate the independent association between hsCRP levels and coronary or aortic atherosclerosis, the severity of coronary atherosclerosis (number of stenotic vessels), the severity of aortic atherosclerosis (quartiles) and clinical variables (age, gender, blood pressures, diabetes, smoking, total cholesterol levels, HDL-cholesterol levels, and statin use) were entered into a multivariate logistic regression model. In multivariate analysis, aortic atherosclerosis was found to be a significant factor associated with hsCRP levels independent of atherosclerotic risk factors, but no such significance was found for coronary atherosclerosis. The odds ratio for aortic atherosclerosis (per grade of quartiles) was 1.7 (95%CI 1.2−2.4, $p<0.005$) for an hsCRP level of >1.0 mg/L.

### Table 2. Correlations between plasma hsCRP levels and the severities of coronary and aortic atherosclerosis

<table>
<thead>
<tr>
<th>Variables</th>
<th>ALL</th>
<th>Statin (−)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r^*$</td>
<td>$p$ value</td>
</tr>
<tr>
<td>Coronary atherosclerosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of &gt;50% stenotic segments (median 1.0, range 0 to 9)</td>
<td>0.22</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Number of &gt;25% stenotic segments (median 2.0, range 0 to 11)</td>
<td>0.21</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Aortic atherosclerosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic plaque slice number  (median 1.0, range 0 to 9)</td>
<td>0.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thoracic plaque extent score  (median 3.1, range 0 to 27)</td>
<td>0.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Abdominal plaque slice number (median 4.0, range 0 to 9)</td>
<td>0.34</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Abdominal plaque extent score (median 7.4, range 0 to 28)</td>
<td>0.33</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total plaque slice number     (median 5.0, range 0 to 18)</td>
<td>0.39</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total plaque extent score     (median 10.5, range 0 to 54)</td>
<td>0.38</td>
<td>&lt;0.001</td>
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</table>

$^*$ By Spearman’s rank correlation test.

**Discussion**

We investigated the associations between hsCRP levels and the severities of both coronary and aortic atherosclerosis in 136 patients (96 patients with CAD and 40 patients without CAD) who underwent coronary angiography and aortic MRI. Plasma hsCRP levels tended to increase depending on the number of stenotic coronary vessels and correlated weakly with the number of stenotic segments. Notably, hsCRP levels increased stepwise on the quartiles by the total aortic plaque extent score and correlated best with the total aortic plaque extent score. In the multiple regres-
sion analysis, the severity of aortic atherosclerosis was found to be a significant factor associated with hsCRP levels independent of atherosclerotic risk factors. In contrast, there was no significant association between coronary atherosclerosis and hsCRP levels.

With respect to the association between hsCRP and the severity of coronary atherosclerosis, some studies showed the significant correlation between hsCRP levels and the severity of coronary stenosis \(^4\)\(^5\)\(^6\), whereas other studies did not \(^7\)\(^8\)\(^9\). Azar et al. \(^8\) reported no correlation between hsCRP levels and the extent score of coronary stenosis using a simple linear correlation test in 98 patients. Hoffmeister et al. \(^9\) also reported no correlation with the coronary extent score in 312 patients with CAD. In contrast, Zebrack et al. \(^5\) showed a correlation between hsCRP levels and the coronary extent score in 2554 patients with CAD; however, the correlation coefficients were very low (0.06–0.08). Erren et al. \(^6\) reported that hsCRP levels correlated with the coronary extent score using Spearman’s rank correlation test \((r = 0.29)\). We also reported a correlation between hsCRP levels and the severity of coronary stenosis by Spearman’s rank correlation test in 273 patients with CAD \(^10\). Even in our previous study, the correlations were weak \((r = 0.20–0.23)\), and there was some overlap in hsCRP levels between patients with and without CAD; therefore, we hypothesized that hsCRP levels would be more likely to reflect the degree of atherosclerosis in other vascular beds than in the coronary arteries.

With respect to the association between hsCRP and the severity of aortic atherosclerosis, Takasu et al. \(^20\) reported that hsCRP levels did not correlate with the severity of calcification in the thoracic aorta using computed tomography (CT); however, Agmon et al. \(^21\) demonstrated that hsCRP levels were associated with the presence and severity of thoracic aortic plaques using TEE in 386 subjects. Regarding abdominal aortic atherosclerosis, the Rotterdam study \(^22\) showed the association between hsCRP levels and the severity of abdominal aortic calcification by radiography in 6582 subjects. In this study, no correlation was found between hsCRP levels and the severity of coronary calcification. Hommels et al. \(^23\) also showed the association between hsCRP and the severity of abdominal aortic atherosclerotic lesions detected by angiography in 95 hypertensive patients. Moreover, the PDAY study \(^24\) reported the association between hsCRP and the severity of abdominal aortic atherosclerosis by autopsy in 1255 subjects who had died of external causes; therefore, these findings suggest that hsCRP levels must reflect the severity of atherosclerosis in the thoracic and abdominal aortas.

TEE provides high-resolution images of the thoracic aorta, but TEE can assess only a small portion of the abdominal aorta; however, MRI has recently become a useful tool to non-invasively evaluate atherosclerotic plaque in both thoracic and abdominal aortas in the same examination session \(^13\)\(^–\)\(^16\). Using MRI, we \(^14\) and others \(^16\) previously investigated the associations of thoracic and abdominal aortic plaques with atherosclerotic risk factors. We showed that plaques in the thoracic and abdominal aortas were characteristic associated with hyperlipidemia and smoking, respectively \(^14\). Tribouilloy et al. \(^20\) reported an association between LDL-cholesterol levels and thoracic plaques by TEE, whereas Giral et al. \(^26\) showed no association between LDL-cholesterol and abdominal plaques by ultrasound. An autopsy study reported that patients with hyperlipidemia had severe plaque in the thoracic aorta \(^11\). In contrast, autopsy studies reported smoking to be more closely associated with plaques in the abdominal aorta than in the thoracic aorta \(^27\). Giral et al. \(^26\) showed smoking to be associated with abdominal plaques by ultrasound. The thoracic and abdominal aortas may thus have different susceptibilities to atherosclerotic risk factors. The abdominal aorta tapers geometrically and has higher blood pressures \(^28\), and is stiffer with less elastin and more collagen \(^28\). Vasa vasorum is common in the thoracic aorta but rare in the abdominal aorta \(^26\). These features may explain the different susceptibilities to risk factors between the aortas. Because patients have various risk factors and because thoracic and abdominal aortas may have different susceptibilities to risk factors, it appears to be preferable to evaluate the total degree of aortic atherosclerosis rather than the degree of atherosclerosis in either the thoracic or abdominal aorta. Using MRI, we previously reported the associations of thoracic and abdominal aortic plaques with the extent of coronary artery stenosis \(^15\). The extents of plaque in both thoracic and abdominal aortas correlated with the extent of coronary stenosis; however, the total plaque extent in the aortas was found to be most closely associated with the extent of coronary stenosis, and this factor was considered to be the best predictor for CAD.

In the present study, we investigated the associations between plasma hsCRP levels and the severities of coronary and aortic atherosclerosis in 136 patients. Although both thoracic and abdominal aortic plaque extent scores correlated with hsCRP levels, the total aortic plaque extent score correlated best with hsCRP levels. These observations suggest that such levels appear to reflect the total degree of aortic atherosclerosis rather than the degree of atherosclerosis in either tho-
ractic or abdominal aortas. Plasma hsCRP levels tended to increase depending on the number of stenotic coronary artery vessels and correlated weakly with the number of stenotic coronary segments, whereas hsCRP levels increased stepwise in quartiles by the total aortic plaque extent score. In multivariate analysis, the degree of aortic atherosclerosis was a significant factor associated with hsCRP levels independent of atherosclerotic risk factors, whereas coronary atherosclerosis was not; therefore, hsCRP levels are more likely to reflect the degree of aortic atherosclerosis rather than the degree of coronary atherosclerosis. The use of MRI would be useful for evaluating the degree of atherosclerosis in both the thoracic and abdominal aortas in the same examination session, especially in patients with high hsCRP levels.

Our study has several limitations. First, the number of study patients (136) was relatively small. Moreover, our study was conducted in Japanese patients undergoing angiography, who are generally considered to be a highly selected population at high-risk for CAD. Our results may not be applicable to general or other ethnic populations. Second, MRI was used to evaluate aortic atherosclerosis, but angiography was used to evaluate coronary atherosclerosis. Angiography cannot visualize plaque, and it only shows lumen characteristics. In our study, intravascular ultrasound (IVUS) was not used to evaluate coronary atherosclerosis; however, IVUS cannot evaluate the degree of atherosclerosis throughout the coronary arteries. Third, in the thoracic aorta, we did not evaluate the arch or ascending aorta to reduce the examination time. Because plaques were reported to be more prevalent in the thoracic descending aorta (45%) than in the arch (31%) or the ascending aorta (8%)

In conclusion, plasma hsCRP levels correlated with the severities of both coronary and aortic atherosclerosis; however, hsCRP levels correlated better with the severity of aortic atherosclerosis than with the severity of coronary atherosclerosis, and aortic atherosclerosis was an independent factor associated with hsCRP levels, thus suggesting that hsCRP levels are more likely to reflect the severity of aortic atherosclerosis than the severity of coronary atherosclerosis.

References


