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Magnetic Resonance Evaluation of the Associations of Thoracic and Abdominal Aortic Plaques with the Presence and Extent of Coronary Artery Stenosis

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ABSTRACT

The association between coronary artery disease (CAD) and thoracic aortic plaques has often been reported using transesophageal echocardiography. However, studies showing the association between CAD and abdominal aortic plaques are scarce. CMR can visualize plaques in both the thoracic and abdominal aortas. Using CMR, we investigated the associations of thoracic and abdominal aortic plaques with the presence and extent of coronary artery stenosis in 146 patients undergoing coronary angiography, of whom 108 had CAD. The prevalence of thoracic and abdominal aortic plaques was higher in patients with CAD than in those without CAD (73% and 94% vs. 32% and 79%, $p < 0.025$). Stepwise increases in the prevalence and extents of both thoracic and abdominal plaques were found depending on the number of stenotic coronary vessels. Plaque extent in the thoracic aorta correlated with the numbers of >50% and >25% stenotic coronary segments (rs = 0.30 and 0.41). Plaque extent in the abdominal aorta also correlated with the numbers of >50% and >25% stenotic segments (rs = 0.40 and 0.44). Notably, the total plaque extent in the aortas correlated best with the numbers of >50% and >25% stenotic coronary segments (rs = 0.41 and 0.49, $p < 0.001$), and this factor was found to be the best predictor for the presence of CAD by the receiver-operating-characteristics curve analysis. Thus, the total plaque extent in the aortas was found to be more closely associated with the presence and extent of coronary stenosis than the thoracic or abdominal aortic plaque extent.

INTRODUCTION

The atherosclerotic process that results in coronary artery disease (CAD) is recognized to be a generalized process that may involve the entire vasculature (1, 2). The association between CAD and thoracic aortic plaques has often been reported using transesophageal echocardiography (TEE) (3–7). However, studies showing an association between CAD and abdominal aortic plaques are scarce. An autopsy study reported plaques in the abdominal aorta, but not in the thoracic aorta, to be severe in patients with cardiac catastrophe (2). Using computed tomography, Takasu el al. (8) reported both thoracic and abdominal aortic plaques to be associated with the presence of CAD.

Recently, cardiovascular magnetic resonance (CMR) has become a useful tool for non-invasively evaluating atherosclerotic plaques in both the thoracic and abdominal aortas (9–11). We (12–14) and others (15) showed good correlations regarding the aortic plaque extent between in vivo and ex vivo CMR findings and histopathology in animal models. In humans, we reported that MRI evaluations of the thoracic aorta closely correlated with TEE findings (9). Using CMR, we previously reported the associations of thoracic and abdominal aortic plaques with risk factors and CAD in 102 patients undergoing coronary angiography (10). We showed the prevalence of thoracic and abdominal aortic plaques to be high in patients with CAD and to increase as the number of stenotic coronary vessels increased. Moreover, we reported complex plaques in the abdominal aorta to be associated

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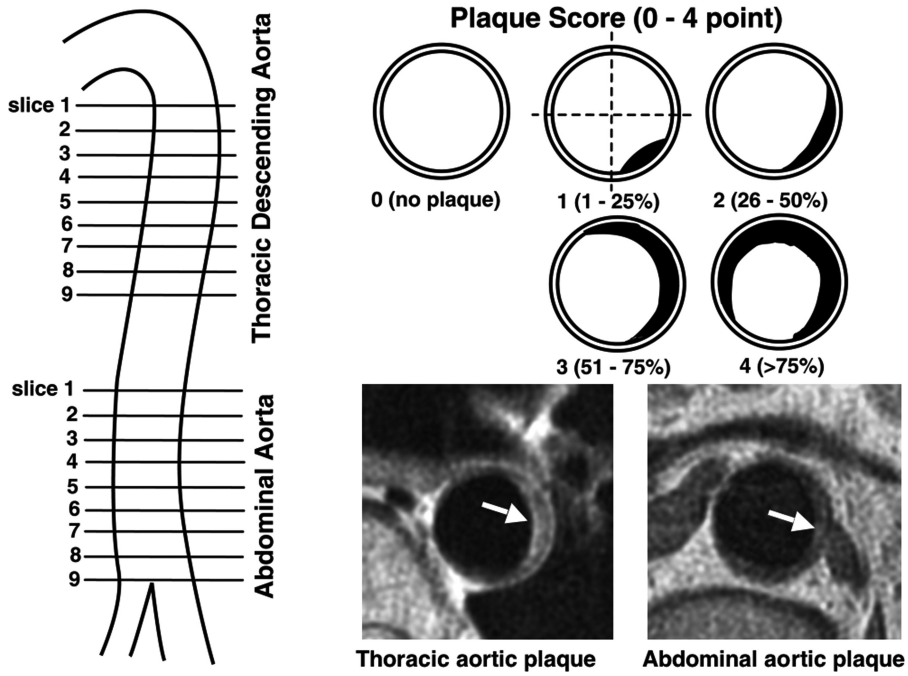

Figure 1. MRI slices of the aortas and plaque scores. In each patient, 9 slices of thoracic aorta and 9 slices of abdominal aorta were obtained at 12 mm intervals, which each covered about 10 cm portions of thoracic aorta below the arch and of abdominal aorta above the bifurcation. In each slice, the plaque extent was scored from 0 to 4 points by the percentage of the luminal surface involved by plaque. Arrows indicate aortic plaques.

with myocardial infarction (MI) and complex coronary lesions (16). However, the association between aortic plaques and the extent of coronary artery stenosis has not been fully elucidated yet. The present study was done to elucidate the associations of thoracic and abdominal aortic plaques with the extent of angiographic coronary stenosis (the number of >50% stenotic vessels, the number of >50% stenotic segments, and the number of >25% stenotic segments) and to elucidate the predictive values of thoracic and abdominal aortic plaques for CAD in 146 patients undergoing coronary angiography.

MATERIALS AND METHODS

Patient population

The study patients consisted of 146 patients (male, 76%; mean age, 64 ± 9 yrs; range 40 to 80 yrs) undergoing coronary angiography for suspected or known CAD at National Defense Medical College Hospital, who were the same patients in our recent study (16). Patients with a history of cardiac surgery, aortic disease, or valvular or congenital heart disease were excluded. Of the 146 patients, 108 (74%) had CAD (>50% stenosis) on coronary angiograms, of whom 26 had a history of percutaneous coronary intervention (PCI) and 44 had MI. The diagnosis of acute and old MI was given to 25 and 19 patients, respectively. Our study was approved by institutional ethics committee. After written informed consent was obtained, CMR of the aortas was performed at Iruma Heart Hospital within 2 weeks of angiography. However, of the 25 patients with acute MI, 13 had CMR in 1 to 3 months after the onset of MI, and 12 did it in 4 to 8 months because of stent implantation. Of the 146 patients, 85 (58%) had hypertension (blood pressures ≥140/90 mm Hg or on drugs), of

Table 1. Clinical characteristics in patients with and without CAD

	CAD(-) (n = 38)	(+)*vs*(−)	CAD(+) (n = 108)
Age (yrs)	63 ± 9	*NS*	64 ± 9
Gender (male)	24 (63%)	*NS*	87 (81%)
Hypertension	21 (55%)	*NS*	64 (59%)
Systolic blood pressure (mmHg)	126 ± 16	*<0.05*	133 ± 20
Hyperlipidemia	15 (39%)	*NS*	64 (59%)
Total cholesterol (mg/dl)	206 ± 29	*NS*	205 ± 35
HDL-cholesterol (mg/dl)	57 ± 13	*<0.005*	50 ± 13
Diabetes mellitus	7 (18%)	*NS*	29 (27%)
Smoking	14 (37%)	*NS*	53 (49%)
Thoracic aorta			
Plaque (+)	12 (32%)	*<0.001*	79 (73%)
Plaque slice number	0.0	*<0.005*	1.5
Abdominal aorta			
Plaque (+)	30 (79%)	*<0.025*	102 (94%)
Plaque slice number	2.0	*<0.001*	4.0

Data are presented as the mean value ±SD or the number (%) of patients, except for plaque slice numbers that are presented as the median value.

Table 2. Factors associated with the presence of CAD (Multiple logistic regression analysis of the 146 study patients)

Variables	Odds ratio	(95% CI)	P value
Hyperlipidemia	2.7	(1.1–6.7)	*<0.05*
HDL-cholesterol	0.9	(0.8–0.9)	*<0.05*
Thoracic aortic plaques	5.2	(2.0–13.5)	*<0.002*

The dependent variables were the presence of CAD.
The factors analyzed included age, gender, hypertension, hyperlipidemia, HDL-cholesterol, diabetes, smoking, thoracic aortic plaque, and abdominal aortic plaque.

whom 68 were on antihypertensive drugs, and 79 (54%) had hyperlipidemia (total cholesterol level >240 mg/dL or on drugs), of whom 57 were taking a statin. Diabetes mellitus (fasting glucose level ≥126 mg/dL or on treatment) was present in 36 (25%) patients, and 67 (46%) were smokers (≥10 packs-year). Fasting blood samples were taken on the day of angiography. Serum lipid levels were measured by standard laboratory methods.

Coronary angiography

Coronary angiograms were recorded using the Judkins technique and a cineangiogram system (Toshiba, Tokyo, Japan). Coronary arteries were divided into 27 segments defined by Coronary Artery Surgery Study (CASS) classification (17). The degree of stenosis in each segment was evaluated according to 5 grades (≤25%, 26–50%, 51–75%, 76–90%, >90% stenosis). 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 CMR data. The intra-observer agreement for the assessment of the grade of stenosis was evaluated in 20 patients (540 segments), and it was found to be 98% of segments. CAD was defined as at least one coronary artery having >50% luminar diameter stenosis on angiograms. The extent of coronary artery stenosis was represented as the number of >50% stenotic vessels, the number of >50% stenotic segments, and the number of >25% stenotic segments.

CMR of the aorta

Aortic CMR was performed on Signa 1.5T Cvi scanner with a phased-array body coil (GE Medical Systems, Mount Prospect, IL). 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, 256 × 256 acquisition matrix, and 32 echo-train. As in our previous studies (10, 16, 18), 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 portion of the thoracic aorta below the arch and 10 cm portion of the abdominal aorta above the bifurcation of the common iliac artery (Fig. 1). For each patient, we assessed the presence and extents of plaque in 9 slices of the thoracic aorta and 9 slices of the abdominal aorta. As in our previous study (10), 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%), and 4 (>75%) point. The plaque extents in the thoracic and abdominal aortas were represented as the number of slices with plaque (plaque slice number) and the sum of scores of 9 slices (plaque extent score). The plaque extents were evaluated by two observers, and

Table 3. Associations between the number of >50% stenotic coronary vessels and the extents of plaques in thoracic and abdominal aortas

	CAD(-) (n = 38)	1-VD (n = 47)	2-VD (n = 39)	3-VD (n = 22)	P value
Age (yrs)	63± 9	62± 9	66± 7	66± 8	*NS*
Gender (male)	24 (63%)	37 (79%)	33 (85%)	17 (77%)	*NS*
Thoracic aorta					
Plaque (+)	12 (32%)	32 (68%)	28 (72%)	19 (86%)	<0.001
Plaque slice number	0.0	1.0	1.0	2.0	<0.005
Plaque extent score	0.0	2.0	2.0	2.5	<0.005
Abdominal aorta					
Plaque (+)	30 (79%)	43 (91%)	37 (95%)	22 (100%)	<0.05
Plaque slice number	2.0	4.0	4.0	4.5	<0.001
Plaque extent score	3.5	8.0	8.0	7.5	<0.001
Thoracic and abdominal aortas					
Plaque (+)	30 (79%)	44 (94%)	37 (95%)	22 (100%)	<0.025
Total plaque slice number	3.0	6.0	6.0	7.0	<0.001
Total plaque extent score	4.0	9.0	10.0	12.0	<0.001

Data are presented as the mean value ±SD or the number (%) of patients, except for plaque slice number and plaque extent score that are presented as the median value.
Total plaque slice number was defined as a total of plaque slice numbers in the thoracic and abdominal aortas, and total plaque extent score was defined as a total of plaque extent scores in the aortas. *1-VD,* 1-vessel disease; *2-VD,* 2-vessel disease; *3-VD,* 3-vessel disease.

Table 4. Correlations between the numbers of stenotic coronary segments and the extents of plaques in thoracic and abdominal aortas

	Number of >50% stenotic coronary segment		Number of >25% stenotic coronary segment	
	rs*	*p value*	rs*	*p* value
Thoracic aorta				
Plaque slice number	0.31	<0.002	0.41	<0.002
Plaque extent score	0.30	<0.001	0.41	<0.001
Abdominal aorta				
Plaque slice number	0.39	<0.001	0.43	<0.001
Plaque extent score	0.40	<0.001	0.44	<0.001
Thoracic and abdominal aortas				
Total plaque slice number	0.40	<0.001	0.49	<0.001
Total plaque extent score	0.41	<0.001	0.49	<0.001

*By Spearman's rank correlation test.

any 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 (10).

Statistics

Differences between 2 groups were evaluated by the unpaired t-test for parametric variables, by Mann-Whitney's U-test for nonparametric variables and by the chi-square test for categorical variables. Differences among 3 or more groups were evaluated by ANOVA with Scheffe's test for parametric variables, by Kruskal-Wallis rank test for nonparametric variables, and by the chi-square test for categorical variables. Correlations between the extents of aortic plaques and that of coronary stenosis were evaluated by Spearman's rank correlation test. A multiple logistic regression analysis was used to elucidate the associations between aortic plaques and CAD. The diagnostic abilities of aortic plaques for CAD were evaluated by the receiver-operating-characteristics (ROC) curve analysis, and the areas under ROC curves (AUC) were compared. A p value of <0.05 was considered statistically significant. The results are presented as the mean value $\pm$ SD or the median value.

RESULTS

Of the 146 patients, 108 had CAD (>50% stenosis). Compared with 38 patients without CAD, 108 with CAD had higher blood pressures and lower HDL-cholesterol levels (Table 1). Plaques were more prevalent in the abdominal aorta than in the thoracic aorta ($p < 0.001$). Patients with CAD more often had plaques in the thoracic (73% vs. 32%) and abdominal (94% vs. 79%) aortas than those without CAD ($p < 0.025$). The plaque slice numbers in the thoracic and abdominal aortas were also greater in patients with CAD than in those without CAD (median 1.5 and 4.0 vs. 0.0 and 2.0, $p < 0.005$) (Table 1).

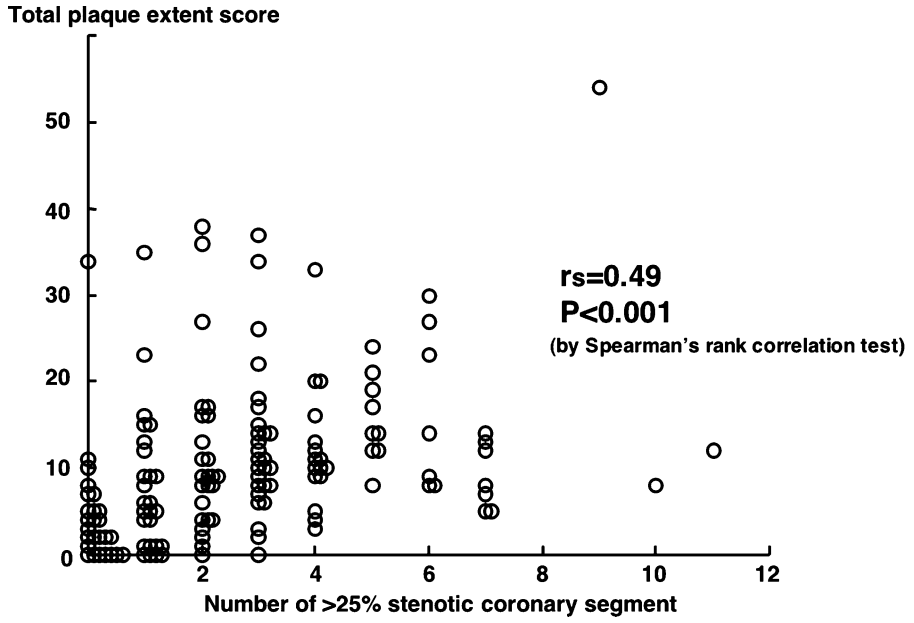

Figure 2. Correlation between the total aortic plaque extent and the number of >25% stenotic coronary segment.

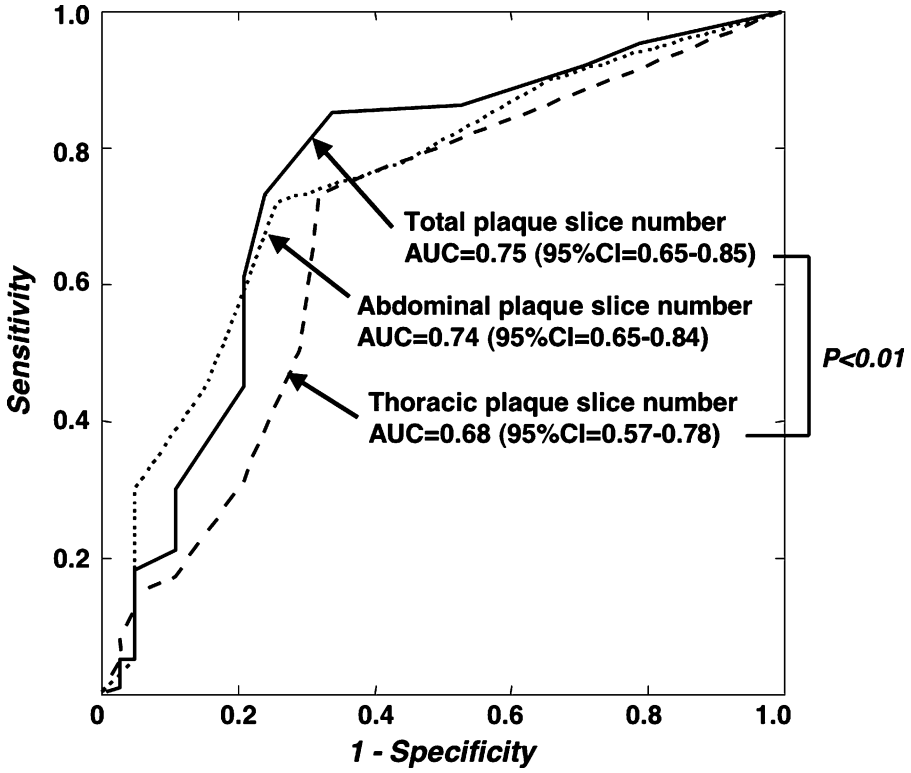

Figure 3. ROC curves of thoracic plaque slice number, abdominal plaque slice number, and total plaque slice number for the prediction of CAD. The AUC for total plaque slice number was found to be largest ($p < 0.01$).

To identify any independent association between aortic plaques and CAD, clinical variables (age, gender, hypertension, hyperlipidemia, HDL-cholesterol, diabetes, and smoking) and aortic plaques were entered into a multivariate logistic regression model. In multivariate analysis, thoracic aortic plaques were an independent factor associated with the presence of CAD (odds ratio = 5.2; 95%CI = 2.0–13.5; $p < 0.001$), while abdominal aortic plaques were not (Table 2).

Of the 108 patients with CAD, 47 had 1-vessel, 39 had 2-vessel, and 22 had 3-vessel disease. As shown in Table 3, stepwise increases in the prevalence and extents of thoracic and abdominal aortic plaques were found depending on the number of >50% stenotic coronary vessels. The plaque slice number and plaque extent score in the thoracic aorta correlated with the numbers of >50% stenotic coronary segments (rs = 0.31 and rs = 0.30) and >25% stenotic segments (rs = 0.41 and rs = 0.41) (Table 4). The plaque slice number and plaque extent score in the abdominal aorta also correlated with the numbers of >50% stenotic segments (rs = 0.39 and rs = 0.40) and >25% stenotic segments (rs = 0.43 and rs = 0.44). Notably, the total plaque slice number (a total of plaque slice numbers in the thoracic and abdominal aortas) and the total plaque extent score (a total of plaque extent scores in the aortas) were found to correlate best with the numbers of >50% stenotic segments (rs = 0.40 and rs = 0.41) and >25% stenotic segments (rs = 0.49 and rs = 0.49) (Fig. 2). The ability of aortic plaques to predict CAD was assessed by the ROC curve analysis (Fig. 3). The AUC for the total plaque slice number was thus found to be largest ($p < 0.01$). Sensitivity, specificity, and positive and negative predictive values of aortic plaques for CAD are shown in Table 5.

DISCUSSION

The association between CAD and thoracic aortic plaques has often been reported using TEE (3–7). Thoracic plaques have been shown to be more strongly associated with CAD than carotid or femoral artery plaques detected by ultrasonography (7) and to be associated with cardiovascular events (6, 19). Although an autopsy study reported plaques in the abdominal aorta, but not in the thoracic aorta, to be severe in patients with cardiac catastrophe (2), studies showing the association between CAD and abdominal aortic plaques are scarce, and little attention has so far been paid to abdominal plaques. Using CMR, we investigated plaques in the thoracic and abdominal aortas in 146 patients undergoing coronary angiography. The prevalence and extents of plaques in both the thoracic and abdominal aortas were greater in patients with CAD than in those without CAD, and the extents of both thoracic and abdominal plaques correlated with the extent of coronary stenosis. Notably, the total plaque extent in the aortas appeared to correlate better with the

Table 5. Sensitivity, specificity, and positive and negative predictive values of aortic plaques for predicting the presence of CAD

	N = 146 Patients	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Thoracic plaque slice number					
>1 slices	(n = 91)	73%	68%	87%	47%
>2	(n = 65)	50%	71%	83%	33%
>3	(n = 42)	31%	79%	81%	29%
Abdominal plaque slice number					
>1 slices	(n = 132)	94%	21%	77%	57%
>2	(n = 122)	90%	34%	80%	54%
>3	(n = 101)	78%	55%	83%	47%
>4	(n = 88)	72%	74%	89%	48%
Total plaque slice number					
>1 slices	(n = 133)	95%	21%	77%	62%
>2	(n = 126)	92%	29%	79%	55%
>3	(n = 113)	86%	47%	82%	55%
>4	(n = 105)	85%	66%	88%	61%
>5	(n = 88)	73%	76%	90%	50%

extent of coronary stenosis than the plaque extents in either the thoracic or abdominal aortas. The total aortic plaque extent was the best predictor for the presence of CAD.

We previously reported that plaques in the thoracic and abdominal aortas were characteristically associated with hyperlipidemia and smoking, respectively (10). Tribouilloy et al. (20) reported an association between LDL-cholesterol levels and thoracic plaques by TEE, whereas Giral et al. (21) showed no association between LDL-cholesterol and abdominal plaques by ultrasound. An autopsy study reported patients with hyperlipidemia to have severe plaques in the thoracic aorta (1). In contrast, autopsy studies reported smoking to be more closely associated with plaques in the abdominal aorta than in the thoracic aorta (22, 23). Giral et al. (21) 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 pressure waves reflecting off of the iliac and other arteries, resulting in a higher pulsatile stress in the abdominal aorta than in the thoracic aorta (24). The abdominal aorta is also stiffer with less elastin and more collagen (24). Vasa vasorum is common in thoracic aorta but rare in abdominal aorta, suggesting that the oxygen and nourishment of the abdominal aorta comes mainly by diffusion from the aortic lumen (25). These may be the reasons for different susceptibilities to risk factors between the aortas. In our study, thoracic aortic plaques were an independent factor for CAD, while abdominal aortic plaques were not. Takasu et al. (8) also reported thoracic plaques to be more closely associated with CAD than abdominal plaques by computed tomography. The presence of thoracic plaques may thus be a better marker of CAD than abdominal plaques. However, our study showed total aortic plaque extent to be most closely associated with the extent of coronary artery stenosis and to be the best predictor for CAD. Because patients have various risk factors and because the thoracic and abdominal aortas may have different susceptibilities to risk factors, it appears to be preferable to evaluate atherosclerosis in both the aortas than in either the thoracic or abdominal aortas. MRI is a useful tool for evaluating atherosclerosis in multiple vascular beds in the same exam session, thereby determining the degree of systemic atherosclerotic involvement and predicting the degree of coronary atherosclerosis more accurately.

Our study has several limitations. First, CMR was used to evaluate aortic atherosclerosis, but angiography was used to evaluate coronary atherosclerosis. Angiography cannot visualize plaques, and it only shows lumen characteristics. Although intravascular ultrasound (IVUS) can visualize coronary plaques, IVUS was not used in our study. Second, 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%) (6) and because plaques in the descending aorta were reported to be a stronger factor for CAD than those in the arch or ascending aorta (4), we only assessed the descending aorta. Third, since aortic images were obtained at 12 mm interval, we evaluated these images not blinded to the adjacent slices. This may have caused some bias and have confounded the results. Finally, our study was 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 the general or other ethnic populations.

In summary, the prevalence of plaques in both thoracic and abdominal aortas was high in patients with CAD. Although only thoracic aortic plaques were an independent factor for CAD, the extents of plaques in both the thoracic and abdominal aortas correlated with the extent of coronary artery stenosis. As a result, 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.

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