Correlation of thoracic aortic distensibility with aortic and coronary atherosclerotic plaques

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Purpose

To determine the relationship between aortic distensibility and the presence, extent and composition of thoracic aortic plaques (TAP)
Methods and materials

2.1. Study population

With institutional review board approval the study was conducted on 150 patients who were referred to three cardiac imaging centers from January 2012 up to August 2013 for MSCT coronary angiography.

Exclusion criteria were aortic aneurysm, arrhythmia, hypertrophic cardiomyopathy, Marfan's disease and Ehlers-Danlos syndrome. Patients with history of coronary artery bypass surgery or coronary stenting and those who underwent aortic valve or thoracic aorta surgery were also excluded from the study since the deployed stents or surgical clips prohibited their coronary calcium scoring, hence no routine coronary calcium scan was carried out in those patients.

The patients' demographics, medical history and laboratory data were collected prior to CT acquisition. Use of hypoglycemic agents or a fasting blood sugar ≥ 126 mg/dL was considered as diabetes mellitus. Systolic/diastolic blood pressure ≥ 140/90 mmHg (130/80 mmHg in diabetic patients) or use of antihypertensive medications was considered as hypertension. Total cholesterol > 200 mg/dL, Low-density lipoprotein cholesterol > 150 mg/dL, or use of cholesterol-lowering medication was determined as hypercholesterolemia. Body mass index (BMI) was calculated as the weight in kilograms divided by the height in square meters.

2.2. MSCT imaging protocol for data acquisition

2.2.1. MSCT protocol

Cardiac MSCT scans were performed using three multislice CT scanners including two 64-slice single-source CT scanners (Brilliance 64, Philips Medical Systems and SOMATOM Sensation 64, Siemens Medical Solutions) and a 256-slice dual-source CT scanner (SOMATOM Definition Flash, Siemens Healthcare). The data acquisition and image reconstruction techniques for both coronary CTA and calcium scoring were explained elsewhere in detail [23]; nevertheless, they are also briefly mentioned here. Parameters of cardiac CT scan are summarized in Table1.

In all three scanners the scan field was set from tracheal carina to the diaphragm and images were acquired in the craniocaudal direction during a single breath hold while the electrocardiogram (ECG) was recorded simultaneously to allow retrospective gating of the data. Using a dual-head injector, a volume of 60-85 mL of a non-ionic contrast medium [either Iopromide (Ultravist) 370 mg I/mL; Bayer Schering Pharma, Berlin; Germany or Iodixanol (Visipaque) 320 mg I/mL; GE Healthcare Inc., Cork; Ireland], followed by 50-60 mL of saline solution chaser, was injected in an antecubital vein via an 18-gauge catheter.
at a mean flow rate of 5 mL/s; both volume and flow rate were adjusted according to the patients' body habitus. Images were reconstructed with a slice thickness of 0.67 mm and an interval of 0.4 mm. Test bolus injection was used to find the optimal time of aortic root opacification and determine the start time of the scan.

Data were reconstructed at 35% and 75% of R-R intervals of ECG, representing end systole and end diastole, respectively. Thereafter, acquired images were transferred to a single off-line workstation (Extended Brilliance Workspace, Philips Medical Systems, Cleveland, OH) for post-processing, measurements and subsequent analysis.

2.2.2. Calcium scoring technique

Before injecting contrast medium, non-contrasted cardiac CT using the three aforementioned CT machines was performed in a longitudinal scan field from aortic arch down to the diaphragm, while simultaneously ECG was recorded. The obtained data were reconstructed at 50-55% of R-R interval. The corresponding images were reconstructed with a slice width of 2.5-3 mm and slice interval of 1.25-1.5 mm and the tube voltage was 120 kVp for all CT scanners.

2.3. Measurements

2.3.1. Thoracic aorta and coronary plaque

All aortic and coronary plaques were characterized and the related measurements were performed. Thoracic aorta or coronary plaque was defined as any visible structure separate from the contrast-enhanced arterial lumen which could be assigned to the arterial wall in at least 2 independent planes for coronary artery and in 2 phases in transaxial images of aorta. Calcified plaque was defined as any plaque with a density of >130 Hounsfield units (HU) and non-calcified plaque was defined as any plaque with a density of #130 HU.

Thoracic aorta and coronary calcium score (Agatston score) and plaque burden (area) were measured for all patients using dedicated calcium scoring software on aforementioned offline workstation (Heart Beat CS Software Module).

2.3.2. Aortic distensibility index

ADI was calculated as: lumen/(lumen in ED × systemic pulse pressure)× 10^3, where the lumen is end-systolic minus end-diastolic (ED) cross-sectional-area, and pulse pressure is systolic blood pressure minus diastolic blood pressure [14]. Blood pressure measurements were performed immediately before MSCT scan acquisition while the patient was in the supine position.
ADI was calculated for the ascending (A-ADI) and proximal descending aorta (PD-ADI) at the level of pulmonary trunk bifurcation, and distal descending aorta (DD-ADI) at the level middle cardiac vein entering coronary sinus. Total ADI (T-ADI) was the mean of these three ADIs. In addition, Local ADI (L-ADI) was calculated for any level of TAP (except for aortic arch) (Figure1).

The measurements of 20 randomly selected patients were performed by two expert radiologists on the mentioned workstation. The inter-observer agreement was almost perfect (Kappa=0.81-0.89 for all aortic levels). The intraclass correlation coefficient (ICC) for intra-observer agreements was also nearly complete (ICC=0.91 - 0.97, p<0.0001 for all levels).

2.4. Statistical analyses

Statistical analysis was performed using the Statistical Package for Social Sciences, version 17.0 (SPSS, Chicago, Illinois). Continuous and categorical variables are presented as the mean ± standard deviation and percentage, respectively. The comparison between groups was carried out using the student's t test or analysis of variance (ANOVA), and chi-square test for continuous and categorical variables, respectively. We used Pearson's or Spearman's correlation tests to examine the association between study measures. Logistic regression was used to determine the association between ADI and covariates. Adjustment for age, gender, diabetes mellitus, hypercholesterolemia, hypertension and cigarette smoking was also performed in the regression models. The level of significance was set at P<0.05 (two-tailed).
Results

3.1. Baseline characteristics

The overall characteristics of the patients are summarized in Table 2. TAP was detected in 75 participants (50%). The plaque was calcified in 64 patients and non-calcified in 11 patients. All patients with TAP had plaque(s) in descending thoracic aorta, and 9 cases had both ascending and descending thoracic aortic plaques. Patients with TAP were more likely to have older age, diabetes mellitus, hypercholesterolemia, coronary plaque, and higher coronary calcium score. A-ADI, PD-ADI, DD-ADI, and T-ADI for the entire cohort were 1.45 ± 1.34, 1.52 ± 1.50, 1.42 ± 1.27, and 1.47 ± 1.12 mm Hg \(^{\#1}\) 10\(^3\), respectively. All ADIs of patients with TAP were significantly lower than patients without TAP (p<0.0001).

3.2. Thoracic aorta and coronary plaques

There was a significant association between TAP and coronary plaques; frequency of coronary atherosclerotic plaques in patients with TAP was significantly higher than that of patients without TAP (53.3% vs. 6.7%; p<0.007), giving the odds ratio of 2 (95% confidence interval: 1.55-2.56). We found also a significant positive correlation between calcium scores of TAPs and coronary plaques (r=0.35, p=0.01). The coronary calcium score of patients with TAP was significantly higher than patients without TAP (Table 2).

3.3. Correlations of ADI

T-ADI was positively correlated with A-ADI (r=0.82, p<0.0001), PD-ADI (r=0.64, p<0.0001), DD-ADI (r=0.83, p<0.0001), and negatively correlated with age (r=-0.61, p<0.0001), systolic blood pressure (r=-0.35, p=0.001), pulse pressure (r=-0.55, p<0.0001), and aortic calcium score & plaque burden (r=-0.57, p<0.0001; r=-0.6, p=0.001, respectively).

L-ADI was positively correlated with A-ADI (r=0.3, p=0.02), PD-ADI (r=0.64, p<0.0001), DD-ADI (r=0.43, p=0.001), and T-ADI (r=0.35, p=0.005), and negatively correlated with age (r=-0.34, p=0.008), and aortic calcium score & plaque burden (r=-0.77, p=0.001; r=-0.79, p=0.002, respectively).

In multivariate analysis adjusted for cardiovascular risk factors, including age, gender, diabetes mellitus, hypercholesterolemia, hypertension and cigarette smoking, presence of TAP was independently correlated with ADI (adjusted odds ratio [95% confidence interval]= -1.4 [-1.3 - -1.7] for any plaque and -2.3[2.1-2.9] for calcified plaque).

3.4. ADI stratified by aortic plaque characteristics
The comparison of ADI between patients with calcified-TAP, non-calcified-TAP and without TAP are shown in Figure2. A-ADI, PD-ADI, DD-ADI, and T-ADI in patients with calcified-TAP were significantly lower than patients without TAP or with non-calcified-TAP (p<0.0001, for all).

L-ADI in patients with calcified-TAP was significantly lower than patients with non-calcified-TAP (p<0.0001).

There were no statistically significant differences between ADIs of patients without TAP and those with non-calcified-TAP.

L-ADI in patients with aortic calcium score <100 was significantly lower than patients with aortic calcium score ≥100 (0.58 ± 0.2 vs. 0.32 ± 0.14, p=0.02).

L-ADI in patients with aortic calcium area <100 mm$^2$ was significantly lower than patients with aortic calcium area ≥100 mm$^2$ (0.58 ± 0.2 vs. 0.32 ± 0.14, p=0.02).

3.5. ADI stratified by coronary plaque

A-ADI, PD-ADI, DD-ADI, and T-ADI in patients with coronary plaque were significantly lower than patients without coronary plaque (p<0.0001, p=0.001, and p=0.001, p<0.0001, respectively) (Figure3).

There was no statistically significant difference between L-ADI of patients with coronary plaque and those without coronary plaque (figure3).
Fig. 1: Measurement of the end-systolic (A1 to C1) and end-diastolic (A2 to C2) cross-sectional-areas for the ascending and proximal descending aorta at the level of pulmonary trunk bifurcation, distal descending aorta at the level the middle cardiac vein (long white arrow) entering into the coronary sinus (short white arrow), and for the level of calcified plaque (black arrow).

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Fig. 2: Comparison of aortic distensibility index (ADI) between patients with and without aortic plaque. (A-ADI: Ascending ADI; PD-ADI: proximal descending ADI; DD-ADI: distal descending ADI; T-ADI: total ADI; L-ADI: local ADI at the level of aortic plaque).

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Fig. 3: Comparison of aortic distensibility index (ADI) between patients with and without aortic plaque. (A-ADI: Ascending ADI; PD-ADI: proximal descending ADI; DD-ADI: distal descending ADI; T-ADI: total ADI; L-ADI: local ADI at the level of aortic plaque).

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**Table 1:** Scan parameters for cardiac CT

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<table>
<thead>
<tr>
<th>Variable</th>
<th>Total cohort (n=150)</th>
<th>Patients without plaque (n=75)</th>
<th>Patients with plaque (n=75)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.0 ± 13.5</td>
<td>50.37 ± 12.24</td>
<td>66.92 ± 8.86</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male</td>
<td>74 (49.3%)</td>
<td>41 (54.7%)</td>
<td>33 (44%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>45 (30.0%)</td>
<td>4 (11.8%)</td>
<td>15 (51.7%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>75 (50%)</td>
<td>35 (46.7%)</td>
<td>40 (53.3%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>84 (56.0%)</td>
<td>30 (40.0%)</td>
<td>54 (72.0%)</td>
<td>0.01</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.0 ± 3.65</td>
<td>26.97 ± 3.14</td>
<td>27.09 ± 4.43</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Smoking</td>
<td>29 (19.3%)</td>
<td>13 (17.3%)</td>
<td>16 (21.3%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Coronary plaque</td>
<td>45 (30.0%)</td>
<td>5 (6.7%)</td>
<td>40 (53.3%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Coronary calcium score</td>
<td>173.55 ± 213.52</td>
<td>49.19 ± 166.32</td>
<td>176.54 ± 249.90</td>
<td>0.008</td>
</tr>
<tr>
<td>A-ADI (mmHg⁻¹×10³)</td>
<td>1.45 ± 1.34</td>
<td>2.21 ± 1.37</td>
<td>0.68 ± 0.73</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PD-ADI (mmHg⁻¹×10³)</td>
<td>1.52 ± 1.50</td>
<td>2.2 ± 1.63</td>
<td>0.84 ± 0.96</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>DD-ADI (mmHg⁻¹×10³)</td>
<td>1.42 ± 1.27</td>
<td>2.1 ± 1.17</td>
<td>0.76 ± 1.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>T-ADI (mmHg⁻¹×10³)</td>
<td>1.47 ± 1.12</td>
<td>2.17 ± 1.02</td>
<td>0.76 ± 0.71</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>L-ADI (mmHg⁻¹×10³)</td>
<td>0.63 ± 0.61</td>
<td>-</td>
<td>0.631 ± 0.61</td>
<td></td>
</tr>
</tbody>
</table>

BMI: body mass index; ADI: aortic distensibility index; A-ADI: Ascending ADI; PD-ADI: proximal descending ADI; DD-ADI: distal descending ADI; T-ADI: total ADI; L-ADI: local ADI at the level of plaque.

**Table 2:** Overall and comparative characteristics of the patients

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Conclusion

Similar to the previous studies demonstrating relationship between the presence of TAP and classic risk factors of coronary atherosclerosis [1,2,3,4], in the present study, patients with TAP were more likely to have older age, diabetes mellitus, hypercholesterolemia, coronary plaque, and higher coronary calcium score. This may indicate the similar atherosclerotic process and its risk factors in coronary arteries and aorta. The significant association between TAP and coronary plaque in our study and previous studies [5,6,7,8,9,10,11] further supports the possibility of same pathogenesis of aortic and coronary atherosclerosis.

In addition to more comprehensive estimation of aortic distensibility compared to the mentioned previous studies, the novelty of the present study was demonstration of the correlation between aortic distensibility and total aortic calcium score. In other words, we showed that the more calcium in aortic wall, the stiffer the aorta.

There were no significant differences in ADIs between patients with non-calcified TAPs and those without TAPs in our study. This may be explained by the fact that non-calcified plaques represent early stages of atherosclerosis with higher preserved elasticity than calcified plaques which develop at later stages of the atherosclerosis process.

Considering the interrelationship between thoracic aortic calcification, decreased aortic distensibility, and coronary calcification, as in our study, and also the significant adverse effects of these factors, it seems to be logical to consider the thoracic aortic calcification may be clinically almost as important as coronary calcification, especially in the presence of other cardiovascular risk factors. In this regard, thoracic aortic calcium score may be a possible substitute for coronary calcium score in patients that the evaluation of their coronary calcium score is impossible due to the presence of coronary stents or deployed surgical clips.

In summary, our study demonstrated that a less distensible thoracic aorta is associated with calcified aortic and coronary plaques. Aortic plaque is an independent predictor of the stiffer aorta.
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