INTRODUCTION
Atherosclerosis affects different vascular beds and polyvascular involvement is a common manifestation. The lower extremity peripheral arterial disease (PAD) is associated with high prevalence of coronary artery disease (CAD) and increased cardiovascular risk. Importantly, despite high prevalence of CAD, the significant proportion of subjects with PAD does not report cardiac symptoms. To date, the prevalence and extent of CAD in PAD was estimated using CTCA.

OBJECTIVES
The aim of the study was to determine the value of CTCA to assess coronary artery atherosclerosis and to evaluate the relationship between coronary artery plaque characteristics and severity of peripheral atherosclerosis in subjects with lower-extremity PAD and no cardiac symptoms.

RESULTS
CTCA revealed CAD in 56 subjects. Twenty-two had obstructive CAD. The mean ankle–brachial index (ABI) was 0.64 ± 0.16. Twenty-six individuals demonstrated abnormal carotid artery intima–media thickness (IMT). ABI lower than median, if compared with ABI equal of higher than median, was associated with a higher proportion of obstructive multivessel to single vessel CAD (8:4 vs. 1:9; \( P = 0.01 \)) and higher number of coronary artery segments with mixed plaques (2.3 ± 2.2 vs. 1.2 ± 1.3; \( P = 0.02 \)). Comparing patients with abnormal and normal IMT, the former demonstrated higher proportion of obstructive multivessel to single-vessel CAD (7:3 vs. 2:10; \( P = 0.01 \)) and higher number of coronary artery segments with noncalcified (1.9 ± 3.2 vs. 0.6 ± 1.4; \( P = 0.04 \)) and mixed plaques (2.3 ± 2.1 vs. 1.3 ± 1.7; \( P = 0.049 \)).

CONCLUSIONS
CTCA may be effective to detect CAD in subjects with lower-extremity PAD and no cardiac symptoms. The low ABI and abnormal IMT are associated with more extensive CAD and higher burden of high-risk coronary artery plaques.
invasive coronary angiography, which showed that even >70% of subjects with PAD may have CAD. Recently, computed tomography coronary angiography (CCTA) has emerged as a new imaging modality, which enables noninvasive evaluation of coronary artery atherosclerosis. Currently, the European Society of Cardiology guidelines on the management of coronary artery disease has clarified the role of CCTA to detect CAD in subjects with low intermediate pretest probability of the disease. Moreover, prior studies have demonstrated that the extent and severity of CAD as well as the composition of coronary artery plaques determined by CCTA are related to clinical outcomes and may be used to stratify cardiovascular risk. Nevertheless, the value of CCTA to assess coronary artery atherosclerosis in subjects with lower extremity PAD and no cardiac symptoms has not been evaluated. Importantly, ESC guidelines on the diagnosis and treatment of peripheral artery diseases only raise the question of whether such identification of CAD would improve clinical outcomes. Of note, no data exist to assess the relationship between coronary artery plaque characteristics and severity of peripheral atherosclerosis. Therefore, the aim of the study was to determine the value of CCTA to assess the presence and extent of CAD and to evaluate the relationship between coronary artery plaque characteristics and severity of peripheral atherosclerosis in subjects with lower extremity PAD and no cardiac symptoms.

**PATIENTS AND METHODS**  
**Study population**  
Sixty-five patients with intermittent claudication due to PAD and no cardiac symptoms were enrolled in the study. The exclusion criteria included age <50 years, known coronary artery disease, impaired left ventricular systolic function, irregular heart rate, significant valvular heart disease, prior peripheral artery intervention and/or surgery, prior stroke, respiratory failure, obesity, pregnancy, known iodinated contrast allergy, impaired renal function (serum creatinine ≥1.5 mg/dl) and thyroid disorders. All patients underwent carotid ultrasound evaluation, CCTA and estimation of the ankle-brachial index (ABI). This study was approved by the local ethics committee and informed consent was obtained from each patient.

**Ankle-brachial index**  
A hand-held Doppler probe (Hadeco Bidop ES-100V3, Hadeco Inc, Arima, Japan) was used by qualified sonographer blinded to clinical, carotid ultrasound, and CCTA data to obtain systolic pressures in the right and left brachial, dorsalis pedis, and posterior tibial arteries, as previously described. Briefly, ABI was calculated by dividing the mean of the dorsalis pedis and posterior tibial pressures in each leg by the mean of the 4 brachial pressures. Each pressure was measured twice. Zero values for the dorsalis pedis and posterior tibial pulses were set to missing for the ABI calculation. Average brachial pressures in the arm with highest pressure were used when 1 brachial pressure was higher than the opposite brachial pressure in both measurement sets and the 2 brachial pressures differed by 10 mmHg or more in at least 1 measurement set because, in such cases, subclavian stenosis was possible. The lowest leg ABI was used in analyses.

**Carotid artery intima–media thickness**  
The evaluation of carotid intima–media thickness (IMT) were assessed using high-resolution B-mode 3–9 MHz linear transducer (IE 33 ultrasound system, Philips Medical Systems, Andover, Massachusetts, United States) as previously described. Briefly bilateral measurements were made at the posterior wall of the bulb of the common carotid artery and at the internal carotid artery through a program of automatic edge detection (QLAB version 7.1 software, Philips Medical Systems, Andover, Massachusetts, United States). The highest IMT was used for analysis.

**Computed tomography coronary angiography acquisition**  
CTCA was performed using dual source computed tomography (DSCT) (Somatom Definition, Siemens Medical Solutions, Erlangen, Germany). In patients with a heart rate above 70/min and no contraindication to β-blocker, 25 to 100 mg of metoprolol was given orally 1 hour before CTCA examinations to achieve heart rate below 70/min. In cases of insufficient heart rate reduction or the presence of contraindications to β-blockade, CTCA was performed at heart rates higher than 70/min. Patients were premedicated with sublingual nitrates directly before CTCA acquisition.

A noncontrast axial scan was performed craniocaudally (3-mm section thickness without gap) at 70% of R–R interval with prospective ECG triggering to obtain coronary artery calcium score (CACS). Subsequently, the arrival time of contrast agent to the ascending aorta was determined at the level of the carina with the use of the test bolus method (20 ml of iopromid 370 contrast followed by a 20 ml saline chaser). Iopromid 370 contrast (1.2 ml/kg) was injected at a rate of 5.5 ml/s followed by a 40 ml saline chaser at similar rates. Retrospective ECG-gated image acquisition was performed during inspiratory breath hold. The imaging parameters were: 2 × 64 × 0.6 mm collimation, 330 ms rotation time, table feed of 9.2 mm per rotation, tube voltage 120 kV, an effective tube current of 300 to 400 mA and a temporal resolution of 165 ms. Electrocardiographically controlled tube current modulation was applied in all patients. Tube voltage was reduced to 100kV, when patient’s body mass index was less than 25 kg/m². The effective radiation dose estimate was 10.1 ±2.8 mSv.

Images were reconstructed using a B26f kernel with an image matrix of 512 × 512 pixels. A DSCT scanner utilized a slice thickness of 0.75 mm and an increment of 0.5 mm with an ECG-gated half-scan algorithm. A multiphase reconstruction
Computed tomography coronary angiography analysis II CTCA examinations were evaluated by experienced readers who were blinded to the clinical data, ABI, and IMT measurements. Coronary artery calcium was defined as a plaque of at least 3 contiguous pixels with attenuation of 130 HU or greater. The CACS was calculated according to the Agatston method. The total CACS was calculated from the sum of each of the 4 major epicardial vessels (left main, left anterior descending, circumflex, and right coronary arteries).

A modified American Heart Association 16-segment model of the coronary arteries was employed and segments with a diameter of ≤1.5 mm were excluded from the analysis. The remaining segments were determined to be interpretable or uninterpretable, and interpretable segments were assessed for the presence and location of atherosclerotic plaques using axial and curved multiplanar reconstructions. Coronary plaques were defined as >1 mm² area structures within and/or adjacent to the coronary artery lumen with clear, well-defined borders. By comparing the plaque and contrast-enhanced vessel lumen density, 3 different types of plaques were reported: Plaques with higher density than the contrast enhanced lumen were defined as calcified, while those with lower density were labeled as noncalcified. Plaques with both calcified and noncalcified elements were considered mixed plaques. When ≥2 plaques of the same composition were present in a single coronary segment, only 1 plaque having that composition was assigned to that particular segment. The degree of luminal narrowing was compared to the nearest normal-appearing segment and plaques were classified as obstructive (≥50% luminal narrowing) or nonobstructive (<50% luminal narrowing). Coronary arteries were considered abnormal if plaque was present and normal if no plaque was identified. Patients with ≥1 obstructive plaque were considered to have obstructive CAD while those with only nonobstructive plaque(s) were labeled as having nonobstructive CAD.

Statistical analysis Continuous data are presented as a mean ± standard deviation and categorical data as prevalence (%). The normal distribution was verified using the Kolmogorov–Smirnov test. Unpaired categorical variables were compared by Fisher exact or χ² tests depending on the sample size. Student’s t test was used to assess differences between continuous variables. Spearman rank correlation was performed to assess the relationship between number of coronary artery segments with obstructive luminal narrowing and indexes of peripheral atherosclerosis. Differences were considered statistically significant at a P value of <0.05. Statistical analyses were performed using the SPSS software (version 12.0, SPSS Inc., Chicago, Illinois, United States).

RESULTS Baseline characteristics A total of 65 subjects (45 men, 20 women, mean age, 62.5 ±7.6 years) with lower extremity PAD formed the study group. All subjects denied typical/atypical cardiac chest pain. According to the Fontaine classification, 38 subjects were in stage Ia and 27 subjects in stage Ib. The reported maximal walking distance till claudication was 318 ±324 m. All subjects demonstrated a decreased ABI of <0.9 and 26 had increased carotid artery IMT. Thirty-three individuals reported current smoking, 25 prior smoking, 43 hypertension, 45 hypercholesterolemia, and 15 diabetes mellitus. Forty-four subjects were on aspirin, 4 on clopidogrel, 2 on ticlopidine, 15 on β-blockers, 19 on angiotensin-converting enzyme inhibitor, 8 on angiotensin receptor blocker, 22 on calcium antagonists, 16 on diuretics, and 1 on spironolactone. Forty subjects were on statins and 1 on fibrates. The clinical characteristics of the patients is shown in Table 1. The mean ABI was 0.64 ±0.16 with the median value of 0.63. Thirty-nine subjects had normal IMT bilaterally, whereas 26 demonstrated abnormal IMT on at least 1 side. Comparing subjects with ABI <median and ABI ≥median, no difference in age, sex, and cardiovascular risk factors prevalence was found (all P = nonsignificant [NS]). Subjects with abnormal IMT were older than those with normal IMT (65.2 ±8.3 vs. 61.2 ±6.4 years, P = 0.009). The prevalence of sex and cardiovascular risk factors in subjects with normal and abnormal IMT did not differ significantly (all P = NS).

Computed tomography coronary angiography feasibility The mean heart rate at scan time was 65 ±8 beats/min. Of a total of 1040 evaluated coronary segments, 165 segments were excluded due to a diameter <1.5 mm. Of the remaining 875 segments, 32 were considered uninterpretable. Segments were uninterpretable for the following reasons: a) severe calcifications: 18 segments, b) motion artifacts: 9 segments, c) image noise or poor opacification: 3 segments and stair step artifacts in 2 segments.

Computed tomography coronary angiography findings Normal coronary arteries were found in 9 patients. Of the remaining 56 patients, 22 had obstructive CAD and 34 nonobstructive CAD. Single-vessel CAD was present in 13 and multivessel CAD in 43 patients. Obstructive single-vessel CAD was found in 13 and obstructive multivessel CAD in 9 patients. Two subjects had obstructive left main coronary artery stenosis. Noncalcified, mixed, and calcified plaques were present in 73, 111, and 51 coronary artery segments, respectively. CTCA characteristics with respect to ABI and IMT values are shown in Table 2. No differences in
**DISCUSSION** The major finding of the study is that CTCA may be regarded as an effective diagnostic tool to depict CAD in subjects with lower CACS values and CAD prevalence were found between subjects with ABI < median and ABI ≥ median as well as between subjects with normal and abnormal IMT. Comparing subjects with ABI < median and ABI ≥ median, the former demonstrated higher proportion of multivessel to single-vessel coronary artery plaque extent (24:2 vs. 19:11, \( P = 0.01 \)), higher proportion of obstructive multivessel to single-vessel CAD (8:4 vs. 1:9, \( P = 0.01 \)) and higher number of coronary artery segments with mixed plaques (2.3 ±2.2 vs. 1.2 ±1.3, \( P = 0.02 \)). The number of coronary artery segments with noncalcified and calcified plaques did not differ between both groups. Subjects with abnormal IMT, when compared with those with normal IMT, demonstrated higher proportion of multivessel to single-vessel coronary artery plaque extent (21:1 vs. 22:12, \( P = 0.01 \)), higher proportion of obstructive multivessel to single vessel CAD (7:3 vs. 2:10, \( P = 0.01 \)), and higher number of coronary artery segments with any plaques (4.9 ±4.3 vs. 2.8 ±2.6, \( P = 0.02 \)), noncalcified plaques (1.9 ±3.2 vs. 0.6 ±1.4, \( P = 0.04 \)), and mixed plaques (2.3 ±2.1 vs. 1.3 ±1.7, \( P = 0.049 \)). The number of coronary artery segments with obstructive luminal narrowing tended to correlate with ABI (\( r = -0.22, P = 0.09 \)) and IMT (\( r = 0.24, P = 0.05 \)).

![Figure](image_url)

**TABLE 1** Baseline characteristics

<table>
<thead>
<tr>
<th></th>
<th>All (n = 65)</th>
<th>Abnormal IMT (n = 26)</th>
<th>Normal IMT (n = 39)</th>
<th>( P )</th>
<th>ABI &lt; median (n = 32)</th>
<th>ABI ≥ median (n = 33)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>men/women</td>
<td>45 (69)/20 (31)</td>
<td>20 (77)/6(23)</td>
<td>25 (64)/14(36)</td>
<td>0.41</td>
<td>20/12</td>
<td>25/8</td>
<td>0.38</td>
</tr>
<tr>
<td>age, y</td>
<td>62.5 ±7.6</td>
<td>65.2 ±8.3</td>
<td>61.2 ±6.4</td>
<td>0.03</td>
<td>62.6 ±6.7</td>
<td>62.5 ±8.5</td>
<td>0.93</td>
</tr>
<tr>
<td>smoking</td>
<td>58 (89)</td>
<td>23 (88)</td>
<td>35 (90)</td>
<td>0.81</td>
<td>29</td>
<td>29</td>
<td>1.0</td>
</tr>
<tr>
<td>current smoker</td>
<td>33 (51)</td>
<td>12 (46)</td>
<td>21 (54)</td>
<td>0.72</td>
<td>16</td>
<td>17</td>
<td>0.90</td>
</tr>
<tr>
<td>exsmoker</td>
<td>25 (38)</td>
<td>11 (42)</td>
<td>14 (36)</td>
<td>0.79</td>
<td>13</td>
<td>12</td>
<td>0.92</td>
</tr>
<tr>
<td>packyears</td>
<td>38.9 ±18.3</td>
<td>41.8 ±19.0</td>
<td>36.6 ±17.8</td>
<td>0.29</td>
<td>38.8 ±14.8</td>
<td>38.9 ±21.4</td>
<td>0.99</td>
</tr>
<tr>
<td>hypertension</td>
<td>43 (66)</td>
<td>19 (73)</td>
<td>24 (62)</td>
<td>0.49</td>
<td>22</td>
<td>21</td>
<td>0.86</td>
</tr>
<tr>
<td>hypercholesterolemia</td>
<td>45 (69)</td>
<td>19 (73)</td>
<td>26 (67)</td>
<td>0.78</td>
<td>22</td>
<td>23</td>
<td>0.85</td>
</tr>
<tr>
<td>diabetes mellitus</td>
<td>15 (23)</td>
<td>8 (31)</td>
<td>7 (18)</td>
<td>0.37</td>
<td>7</td>
<td>8</td>
<td>0.95</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.3 ±4.6</td>
<td>25.9 ±4.7</td>
<td>26.6 ±4.5</td>
<td>0.94</td>
<td>25.3 ±5.0</td>
<td>27.3 ±3.9</td>
<td>0.12</td>
</tr>
<tr>
<td>ABIa</td>
<td>0.64 ±0.16</td>
<td>0.65 ±0.91</td>
<td>0.64 ±0.17</td>
<td>0.81</td>
<td>0.50 ±0.09</td>
<td>0.78 ±0.08</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>GFR, ml/min/1.73 m²</td>
<td>80.3 ±10.9</td>
<td>79.8 ±11.2</td>
<td>80.9 ±10.8</td>
<td>0.69</td>
<td>79.7 ±12.5</td>
<td>81.3 ±8.9</td>
<td>0.57</td>
</tr>
<tr>
<td>TC, mmol/l</td>
<td>5.35 ±1.26</td>
<td>5.28 ±1.23</td>
<td>5.40 ±1.29</td>
<td>0.71</td>
<td>5.23 ±1.32</td>
<td>5.5 ±1.20</td>
<td>0.44</td>
</tr>
<tr>
<td>LDL-C, mmol/l</td>
<td>3.23 ±1.15</td>
<td>3.30 ±1.22</td>
<td>3.17 ±1.11</td>
<td>0.64</td>
<td>3.08 ±1.08</td>
<td>3.37 ±1.21</td>
<td>0.32</td>
</tr>
<tr>
<td>HDL-C, mmol/l</td>
<td>1.45 ±0.39</td>
<td>1.44 ±0.43</td>
<td>1.45 ±0.36</td>
<td>0.87</td>
<td>1.52 ±0.37</td>
<td>1.38 ±0.40</td>
<td>0.13</td>
</tr>
<tr>
<td>TG, mmol/l</td>
<td>1.59 ±0.81</td>
<td>1.73 ±0.89</td>
<td>1.49 ±0.75</td>
<td>0.26</td>
<td>1.42 ±0.57</td>
<td>1.75 ±0.97</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Data are presented as mean ± standard deviation or number (percentage).

a The lowest values were taken for analysis.


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**FIGURE** Computed tomography coronary angiography in a 59-year-old man with peripheral arterial disease and no chest pain showed 2 vessel obstructive coronary artery disease. The ankle-brachial index and carotid artery intima media thickness was 0.52 and 1.2 at the right side, and 0.82 and 1.1 at the left side, respectively. Coronary artery calcium score was 996. Curved multiplanar reconstructions demonstrate significant narrowing of mid-portion of the left anterior descending artery (A) and mid-portion of the right coronary artery (B).
extremity PAD and no cardiac symptoms. The extent of coronary artery atherosclerosis and number of disease vessels is related to the severity of lower-limb ischemia and IMT. Importantly, subjects with lower ABI and abnormal IMT demonstrated a higher burden of high-risk coronary artery plaques, which are known to be associated with higher incidence of adverse cardiac events.

To date, numerous studies have demonstrated high prevalence of CAD in subjects with lower extremity PAD. The findings of the current study correspond to prior observations and show that obstructive CAD is frequent in subjects with PAD and no cardiac symptoms. Interestingly, we have found the presence of obstructive CAD in one-third of the enrolled subjects with high percentage of multivessel obstructive CAD.

The current study shows that obstructive CAD, which are known markers of polyvascular atherosclerosis, in consequence, the presence of low ABI or abnormal IMT might be used as indicators of coronary artery involvement in subjects with PAD and no cardiac symptoms. Prior studies have shown that both IMT and ABI are strong predictors of cardiovascular events including death and myocardial infarction. Interestingly, we observed an association between lower ABI and higher number of mixed coronary artery plaques as well as abnormal IMT and higher number of noncalcified and mixed coronary artery plaques. These associations might reflect differences in atherosclerotic process in studied subgroups and might explain the link between higher number of cardiovascular events and abnormal ABI and IMT.

Table 2: Computed tomography coronary angiography characteristics with regard to intima-media thickness and ankle-brachial index values

<table>
<thead>
<tr>
<th></th>
<th>All (n = 65)</th>
<th>Abnormal IMT (n = 26)</th>
<th>Normal IMT (n = 39)</th>
<th>P</th>
<th>ABI &lt; median (n = 32)</th>
<th>ABI ≥ median (n = 33)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CACS</td>
<td>355 ± 644</td>
<td>344 ± 555</td>
<td>362 ± 708</td>
<td>0.91</td>
<td>405 ± 702</td>
<td>306 ± 590</td>
<td>0.55</td>
</tr>
<tr>
<td>CAD</td>
<td>56 (86)</td>
<td>22 (85)</td>
<td>34 (87)</td>
<td>1.00</td>
<td>26 (81)</td>
<td>30 (91)</td>
<td>0.30</td>
</tr>
<tr>
<td>SVD/MVD</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>obstructive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>13 (20%)/43 (66%)</td>
<td>1 (4%)/21 (81%)</td>
<td>12 (31%)/22 (56%)</td>
<td>0.009</td>
<td>2 (6%)/24 (75%)</td>
<td>11 (33%)/19 (58%)</td>
<td>0.01</td>
</tr>
<tr>
<td>CACS</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>plaques, seg</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>calcified</td>
<td>22 (34%)</td>
<td>10 (38%)</td>
<td>12 (31%)</td>
<td>0.71</td>
<td>12 (38%)</td>
<td>10 (30%)</td>
<td>0.59</td>
</tr>
<tr>
<td>noncalcified</td>
<td>13 (20%)/9 (14%)</td>
<td>3 (11%)/7 (27%)</td>
<td>10 (26%)/2 (5%)</td>
<td>0.03</td>
<td>4 (13%)/8 (25%)</td>
<td>9 (27%)/1 (3%)</td>
<td>0.01</td>
</tr>
<tr>
<td>plaques, seg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mixed plaques,</td>
<td>3.6 ± 3.5</td>
<td>4.9 ± 4.3</td>
<td>2.8 ± 2.6</td>
<td>0.02</td>
<td>3.9 ± 4.0</td>
<td>3.3 ± 2.9</td>
<td>0.47</td>
</tr>
<tr>
<td>calcified</td>
<td>1.1 ± 2.4</td>
<td>1.9 ± 3.2</td>
<td>0.6 ± 1.4</td>
<td>0.04</td>
<td>1.1 ± 2.9</td>
<td>1.2 ± 1.9</td>
<td>0.92</td>
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<tr>
<td>plaques, seg</td>
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<td></td>
</tr>
<tr>
<td>mixed plaques,</td>
<td>1.7 ± 1.9</td>
<td>2.3 ± 2.1</td>
<td>1.3 ± 1.7</td>
<td>0.049</td>
<td>2.3 ± 2.2</td>
<td>1.2 ± 1.3</td>
<td>0.02</td>
</tr>
<tr>
<td>calcified</td>
<td>0.8 ± 1.3</td>
<td>0.9 ± 1.7</td>
<td>0.7 ± 1.0</td>
<td>0.48</td>
<td>0.6 ± 0.9</td>
<td>1.0 ± 1.7</td>
<td>0.26</td>
</tr>
</tbody>
</table>

Data are presented as mean ± standard deviation or number (percentage).

Abbreviations: CACS – coronary artery calcium score, CAD – coronary artery disease, MVD – multivessel disease, SVD – single-vessel disease, others – see Table 1

There are several limitations of the study that should be acknowledged. First, the number of studied patients was small. Second, due to limitation in spatial resolution of CCTA, we have excluded coronary artery segments with a diameter of ≤1.5 mm. Consequently, we might have underestimated the extent and severity of coronary artery plaque burden. Finally, we have not performed invasive coronary angiography to assess the diagnostic value of CCTA.
Concluding, CCTA may be an effective diagnostic tool to detect CAD in subjects with PAD and no cardiac symptoms. The low ABI and abnormal IMT are associated with more extensive CAD and higher burden of high-risk coronary artery plaques, suggesting potential explanation of the link between increased cardiovascular risk and abnormal ABI and IMT values.

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REFERENCES

ARTYKUŁ ORYGINALNY

Charakterystyka zmian miażdżycowych w tętnicach wieńcowych w tomografii komputerowej u chorych z miażdżycą tętnic kończyn dolnych bez dolegliwości sercowych

Tomasz Miszalski-Jamka1,2, Sabina Lichołai1, Krzysztof Karwat1, Bartosz Laskowicz1, Agnieszka Okraska-Bylica1, Tadeusz Wilkosz2, Małgorzata Konieczyńska1, Mariusz Trystuła3, Lidia Słowik4, Małgorzata Urbańczyk1, Mieczysław Pasowicz2, Przemysław Jaźwiec2

1 Ośrodek Diagnostyki, Prewencji i Telemedycyny, Szpital im. Jana Pawła II w Krakowie, Kraków
2 Zakład Radiologii Lekarskiej i Diagnostyki Obrazowej, 4. Wojskowy Szpital Kliniczny z Polikliniką we Wrocławiu, Wrocław
3 Ośrodek Chirurgii Serca, Naczyni i Transplantologii, Szpital im. Jana Pawła II w Krakowie, Kraków
4 Oddział Kliniczny Angiologii, Szpital Uniwersytecki w Krakowie, Kraków

Adres do korespondencji:
dr n. med. Tomasz Miszalski-Jamka, Zakład Radiologii Lekarskiej i Diagnostyki Obrazowej, 4. Wojskowy Szpital Kliniczny z Polikliniką, ul. Węgla 5, 50-981 Wrocław, tel./fax: +48-71-766-04-80, e-mail: miszalt@mp.pl

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STRESZCZENIE

wprowadzenie Tomografia komputerowa tętnic wieńcowych (computed tomography coronary angiography – CTCA) umożliwia nieinwazyjną ocenę miażdżycy tętnic wieńcowych. Wartość CTCA w ocenie choroby wieńcowej (coronary artery disease – CAD) u chorych z miażdżycą tętnic kończyn dolnych (peripheral artery disease – PAD) bez objawów sercowych nie jest znana. Ponadto związek między charakterystyką zmian miażdżycowych tętnic wieńcowych a nasileniem miażdżycy obwodowej w tej grupie chorych nie został dostatecznie zbadany.

cel Określenie przydatności CTCA w ocenie miażdżycy tętnic wieńcowych oraz ustalenie związku pomiędzy charakterystyką zmian miażdżycowych tętnic wieńcowych a nasileniem miażdżycy obwodowej u chorych z PAD bez objawów sercowych.

pacienci i metody CTCA wykonano u 65 chorych (45 mężczyzn, 20 kobiet, średnia wieku 62,5 ± 7,6 roku) z PAD bez objawów sercowych.

wyniki CAD stwierdzono w CTCA u 56 chorych. Istotne zwężenie tętnic wieńcowych wystąpiło u 22 chorych. Wskaźnik kostka–ramię (ankle–brachial index – ABI) wynosił 0,64 ± 0,16. Nieprawidłową grubość kompleksu intima–media (IMT) w tętnicach szyjnych stwierdzono u 26 osób. ABI mniejszy od mediany wartości tego parametru, w porównaniu z ABI większym lub równym medianie, był związany z większą częstością występowania istotnej choroby wielonaczyniowej w porównaniu do jednonaczyniowej (8:4 vs 1:9; p = 0,01) oraz większą liczbą segmentów tętnic wieńcowych ze zmianami miażdżycowymi o charakterze mieszonym (2,3 ± 2,2 vs 1,2 ± 1,3; p = 0,02). Chorzy z nieprawidłowym IMT, w porównaniu z tymi z prawidłowym IMT, charakteryzowali się większą częstością występowania istotnej choroby wielonaczyniowej w porównaniu do jednonaczyniowej (7:3 vs 2:10; p = 0,01) oraz większą liczbą segmentów tętnic wieńcowych z niewątpliwymi zmianami miażdżycowymi (1,9 ± 3,2 vs 0,6 ± 1,4; p = 0,04) oraz tymi o charakterze mieszonym (2,3 ± 2,1 vs 1,3 ± 1,7; p = 0,049).

wnioski CTCA może być skuteczna w rozpoznawaniu CAD u chorych z PAD bez objawów sercowych. Niski ABI oraz nieprawidłowa IMT są związane z bardziej nasiloną CAD oraz większą częstością występowania w tętnicach wieńcowych blaszek miażdżycowych wiążących się z dużym ryzykiem sercowo–naczyniowym.