

Aortic elastic properties predict occult coronary artery disease: a multidetector row computed tomography study

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Abstract

Background: Multidetector row computed tomography (MDCT) is an attractive noninvasive imaging modality to detect coronary atherosclerotic plaques which may be underestimated by conventional angiography. However, its routine clinical use is limited due to contrast-associated problems, high cost, inapplicability at bedside and exposure to radiation. Thus, exploring safer and more practical measurements to predict occult coronary artery disease (CAD) is required.

Aim: To demonstrate the predictive value of aortic elastic properties for occult CAD diagnosed by MDCT.

Methods: Forty subjects with angiographically normal coronary arteries were consecutively included in our study. They underwent MDCT including indications and were divided into a no CAD group (23 subjects, 11 males, 46 ± 8 years) and an occult CAD group (17 subjects, 12 males, 48 ± 10 years), with respect to the presence of coronary plaque. As a control group, 19 consecutive patients with angiographically proven CAD (16 males, 52 ± 6 years) were included. Aortic stiffness index (ASI), aortic distensibility and aortic strain were calculated from the aortic diameters measured by echocardiography and blood pressure obtained by sphygmomanometry.

Results: It was found that ASI, aortic distensibility and aortic strain were significantly different in the occult CAD group compared to the no CAD group ($p = 0.008$, $p = 0.01$, $p = 0.03$, respectively) and to the evident CAD group ($p = 0.01$, $p = 0.02$, $p = 0.02$). They also differed significantly between the no CAD and the evident CAD groups. Receiver operating characteristics analysis for ASI, to distinguish between the occult CAD group and the no CAD group, revealed an area under the curve of 0.80 (confidence interval 0.68–0.94, $p = 0.004$) and that the cut-off value of 3.42 could significantly predict patients with occult CAD (sensitivity: 78%; specificity: 63%).

Conclusions: Measurement of ASI is an easily applicable and safe method with its non-radiographic ability for the assessment of aortic stiffness, and it may be useful to predict subclinical atherosclerosis in clinical practice. A cut-off value of 3.42 for ASI may guide to refer individuals to preventive strategies to reduce atherosclerosis progression.

Key words: coronary artery disease, computed tomography, atherosclerosis, angiography

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INTRODUCTION

Diffuse coronary artery disease (CAD) is an important predictor of adverse cardiovascular (CV) events [1]. The extent and nature of overall CAD are usually underestimated by conventional coronary angiography, and more accurately

quantified with intravascular ultrasound (IVUS). Multidetector row computed tomography (MDCT) is inferior to IVUS, but it might be a valuable noninvasive alternative to assess overall CAD burden [2]. Nevertheless, the widespread use of MDCT in clinical practice is limited because of exposure to

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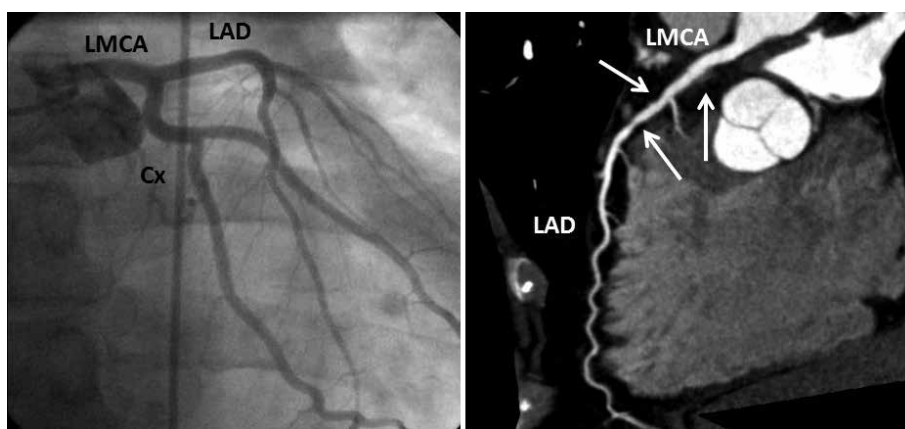


Figure 1. Multidetector row computed tomography demonstrated atherosclerotic plaques with positive remodelling (white arrows) in left main coronary artery (LMCA) and proximal left anterior descending artery (LAD) (right side), while conventional coronary angiography revealed normal coronary arteries (left side) in a patient with occult coronary artery disease; Cx — circumflex artery

radiation, high cost, use of an iodinated intravenous contrast agent, and the contrast-associated problems. Thus, exploring safer and more practical measurements is required for the selection of high-risk individuals for CAD instead of routine MDCT procedure.

Aortic stiffness is an independent predictor of all-cause and CV mortality and morbidity [3–5]. Arterial stiffening occurs normally with ageing, but it also correlates with the prevalence of atherosclerosis [6]. The most obvious consequences of arterial stiffening are increased pulsatile blood pressure due to higher systolic blood pressure (SBP) and lower diastolic blood pressure (DBP), thereby causing increased left ventricular (LV) afterload and altering coronary perfusion [7]. Aortic stiffness has also been shown to relate to the degree of CAD assessed by conventional coronary angiography [8]. However, the role of aortic stiffness in predicting patients with occult CAD identified by MDCT is unclear.

The aim of this study was to explore the predictive value of aortic elastic properties for patients who had coronary plaques documented by MDCT although their prior conventional coronary angiography had revealed normal coronary arteries.

METHODS

Study population

Forty subjects with normal coronary arteries identified by conventional angiography were consecutively included in our study. All subjects underwent careful physical examination. Prior history of medication and CV risk factors were individually recorded. MDCT was performed to all subjects including indications to detect any coronary plaque formation. Twenty-three subjects had no atherosclerotic plaque in MDCT (the 'no CAD group'). Coronary plaques were detected by MDCT in the remaining 17 subjects, while their conventional coronary angiographies revealed normal coronary arteries

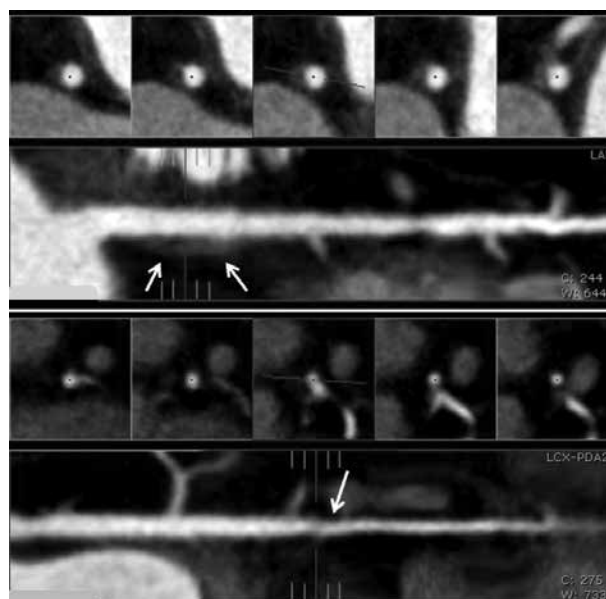


Figure 2. The patient in Figure 1 showed a huge atherosclerotic plaque (white arrows) without any luminal narrowing in another view of left main coronary artery in multidetector row computed tomography imaging (upper side). There were also small atherosclerotic plaques (white arrow) in circumflex artery (lower side)

(the 'occult CAD group') (Figs. 1, 2). Patients with a history of revascularisation, acute coronary syndrome, chronic heart failure, atrial fibrillation, congenital heart disease, more than mild valve disease or more than mild pulmonary hypertension were not included.

A control group was included in this study. This consisted of 19 consecutive patients with angiographically proven CAD (the 'evident CAD group'). Twelve of them had single-vessel disease, five had double-, and two had triple-vessel disease. Symptomatic patients had undergone revascularisation either

with percutaneous coronary intervention or coronary artery bypass grafting surgery at least six months before participating in the study. Aortic elastic properties of the control group were also evaluated.

The local ethics committee approved the study protocol. Participants received complete and clear explanations about the study and gave written informed consent before participation.

Blood sampling and laboratory tests

Venous blood samples were collected in an overnight fasting state before MDCT procedure. Plasma and serum were separated by centrifugation and stored at -80°C until analysis. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG) and glucose were analysed by enzymatic standard methods. Low-density lipoprotein cholesterol (LDL-C) was calculated according to Friedewald's formula.

Aortic stiffness measurement

Transthoracic echocardiography was performed by one of the authors, who was not informed of the patients' clinical data, using a commercially available ultrasound system (Vingmed System 5 — General Electric, Horten, Norway) with a 2.5 MHz transducer. Recordings were taken with patients in the lateral decubitus position. Three consecutive cycles were averaged for every parameter. Ascending aortic diameters were measured by M-mode echocardiogram at a level of 3 cm above the aortic valve. Internal aortic diameters were measured by means of a caliper in systole and diastole as the distance between the trailing edge of the anterior aortic wall and the leading edge of the posterior aortic wall [9–11]. Aortic systolic diameter (SD) was measured at the time of full opening of the aortic valve and diastolic diameter (DD) was measured at the peak of QRS complex of the electrocardiogram (ECG). Measurements were performed by the same investigator; the intraobserver variability of aortic diameter measurements was 2.1%. Aortic elastic properties were evaluated using the following parameters.

The percentage change of the aortic root was calculated as $\Delta\text{Ao} (\%) = 100 \times (\text{SD} - \text{DD})/\text{DD}$ to obtain aortic strain [9, 11, 12]. Aortic distensibility was calculated from the formula: $\Delta\text{A} : (\text{A} \times \text{PP}) = \pi \times [(\text{SD}/2)^2 - (\text{DD}/2)^2] : [\pi \times (\text{DD}/2)^2 \times \text{PP}]$, with 'A' being the cross-sectional aortic lumen area and 'PP' being pulse pressure [13]. The aortic stiffness index (ASI) was calculated as $\beta = \ln(\text{SBP}/\text{DBP}) / (\text{SD} - \text{DD})/\text{DD}$ [9, 11–14]. The auscultatory method of blood pressure measurements was performed using a properly calibrated and validated, conventional sphygmomanometer. Subjects were seated quietly for at least 5 min in a chair, with feet on the floor and arm supported at heart level. A cuff bladder encircling at least 80% of the arm was used and a pressure drop rate of approximately 2 mm Hg/s was applied. SBP is the point at which the first of two or more sounds are heard (Korotkoff phase 1) and DBP is the point before the disappearance of

sounds (Korotkoff phase 5) [15]. Three consecutive measurements were averaged. PP was simply defined as the difference between SBP and DBP.

MDCT scan protocol, data acquisition

MDCT was performed with a 64-slice MDCT scanner (Philips Brilliance, Philips Medical Systems, Eindhoven, Netherlands). Imaging parameters were as follows: gantry rotation time of 330 ms, tube voltage of 120 kV, and effective tube current of 800 mA. Pharmacologic premedication with β -blockers for optimisation of the heart rate was performed as previously described [16]. The ECG was digitally recorded during data acquisition and was stored with the unprocessed computed tomography (CT) data set. Depending on the total scan time, 80–110 mL non-ionic contrast material (Iomeron 400, Bracco s.p.a., Milan, Italy) was administered in the ante-cubital vein with a flow rate of 5.0 mL/s, followed by a 50 cc saline bolus. Automated peak enhancement detection in the descending aorta was used for timing of the bolus using a threshold of +130 Hounsfield units (HU). Data acquisition was performed during an inspiratory breath hold of approximately 10 s. All data sets were reconstructed with retrospective gated data method in 45%, 80% and as default 75% of the RR interval, with a slice thickness of 0.9 mm. In a case of high-density blooming artifacts, sharper reconstruction kernels and changes in centre and width were explored to improve image quality. Finally, acquired CT data was transferred to a dedicated workstation (EBW; Philips Medical Systems). The reconstruction algorithm gets data from a single heart beat obtained during half-X-ray tube rotation, resulting in a temporal resolution of 165 ms.

MDCT scans were independently analysed by a radiologist and a cardiologist who were unaware of the results of conventional coronary angiography and blood measures. All coronary segments > 2 mm in diameter were evaluated using curved multiplanar reconstruction images. The examined vessels were viewed in images reconstructed along the axis of the vessel of interest and in cross-sectional images perpendicular to the centre line of the vessel (Fig. 2). Plaques per segment were assessed as previously described [17–19]. Noncalcified plaque was defined as any discernible structure that could be assigned to the coronary artery wall that had the CT attenuation below the contrast-enhanced coronary lumen but above the surrounding connective tissue/epicardial fat. Any structure with a CT attenuation of 130 HU that could be visualised separately from the contrast-enhanced coronary lumen (either because it was embedded within noncalcified plaque or because its density was above the contrast-enhanced lumen) was defined as calcified atherosclerotic plaque. To measure plaque volume in each coronary segment, contiguous 1-mm-thick cross-sectional images of the coronary arteries were rendered and displayed with a fixed setting (700-HU window, 200-HU level). Plaque

areas were manually traced and volume was calculated by multiplying area and slice increment [19].

Occult CAD was defined by MDCT as any evidence of atherosclerotic plaque of any size, calcified or noncalcified, and without luminal narrowing.

Statistical analysis

Statistical analysis was performed using a statistical software program (SPSS for Windows, version 15.0; SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean \pm standard deviation whereas categorical variables were expressed as percentages. Parametric data was compared using the student's t-test and the Pearson χ^2 test. Nonparametric data was compared using the Mann-Whitney U test. Spearman's correlation analysis was performed to assess the correlation between variables. Receiver operating characteristic (ROC) curve was identified to assess the discriminative power of ASI to distinguish between the 'no CAD group' and the 'occult CAD group'. A p value $<$ 0.05 was considered as statistically significant.

RESULTS

The clinical characteristics and aortic elastic properties of the no CAD, occult CAD, and evident CAD groups are set out in Table 1. The evident CAD group had significantly lower HDL level than the occult CAD group ($p = 0.03$) and the no

CAD group ($p = 0.07$). The remaining clinical and laboratory parameters did not show significant differences between groups. ASI was significantly higher in the occult CAD group than in the no CAD group ($p = 0.008$), and significantly lower than in the evident CAD group ($p = 0.01$) (Fig. 3). Aortic distensibility also differed significantly in the occult CAD group compared to the no CAD group ($p = 0.01$) and the evident CAD group ($p = 0.02$) (Fig. 4). Similarly, aortic strain was more significantly reduced in the occult CAD group than in the no CAD group ($p = 0.03$) and was significantly greater than in the evident CAD group ($p = 0.02$). These three parameters were also significantly different between the no CAD group and the evident CAD group ($p < 0.001$, all). ASI was significantly correlated with age ($r = 0.40$, $p = 0.002$), DD ($r = 0.37$, $p = 0.003$), aortic distensibility ($r = -0.72$, $p < 0.001$), aortic strain ($r = -0.66$, $p < 0.001$) and the presence of hypertension ($r = 0.34$, $p = 0.04$). ROC analysis for ASI to discriminate between the occult CAD group and the no CAD group, revealed an area under the curve of 0.80 (confidence interval 0.68–0.94, $p = 0.004$) and that the cut-off value of 3.42 could significantly predict occult CAD, with 78% sensitivity and 63% specificity (Fig. 5).

DISCUSSION

The main finding of this study was the significantly higher ASI and significantly reduced aortic distensibility and aortic strain

Table 1. Clinical characteristics and aortic elastic properties of the study population

	No CAD group (n = 23)	Occult CAD group (n = 17)	Evident CAD group (n = 19)	P*	P [^]	P ⁺
Age [years]	46.1 \pm 8.4	48.9 \pm 10.1	52.5 \pm 6.6	0.59	0.12	0.35
Male gender	11 (47.8%)	12 (70.5%)	16 (84.2%)	0.45	0.33	0.25
Systolic BP [mm Hg]	131 \pm 14.9	132 \pm 14.6	129 \pm 10.3	0.81	0.56	0.41
Diastolic BP [mm Hg]	100 \pm 83.4	110 \pm 84.1	100 \pm 87.6	0.83	0.10	0.18
Hypertension	8 (34.7%)	5 (29.4%)	6 (31.5%)	0.72	0.82	0.88
Family history	18 (78.2%)	10 (58.8%)	11 (57.8%)	0.19	0.16	0.95
Diabetes mellitus	4 (17.3%)	4 (23.5%)	8 (42.1%)	0.63	0.18	0.24
Glucose [mg/dL]	100 \pm 28	111 \pm 41	126 \pm 51	0.41	0.06	0.35
Dyslipidaemia	10 (43.4%)	7 (41.1%)	13 (68.4%)	0.88	0.11	0.10
Total cholesterol [mg/dL]	191.5 \pm 27.7	193.0 \pm 42.5	194.3 \pm 45.5	0.89	0.80	0.93
LDL [mg/dL]	111.6 \pm 29.2	117.2 \pm 26.9	124.8 \pm 42.1	0.78	0.64	0.52
HDL [mg/dL]	47.9 \pm 15.8	43.9 \pm 11.6	36.7 \pm 6.9	0.38	0.007	0.03
Triglyceride [mg/dL]	117 \pm 56	162 \pm 99	174 \pm 114	0.27	0.19	0.85
Smoking	9 (39.1%)	8 (47.0%)	13 (68.4%)	0.62	0.14	0.20
Body mass index [kg/m ²]	27.8 \pm 3.8	27.0 \pm 3.9	27.5 \pm 2.7	0.54	0.79	0.66
Aortic stiffness index (β)	3.28 \pm 0.76	4.43 \pm 1.76	6.64 \pm 3.28	0.008	$<$ 0.001	0.01
Aortic distensibility [cm ² /dyn]	9.63 \pm 3.50	7.27 \pm 4.72	4.36 \pm 2.17	0.01	$<$ 0.001	0.02
Aortic strain [%]	22.4 \pm 6.7	16.7 \pm 9.5	8.6 \pm 3.9	0.03	$<$ 0.001	0.02

*p value for no CAD vs. occult CAD; [^]p value for no CAD vs. evident CAD; ⁺p value for occult CAD vs. evident CAD; BP — blood pressure; CAD — coronary artery disease; LDL — low density lipoprotein; HDL — high density lipoprotein

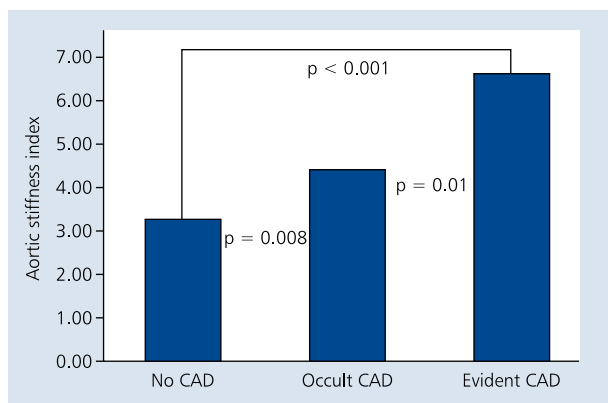


Figure 3. Average aortic stiffness index values of the study population; CAD — coronary artery disease

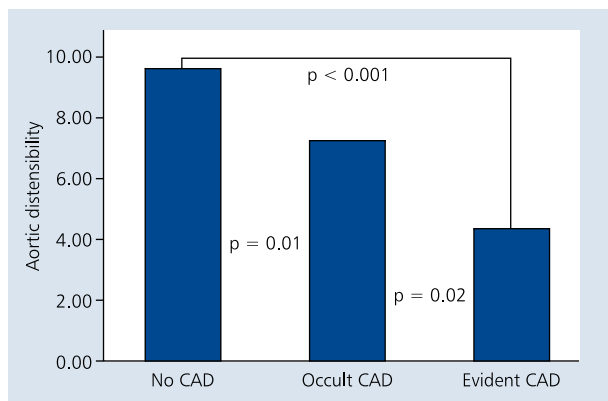


Figure 4. Average aortic distensibility values of the study population; CAD — coronary artery disease

in the occult CAD group compared to the no CAD group. ASI was the best among these parameters in discriminating between the occult CAD and the no CAD groups, and a cut-off value of 3.42 significantly predicted the subclinical atherosclerotic plaques with moderate sensitivity and specificity. Further, the patients with angiographically proven CAD (evident CAD) had significantly worse aortic elastic properties compared to those with occult CAD.

Ageing of the arterial system is accompanied by structural changes, including fragmentation and degeneration of elastin, increase in collagen, thickening of the arterial wall, and vascular stiffening [20]. Several mechanisms may explain the association between increased arterial stiffness and CV mortality. The elevation of SBP, which raises LV afterload and myocardial work, and the decrease in DBP, which reduces coronary perfusion, result in subendocardial ischaemia [7]. Arterial stiffness is also associated with LV hypertrophy in normotensive and hypertensive patients. LV hypertrophy is a known risk factor for congestive heart failure and CV events [21]. As plenty of studies have also shown, arterial stiffness is correlated with atherosclerosis [3–6], probably through

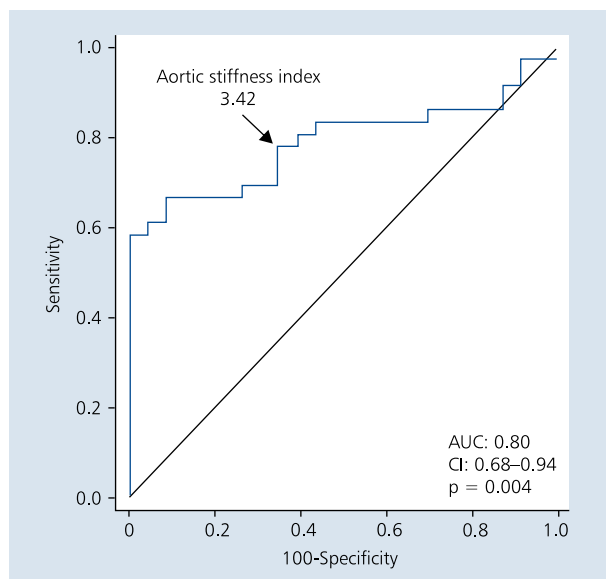


Figure 5. The receiver operating characteristics curve to assess discriminative power of aortic stiffness index for the distinction between occult coronary artery disease and no coronary artery disease group. Cut-off value was found to be 3.42 with 78% sensitivity and 63% specificity; AUC — area under the curve; CI — confidence interval

the effects of cyclic stress on arterial wall thickening [22]. However, to the best of our knowledge, this is the first study to determine the relation of aortic stiffness with subclinical atherosclerosis detected by MDCT.

It has been well established that the presence of coronary stenosis detected by conventional angiography is associated with adverse events, depending on their location, number and severity. However, vulnerable nonstenotic plaques, because of the compensatory expansion of the coronary arteries (positive remodelling), may not be seen in catheter angiography, and rupture of these plaques is generally accepted to be the major cause of myocardial infarction. The visualisation of these atherosclerotic plaques is troublesome but may be of importance for the early detection of occult CAD.

IVUS remains the standard imaging modality for plaque detection [23]. In recent years, MDCT has emerged as a rapid and noninvasive alternative, with high sensitivity and specificity, in determining calcified and noncalcified plaques compatible with IVUS [18, 19].

However, contrast-associated problems, high cost, inapplicability at bedside and exposure to radiation limit the widespread use of MDCT. Echocardiography is an extremely safe, widely available, easily applicable and less expensive imaging modality providing information about aortic stiffness without exposing patients to ionising radiation such as MDCT. One of the simplest ways to calculate aortic stiffness is based on the measurements of aortic dimensions and PP. The PP is simply the difference between SBP and DBP, and depends on

the cardiac output, large-artery stiffness and wave reflection. It can be easily measured using a standard sphygmomanometer. Ascending aorta diameters can also be obtained from the parasternal long axis view on the M-mode tracing at a level 3 cm above the aortic valve [24, 25]. We used ASI, aortic strain and aortic distensibility, calculated from aortic diameters and PP.

The findings of our study suggest these three parameters to determine high risk patients for CAD, and to prevent the unnecessary use of MDCT.

Atherosclerosis is a chronic and multifactorial disease that affects the whole arterial system. Therefore, atherosclerotic changes in any part of this arterial system give clues about the other parts. As the extension and severity of CAD increase, stiffness of the aorta decreases. Parameters such as ASI, aortic distensibility and aortic strain in determining the aortic stiffness were used in previous studies and their relationship with CAD has been demonstrated [12–14]. Hirai et al. [8] showed that these parameters are more elevated in patients with more extensive CAD, indicating their relationship with atherosclerosis progression. Recently, a study by Durmaz et al. [25] showed that aortic strain and distensibility were significantly lower in patients with coronary artery calcium by MDCT than those without. They studied subjects with obstructive CAD.

However, in this study we demonstrated significantly altered aortic stiffness parameters (ASI, aortic strain and distensibility) in patients with occult CAD detected by MDCT although their conventional coronary angiography revealed normal coronary arteries. Moreover, significantly worse aortic elastic properties were also determined in patients with angiographically proven CAD than in patients with occult CAD. It is well known that aortic elastic properties are affected by the risk factors for ischaemic heart disease, such as age, hypertension, hypercholesterolaemia, and smoking [7, 11]. Consistent with previous studies, we found significant correlations with age and the presence of hypertension in this study.

The clinical implications of our study include the role of aortic elastic properties to identify patients with coronary plaques, even if their conventional angiography showed normal coronary arteries. ASI was found to be the best aortic stiffness parameter in the prediction of subclinical atherosclerosis. Since the use of ASI is safer given its non-radiographic nature, it may be helpful for the selection of high risk individuals for CAD prior to MDCT, instead of routine clinical use of MDCT. Moreover, we suggest that patients with a value of ASI > 3.42 might be considered for preventive strategies (such as aspirin, statins, lifestyle changes) to decelerate the progression of atherosclerosis. However, to explore the utility of this parameter in managing CV risk, our data should be supported by further studies with a larger sample size and outcome data.

Limitations of the study

Small sample size and lack of prospective follow-up data for CV events were the major limitations of the study. Presence

of atherosclerotic plaque does not necessarily indicate that the patient is at risk for adverse events. Even if the MDCT had revealed some calcifications, these patients might have an excellent prognosis. Large scale follow-up studies might clarify the relation between aortic elastic properties and adverse CV events in patients with occult CAD. IVUS is superior to MDCT in detecting coronary plaques. As we did not perform IVUS in this study, we may have substantially underestimated plaque burden by MDCT. The lack of aortic pulse wave velocity in our analysis which is a functional parameter of aortic stiffness was another limitation of our study.

CONCLUSIONS

Atherosclerotic coronary plaques may be present even in patients with normal coronary arteries by conventional angiography. MDCT is an attractive tool to detect these occult coronary plaques. However, contrast-associated problems, high cost, inapplicability at bedside and exposure to radiation limit the widespread use of MDCT. Measurement of ASI is an easily applicable and safe method with its non-radiographic ability for the assessment of aortic stiffness, and it may be useful to predict subclinical atherosclerosis in clinical practice. A cut-off value of 3.42 for ASI may serve as a guide to refer individuals to preventive strategies to reduce atherosclerosis progression.

Conflict of interest: none declared

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Streszczenie

Wstęp: Wielodetektorowa tomografia komputerowa (MDCT) jest atrakcyjną nieinwazyjną techniką obrazowania stosowaną w celu wykrycia w naczyniach wieńcowych zmian miażdżycowych, które mogą być niedoszacowane w konwencjonalnej koronarografii. Jednak rutynowe stosowanie tej metody w praktyce klinicznej jest ograniczone z powodu problemów związanych z kontrastem, wysokich kosztów, niemożności zastosowania przyłóżkowego i ekspozycji na promieniowanie. Konieczne są więc badania nad tańszymi i praktyczniejszymi technikami umożliwiającymi prognozowanie bezobjawowej choroby wieńcowej (CAD).

Cel: Badanie przeprowadzono w celu wykazania wartości prognostycznej własności elastycznych aorty w odniesieniu do bezobjawowej CAD rozpoznawanej na podstawie MDCT.

Metody: Do badania włączono 40 kolejnych pacjentów z prawidłowym obrazem tętnic wieńcowych w badaniu angiograficznym. Poddano ich MDCT i podzielono na dwie grupy: bez CAD (23 osoby, 11 mężczyzn, 46 ± 8 lat) i z bezobjawową CAD (17 osób, 12 mężczyzn, 48 ± 10 lat) w zależności od obecności blaszek miażdżycowych w tętnicach wieńcowych. Grupę kontrolną stanowiło 19 kolejnych pacjentów z potwierdzoną w badaniu angiograficznym CAD (16 mężczyzn, 52 ± 6 lat). Wskaźnik sztywności aorty (ASI), rozciągliwość i odkształcenie ściany aorty obliczono na podstawie wymiaru aorty (zmierzonego echokardiograficznie) i ciśnienia krwi (zmierzonego sfigmomanometrem).

Wyniki: Stwierdzono, że ASI, rozciągliwość i odkształcenie ściany aorty były istotnie różne w grupie z bezobjawową CAD w porównaniu z grupą bez CAD (odpowiednio $p = 0,008$; $p = 0,01$; $p = 0,03$) i z potwierdzoną CAD ($p = 0,01$; $p = 0,02$; $p = 0,02$). Parametry te różniły się również istotnie między grupą bez CAD i grupą z potwierdzoną CAD. W analizie krzywych ROC dla ASI, mającej na celu wykazanie różnic między grupą z bezobjawową CAD a grupą bez CAD, stwierdzono, że pole pod krzywą wynosi 0,80 (CI 0,68–0,94; $p = 0,004$) i że wartość progowa równa 3,42 umożliwia prognozowanie występowanie u pacjentów bezobjawowej CAD (czułość: 78%; swoistość: 63%).

Wnioski: Pomiar ASI jest łatwą i bezpieczną metodą nieradiograficznej oceny sztywności aorty, która może być przydatna do prognozowania podklinicznej miażdżycy w praktyce klinicznej. Wartość progowa ASI może stanowić wskazówkę, że należy zastosować u danego pacjenta strategię prewencyjną w celu ograniczenia progresji miażdżycy.

Słowa kluczowe: choroba wieńcowa, tomografia komputerowa, miażdżycy, angiografia

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