

Thoracic aortic plaques, transoesophageal echocardiography and coronary artery disease

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Summary

Background: The purpose of this study was to assess whether the detection of atherosclerotic aortic plaques by transoesophageal echocardiography (TEE) could be used as a marker of coronary artery disease (CAD), relying on their number, cross-sectional surface, depth and localisation.

Methods: The thoracic aortas of 102 consecutive patients (77 men, mean age 67 ± 12 years) undergoing elective cardiac surgery were assessed by TEE. Atherosclerotic plaques were defined as ≥ 5 mm thick focal hyperechogenic zones of the aortic intima and/or lumen irregularities with mobile structures or ulcerations. All patients had undergone prior coronary angiography.

Results: Thoracic aortic plaques were present in 73 patients, 66 of whom had CAD. The presence of aortic plaques detected by TEE identified significant coronary artery disease with a sensitivity of 90% and a specificity of 76%. The maximum

transverse cross-sectional plaque area, the maximum plaque depth and the total plaque number all correlated significantly with the presence of CAD, but not with its severity.

Multivariate regression analysis showed that aortic plaques, hypertension and hypercholesterolaemia were significant predictors of CAD, but aortic plaques were the most significant predictor regardless of age and sex.

Conclusions: This study suggests that detection of atherosclerotic aortic plaques is a useful marker of significant coronary artery disease. Absence of plaques in the patients aged over 70 identified a subgroup with a very low probability of CAD.

Key words: atherosclerosis; thoracic aorta; aortic plaque; coronary artery disease; transoesophageal echocardiography

Introduction

Transoesophageal echocardiography (TEE) allows detection of atherosclerotic intimal lesions in the thoracic aorta [1-3]. Previous studies have shown that the presence of atherosclerotic aortic plaques is a strong independent predictor of coronary artery disease and of embolic events [4-12]. When combined with clinical findings, the visualisation of thoracic aortic plaques may warrant selective use of coronary angiography prior to valvular surgery [11]. In addition, the high potential risk of embolism posed by complex aortic plaques can be successfully diminished by using targeted approaches before cardiac surgery and invasive coronary procedures [13-15].

Previous reports, however, have relied mainly

on qualitative information, based on the presence or absence of simple or complex aortic plaques. Quantitative characteristics and distribution of thoracic aortic plaques imaged by TEE and its predictive value for coronary artery disease (CAD) are less well defined. Some authors have reported that the majority of aortic plaques are located in the descending thoracic aorta and the aortic arch, and that few or none are found in the ascending thoracic aorta [2, 16].

The present study was designed to assess whether the number, distribution and extent of thoracic aortic plaques measured by TEE can be used as a marker of CAD.

Materials and methods

Population

102 patients (77 men, 25 women, mean age 67 ± 12 years) were assessed prospectively by TEE during cardiac surgery. All had undergone prior coronary angiography. 73 patients underwent coronary bypass, 25 valve surgery and 4 combined procedures.

Transoesophageal echocardiography examination (TEE)

After induction of general anaesthesia, preoperative TEE was performed using a commercial ultrasonograph (Hewlett-Packard, Sonos 2500, Andover, MA) and a multiplane 5 MHz transoesophageal transducer. Following a standard cardiac examination, the probe was rotated posteriorly in the stomach. During pullback the aorta was scanned from the abdomen to the aortic arch in the transverse short-axis plane. Subsequently the ascending aorta was assessed by pullback from the aortic valve.

All studies were recorded on videotape for subsequent analysis. The recordings were first interpreted by the echocardiographer and then reviewed by a senior cardiologist unaware of the clinical and angiographic data. There was disagreement on the grading of intimal thickness in three cases out of 102.

Analysis of the TEE studies

The degree of aortic intimal changes was measured and graded using a modification of Ribakove's scoring system [17]. The following criteria were adopted: grade 0 = normal intima; grade I = minimal intimal thickening with increased echo density but smooth and continuous intimal surface; grade II = intimal thickening <5 mm with lumen irregularities and highly echogenic areas disrupting intimal surface; grade III = intimal thickening ≥ 5 mm with

complex, highly echo-dense material protruding into the lumen, presence of mobile protruding lesions (aortic debris) and/or ulceration. An aortic plaque was defined as a localised grade II or grade III lesion.

Quantitative measurement of aortic plaques

For each patient the total number of plaques was counted. On the short axis view, thickness and cross sectional surface were measured for each plaque. The ascending aorta, the aortic arch and the descending thoracic aorta were analysed separately.

Coronary angiography

Selective coronary arteriography had been performed prior to surgery. A senior angiographer interpreted the angiograms. Coronary artery disease was diagnosed when at least one stenosis exceeded 70%, or $>50\%$ in the left main coronary artery. For the purpose of analysis a significant lesion of the left main coronary artery was considered equivalent to two-vessel disease.

Statistical analysis

Sensitivity, specificity, and positive and negative predictive values were computed by 2×2 contingency table for the angiographic and echocardiographic data. χ^2 test was used to compare categorical variables and a two-tailed t test was used to compare continuous variables as an initial step. A p value <0.05 was considered statistically significant. In a second step, to identify independent variables predictive of CAD, variables found to have a significant association with CAD in the univariate analysis were entered into a multivariate stepwise logistic regression model with CAD as the dependent variable.

Results

Baseline characteristics

78 patients had at least one risk factor for cardiovascular disease (Table 1).

Coronary angiography detected significant coronary artery disease in 73 patients. 3-vessel disease was found in 44 patients, 2-vessel disease in 21 and 1-vessel disease in 8. Non-significant lesions were found in 6 patients and in 23 patients the coronary arteries were considered normal.

The aortic wall, assessed by TEE, was normal in 13 patients and exhibited grade I lesions in 16 patients, grade II lesions in 34 and grade III lesions in 39. Plaque distribution is shown in Table 2.

Correlation between aortic plaques and coronary artery disease

For the whole group, the odds on a patient with aortic plaques having CAD were 29.6 (CI: 9.3–93.9).

The presence and severity of CAD in relation to aortic plaque parameters are shown in Table 3. CAD patients had more aortic plaques, but no relationship was found between severity of CAD and various aortic plaque parameters.

The sensitivity of thoracic aortic plaques in detecting CAD was 90% in the whole group. Using an arbitrary cut-off of 70 years for age, we found that sensitivity was lower in young patients but as high as 100% in older patients (Table 4). In this group the very high sensitivity came at the cost of very low specificity (as shown in Table 4).

Aortic plaques and cardiovascular risk factors (CVRF)

Figure 1 shows the number of plaques in the aorta in relation to the number of risk factors for the whole population. The slope of the regression differs significantly from zero ($p = 0.0027$), showing a significant trend with more plaques in pa-

Table 1
Cardiovascular risk factors in patients.

	n	%
Total	102	100
BMI >30	23	23
Hypertension	49	48
Hypercholesterolaemia	42	41
Smoking	30	29
Diabetes mellitus	16	16
Family history	16	16

n = number of patients

Table 2

Distribution and characteristics of aortic plaques in 73 patients with grade II and III lesions.

	Ascending aorta	Aortic arch	Descending aorta	All segments
Number of patients	3 (4%)	57 (78%)	67 (92%)	73
Mean number of plaques per patient	8.0 ± 4	5.9 ± 3.3	5.9 ± 3.1	5.3 ± 3.2
Mean transverse cross sectional area of largest plaque (mm ²)	47.7 ± 26.4	35.4 ± 17.9	34.4 ± 17.8	32.8 ± 18
Mean thickness of thickest plaque (mm)	4.5 ± 1.3	3.9 ± 1.1	4.1 ± 1.2	3.8 ± 1.3

Table 3

Quantitative aortic plaque parameters for patients with and without CAD.

	No CAD	CAD	1-vessel disease	2-vessel disease	3-vessel disease
Patient number	29	73	8	21	44
Patients with aortic plaques	7	66	7	17	42
Mean number of plaques	1.5 ± 2.9	4.8 ± 3.4*	7.1 ± 4.7	3.5 ± 2.9	5.0 ± 3.3
Transverse cross-sectional area of the largest plaque (mm ²)	7.4 ± 13.1	30.1 ± 20.5*	33.2 ± 30.5	31.4 ± 23.2	28.9 ± 17.2
Total plaque transverse cross-sectional area (mm ²)	22.4 ± 46.0	100.1 ± 90.7*	172.4 ± 180.2	78.9 ± 73.9	97.1 ± 69.0
Maximum plaque thickness (mm)	0.9 ± 1.8	3.5 ± 1.7*	3.3 ± 1.6	2.9 ± 1.7	3.7 ± 1.7
Number of plaques in					
descending aorta	1.0 ± 2.63	3.68 ± 2.95*	5.75 ± 3.45	2.67 ± 2.53	3.79 ± 2.89
ascending aorta	0 ± 0	0.04 ± 0.20 NS	0.12 ± 0.35	0 ± 0	0.04 ± 0.21
aortic arch	0.45 ± 0.78	1.07 ± 0.93**	1.25 ± 1.28	0.85 ± 1.01	1.14 ± 0.82

* p < 0.0001, ** p < 0.005 for CAD vs no CAD groups

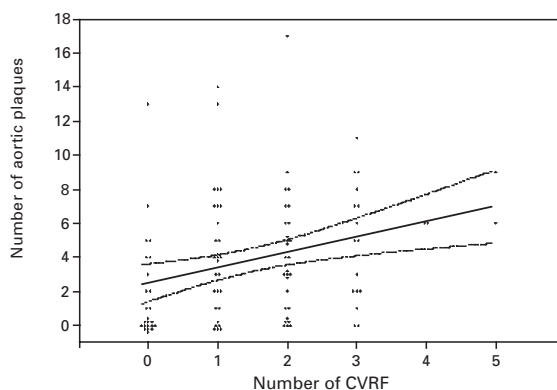
Table 4

Predictive value of aortic plaques for the presence of CAD.

	Total		<70 years		>70 years	
	No CAD	CAD	No CAD	CAD	No CAD	CAD
No aortic plaques	22	7	16	7	6	0
Aortic plaques	7	66	0	30	7	36
Sensitivity	90%		81%		100%	
Specificity	76%		100%		46%	
Positive predictive value	90%		100%		84%	
Negative predictive value	76%		70%		100%	

Figure 1

Number of plaques in the thoracic aorta in relation to the number of risk factors for the whole population. The regression line (solid line) is shown with the 95% confidence interval (dotted lines).



tients with more risk factors, although a wide scatter is observed.

By univariate analysis, various plaque characteristics and CVRF correlated significantly with CAD. By stepwise logistic regression analysis, three parameters were found to be independent predictors of CAD: presence of AP (p < 0.0001), hypercholesterolaemia (p < 0.0007) and hypertension (p < 0.0004).

Discussion

In our study, thoracic aortic plaques detected by TEE were a marker of the presence of CAD, thus confirming previous reports. Sensitivity and specificity for detection of CAD were similar to the findings of Tribouilloy and Fazio [6, 11]. Moreover, the present study adds new information on

the value of aortic plaque detection in different age groups and on the distribution and morphology of plaques.

There is disagreement on the value of aortic plaques as a marker of CAD in the elderly [9]. With advancing age specificity decreases and sensitivity

increases [18]. We also found aortic plaques in the majority of patients over 70, and specificity is therefore low. However, absence of aortic plaques was an excellent marker for absence of significant CAD (negative predictive value 100%). Hence echographic imaging of the aorta may be of use in identifying patients at low risk of CAD in this category of patients. Conversely, the absence of aortic plaques does not exclude CAD in the under-70s.

Our study suggests that not only the presence of aortic plaques, but also their number, maximal thickness, and cross-sectional surface are strong predictors of CAD.

As reported in other studies, plaque distribution is uneven in the thoracic aorta [9–11]. The ascending aorta has the fewest plaques, the transverse arch has an intermediate number and the descending aorta the highest number. A low prevalence of atherosclerosis in the ascending aorta and the absence of correlation between ascending aortic plaques and CAD have been reported previously [16].

In previous studies conventional coronary risk factors were significantly weaker predictors of CAD than were aortic plaques detected by TEE, as confirmed by our findings [6, 10, 11, 19, 20].

Several limitations of the study must be emphasised. First, only patients undergoing cardiac surgery were included. The findings may differ in selected pop-

ulations. Second, we have no follow-up data on our patients and no data on the risk of later coronary events depending on the presence of aortic plaques. Finally, even though we used a multiplane transducer, the number of aortic plaques in the ascending aorta and proximal arch was certainly underestimated, since the segment of the aorta from the level of the crossing with the right pulmonary artery to the level of the left carotid artery departure frequently remained hidden.

In conclusion, the present study suggests that identification of aortic plaques in the aorta could be a very useful non-invasive marker for coronary artery disease and their absence could be used to identify patients over 70 with a very low probability of CAD. Measuring the transverse cross-sectional area, number, maximum thickness and distribution of plaques along the thoracic aorta does not significantly add diagnostic information on the presence or severity of CAD.

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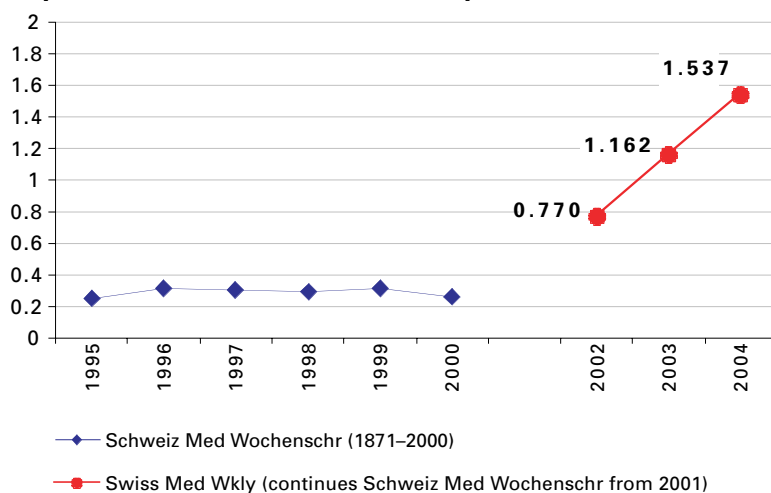
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