

Mitral and aortic annular calcifications and cerebrovascular ischemic episodes in patients with coronary artery disease

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KEY WORDS

aortic calcifications,
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ABSTRACT

INTRODUCTION Atherosclerosis is a systemic pathological process involving the whole arterial bed. Valvular calcifications are associated with cardiovascular risk factors. Significant carotid stenosis accounts for approximately 20% of cerebrovascular ischemic episodes.

OBJECTIVES The aim of the study was to assess the relationship between mitral and aortic annular calcifications, increased carotid intima-media thickness (CIMT), and incidence of cerebrovascular ischemic episodes.

PATIENTS AND METHODS A total of 127 patients with angiographically confirmed coronary artery disease (68 men and 59 women; aged 74 (33–87) years) were divided into 4 groups: with aortic valve calcifications (AVCs), mitral annular calcifications (MACs), both aortic valve and mitral annular calcifications (AMVCs), and no valvular calcifications (no-VCs), based on the echocardiographic assessment of the mitral and aortic valves. CIMT and the presence of atherosclerotic plaques were evaluated by carotid ultrasonography. A history of cerebrovascular ischemic episodes was obtained.

RESULTS The combined prevalence of mitral or aortic valve calcifications in the study population was 59% (AVCs, 55%; MACs, 24%; and AMVCs, 21%). CIMT was significantly increased in the MAC and AMVC groups ($P < 0.05$ for MACs; $P < 0.01$ for AMVCs). Ischemic stroke was more common in the AVC group ($P < 0.05$), while the MAC group had a higher incidence of carotid plaques ($P < 0.05$), transient ischemic attacks (TIA; $P < 0.05$), and strokes ($P < 0.05$) as compared with the no-VC group. In multivariate analysis, only MACs remained independently associated with increased CIMT.

CONCLUSIONS In patients with coronary artery disease, MACs are independently associated with increased CIMT but not with TIA or stroke. There is no relationship between the concomitant presence of mitral and aortic calcifications and carotid atherosclerosis.

INTRODUCTION Atherosclerosis is a systemic pathological process involving the entire arterial bed, and the presence of atherosclerosis in 1 artery increases the risk of its presence in another vascular location.¹ However, there are some sites such as the bifurcation of the common carotid artery or distal abdominal aorta with a predisposition to atherosclerosis owing to abnormal shear stress or turbulent blood flow.² Attachment points of the aortic and mitral valves to their annuli are the sites of turbulent blood flow; thus, they are also at increased risk of atherosclerosis.² There are a number of similarities between

the atherosclerotic plaque formation and early valve lesions. Both processes seem to be initiated by endothelial injury due to increased mechanical stress, followed by extracellular lipid accumulation in the subendothelial region augmented by the presence of oxidized low-density lipoproteins (LDLs), macrophages and other inflammatory cells, and matrix metalloproteinase, with an additional contribution from the activation of the renin-angiotensin-aldosterone system.³ Furthermore, both processes are precipitated by exposure to the same cardiovascular risk factors.⁴ Thus, there have been several studies conducted

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to support the hypothesis that annular calcification is atherosclerotic in nature.² It has been suggested that valvular calcification and atherosclerosis are associated only at the early stages, and that the potential link may be linked to abnormal serum osteoprotegerin levels.⁵ However, there are substantial differences between the 2 diseases, including the absence of smooth muscle cells and the accumulation of larger amounts of calcium and proteins during valvular calcification compared with atherosclerosis.⁶ Moreover, it is not clear why lipid-lowering therapy failed in the studies that were performed to prevent or even slow down the progression of valvular disease.⁷⁻⁹ This observation is in contrast to the hypothesis of the common etiology and pathophysiology of those processes.

Mitral annular calcifications (MACs) and aortic valve calcifications (AVCs), common findings on routine echocardiography, are reported to be associated with numerous cardiovascular risk factors and are considered minor cardioembolic sources, although no causal relationship with stroke has been confirmed so far.

An association between MACs and AVCs and increased carotid intima-media thickness (CIMT) and carotid atherosclerosis has been reported.^{10,11} Several studies have suggested a relationship between MACs and stroke, but an independent association between AVCs and cerebral infarction has been demonstrated solely in the presence of aortic valve stenosis.³ Moreover, to our knowledge, there has been no study evaluating the concomitant presence of valvular calcifications in relation to cerebrovascular episodes or cardiovascular risk factors and disease.

The aim of this study was to further investigate whether there is a relationship between mitral and aortic annular calcifications, increased CIMT, and incidence of cerebrovascular ischemic episodes in patients with documented coronary atherosclerosis.

PATIENTS AND METHODS A group of 127 patients with angiographically confirmed coronary artery disease including stable coronary artery disease and acute myocardial syndromes (68 men and 59 women) and without aortic or mitral valve stenosis was divided into 4 subgroups based on echocardiography: patients with AVCs; patients with MACs; patients with both aortic valve and mitral annular calcifications (AMVCs); and patients with no valvular calcifications (no-VCs).

Standard transthoracic echocardiography was performed with Acuson Sequoia C512 (Siemens Healthcare, Erlangen, Germany). The parasternal long-axis, apical 4-chamber, 2-chamber, and long-axis projections were obtained. Left atrial diameter and left ventricular end-diastolic and end-systolic diameters were measured. Hemodynamic parameters were obtained with pulsed-wave and continuous-wave Doppler ultrasound.

MACs were defined based on the presence of the following features on cross-sectional

echocardiography in parasternal windows: increased focal echogenicity at the base of the posterior leaflet while visualized in parasternal long- and short-axis views. AVCs were determined by irregular cusp thickening and focal hyperechogenicity at their bases in respective projections. Leaflet motion was not impaired in any of the subjects with MACs or AVCs, i.e., there was no turbulence in color Doppler nor increased continuous-wave Doppler gradients and the valve commissures were not fused.^{3,11}

CIMT and carotid atherosclerotic lesions were evaluated by ultrasonography of the carotid arteries (Siemens Sequoia 512CE). B-mode carotid ultrasonography was performed with a linear-array transducer. It was manipulated so that the walls of the common carotid artery were parallel to the transducer. A region of 1.5 cm proximal to the carotid bifurcation was identified, and the CIMT of the posterior wall was evaluated as the distance between the luminal-intimal and medial-adventitial interface. The average of the 5 measurements was used for analyses.

Increased CIMT was defined as CIMT exceeding 0.9 mm, while focal CIMT exceeding 1.5 mm was considered as carotid plaque.¹²

Several atherosclerosis risk factors were assessed on the basis of medical history and laboratory tests, including blood pressure measurement, assessment of the lipid profile and renal function, and collection of data on diabetes, obesity, and smoking.

Hypertension was defined on the basis of the 2013 European Society of Hypertension / European Society of Cardiology (ESC) Guidelines for the management of arterial hypertension,¹³ when either the mean of at least 2 blood pressure measurements was consistently not lower than 140 mmHg of systolic or 90 mmHg of diastolic blood pressure over at least 2 separate days or when antihypertensive therapy was used.

A lipid-lowering therapy or an LDL cholesterol level of 1.8 mmol/l (70 mg/dl) and higher was regarded as diagnostic for hypercholesterolemia, while triglyceride levels of 1.7 mmol/l (150 mg/dl) and higher, for hypertriglyceridemia.¹⁴ Renal function impairment was assessed on the basis of a decreased glomerular filtration rate (eGFR) of less than 60 ml/min/1.73 m² calculated by the Modification of Diet in Renal Disease formula.¹⁵

Diabetes was diagnosed on the basis of the 2013 ESC guidelines.¹⁶ In brief, fasting glycemia equal to or exceeding 7.0 mmol/l (126 mg/dl) on 2 measurements, or 11.0 mmol/l (200 mg/dl) in 1 random blood sample, or the use of hypoglycemic drugs was considered diagnostic. Obesity was diagnosed if the patient's body mass index (BMI) was 30 kg/m² and higher.¹⁴

Data on the smoking status was collected and a history of cerebrovascular ischemic episodes was obtained. Ischemic stroke was defined as an episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction based on abnormalities on computed tomography or

TABLE 1 Characteristics of the study groups

	AVCs (n = 41)	MACs (n = 18)	AMVCs (n = 16)	no-VCs (n = 52)	P (between marked groups)
men/women, %	66/34	50/50	50/50	48/52	0.5
age, y	74 (47–87)	72 (52–81)	76 (60–83)	68 (33–84)	0.1
BMI, kg/m ²	27.95 ± 4.32	26.59 ± 2.62	30.25 ± 3.13	29.47 ± 5.84	0.07 ^a
diabetes	18 (44)	8 (44)	7 (47)	14 (27)	0.2
hypertension	35 (85)	11 (61)	11 (73)	41 (80)	0.7
hypercholesterolemia	28 (68)	12 (67)	9 (60)	36 (71)	0.9
hypertriglyceridemia	10 (24)	1 (5)	4 (27)	15 (29)	0.2
smoking	10 (24)	2 (11)	2 (13)	17 (33)	0.2
CKD, eGFR <60 ml/min/1.73 m ²	14 (34)	8 (44)	8 (53)	15 (29)	0.5
1-vessel disease	7 (17)	3 (17)	3 (19)	8 (15)	1
2-vessel disease	1 (2)	1 (5)	3 (19)	4 (8)	0.2
3-vessel disease	18 (44)	7 (39)	5 (31)	16 (31)	0.6
stable CAD	20 (49)	9 (50)	8 (50)	28 (54)	0.9
ACS	15 (36)	5 (28)	3 (19)	10 (19)	0.4
unstable angina	9 (22)	3 (17)	1 (6)	4 (8)	0.2
NSTEMI	6 (15)	2 (11)	1 (6)	3 (6)	0.5
STEMI	0 (0)	0 (0)	1 (6)	3 (6)	0.3
creatinine, μmol/l	94.14 ± 26.08	89.80 ± 17.56	102.61 ± 34.23	81.51 ± 20.65	0.01 ^b ; 0.04 ^c
eGFR, ml/min/1.73 m ²	66.86 ± 16.99	66.97 ± 15.55	60.80 ± 22.39	73.47 ± 15.32	0.1
glucose, mmol/l	6.00 ± 1.22	5.95 ± 0.50	8.87 ± 4.02	6.40 ± 2.11	0.2
TC, mmol/l	4.47 ± 0.74	4.09 ± 2.12	4.29 ± 0.70	4.33 ± 0.99	0.9
LDL-C, mmol/l	2.58 ± 0.72	2.44 ± 1.62	2.48 ± 0.44	2.51 ± 0.78	0.9
HDL-C, mmol/l	1.31 ± 0.34	1.29 ± 0.62	1.43 ± 0.33	1.35 ± 0.38	0.9
TG, mmol/l	1.92 ± 1.00	0.96 ± 0.10	1.31 ± 0.48	1.22 ± 0.40	0.3
ASA	35 (85)	16 (88)	13 (87)	45 (88)	0.4
clopidogrel	21 (51)	5 (28)	5 (31)	14 (27)	0.2
statins	33 (80)	15 (83)	10 (62)	38 (73)	0.5
β-blockers	28 (68)	13 (72)	11 (69)	40 (77)	0.6
ACEIs	27 (66)	12 (67)	7 (44)	26 (50)	0.3
ARBs	9 (22)	1 (5)	4 (25)	14 (27)	0.3
calcium antagonists	12 (29)	6 (33)	7 (44)	23 (44)	0.3

Data are expressed as mean ± standard deviation, median (interquartile range), or number of patients (percentage).

a MACs vs. AMVCs **b** AVCs vs. no-VCs **c** MACs vs. no-VCs

Abbreviations: ACEI – angiotensin-converting enzyme inhibitor, ACS – acute cardiac syndrome, AMVCs – aortic valve and mitral annular calcifications, ARB – angiotensin receptor blocker, ASA – acetylsalicylic acid, AVCs – aortic valve calcifications, BMI – body mass index, CAD – coronary artery disease, CKD – chronic kidney disease, eGFR – estimated glomerular filtration rate, HDL-C – high-density lipoprotein cholesterol, LDL-C – low-density lipoprotein cholesterol, MACs – mitral valve calcifications, no-VC – no valvular calcifications, NSTEMI – non-ST-segment elevation myocardial infarction, STEMI – ST-segment elevation myocardial infarction, TC – total cholesterol, TG – triglycerides

magnetic resonance imaging or other objective evidence in a defined vascular distribution or clinical evidence of such injury based on symptoms persisting at least for 24 h or until death.¹⁷

TIA was defined according to the American Stroke Association / American Heart Association consensus as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction.¹⁷

The exclusion criteria were as follows: atrial fibrillation, prosthetic valve, aortic or mitral stenosis, hemorrhagic cerebral episodes, past non-cerebrovascular thromboembolic events, and thrombophilia.

Differences in variables between the groups were analyzed by the χ^2 test and *t* test, as appropriate. Logistic regression analysis was performed including demographic and clinical parameters (age, sex, CIMT, carotid plaques, TIA, and stroke). A *P* value of less than 0.05 was considered statistically significant.

RESULTS We studied 127 patients at a median age of 74 years (range, 33–87 years). Baseline demographic, clinical, and laboratory data are presented in **TABLE 1**. The groups did not differ in terms of age, sex, and most cardiovascular risk factors and medications. The combined prevalence

TABLE 2 Differences in clinical outcomes and ultrasound carotid parameters between the study groups

	AVCs	MACs	AMVCs	no-VCs	P value (vs. no-VCs)
increased CIMT, n (%)	17 (41)	13 (72)	12 (80)	18 (35)	0.01 ^a ; 0.005 ^b
carotid plaques, n (%)	32 (78)	16 (88)	10 (67)	33 (65)	0.04 ^a
stroke, n (%)	9 (22)	4 (22)	1 (7)	3 (6)	0.05 ^a ; 0.02 ^c
TIA, n (%)	3 (7)	3 (17)	1 (7)	1 (2)	0.02 ^a

Data are presented as number of patients (percentage).

a MACs vs. no-VCs **b** AMVCs vs. no-VCs **c** AVCs vs. no-VCs

Abbreviations: CIMT – carotid intima–media thickness, TIA – transient ischemic attack, others – see **TABLE 1**

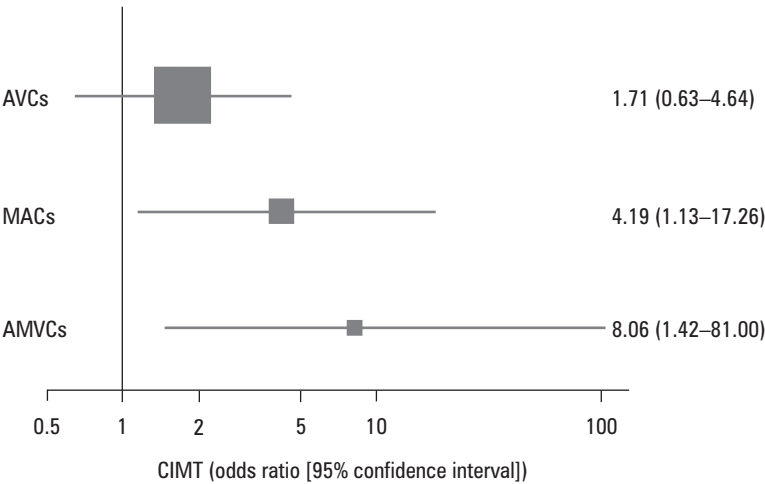


FIGURE 1 Association between valvular calcifications and carotid intima–media thickness (CIMT)

Abbreviations: see **TABLE 1**

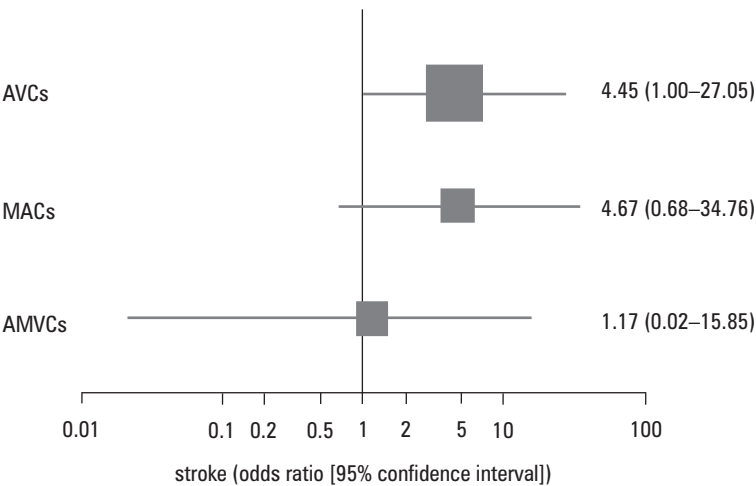


FIGURE 2 Association between valvular calcifications and stroke

Abbreviations: see **TABLE 1**

of MACs and AVCs in the study population was 59% (proportions of AVCs, MACs, and AMVCs were 55%, 24%, and 21%, respectively). Compared with the no-VC group, the MAC group had an increased incidence of CIMT, carotid plaques, TIA, and stroke. In the AVC group, CIMT was increased and stroke was more common compared with the

no-VC group. In patients with AMVCs, only increased CIMT was observed (**TABLE 2**).

Patients with increased CIMT were older (72.5 vs. 66.9 years; $P = 0.01$) but age was not associated with the incidence of carotid plaques, TIA, or stroke. Sex had no effect on clinical outcome and ultrasound carotid parameters (data not shown).

Univariate analysis showed an association between MACs and AMVCs and increased CIMT ($P = 0.001$), while aortic calcifications were associated with stroke ($P = 0.01$; **FIGURES 1** and **2**). Multivariate analysis with stepwise logistic regression showed that MACs were significantly associated with increased CIMT (odds ratio [OR], 4.06; 95% confidence interval [CI], 1.07–15.39; $P = 0.04$), while for AMVCs, the association was close to the level of statistical significance (OR, 5.22; 95% CI, 0.94–29.05; $P = 0.06$). No other parameters were independently associated with clinical and ultrasound variables.

DISCUSSION We demonstrated the relationship between mitral calcifications and increased CIMT in patients with angiographically confirmed coronary artery disease. Patients with periannular calcification of either mitral or aortic valves had increased CIMT. Moreover, MACs were associated with the occurrence of carotid plaques, TIA, and stroke, while stroke was more common in the AVC compared with the no-VC group.

Both MACs and AVCs were previously shown to be related to atherosclerosis of the various parts of the vascular bed, such as aortic atheromas and carotid and coronary artery disease.¹⁰ Moreover, MACs were shown to be a marker of generalized atherosclerotic disease, while AVCs were linked to a greater plaque burden and more calcified coronary lesions on computed tomography.¹⁸ Several studies have also suggested an association between MACs and stroke,^{19,20} but the relationship of AVCs with cerebral infarction has been demonstrated only in the presence of aortic valve stenosis.^{3,21} It has not been clarified whether there is a causal relationship between AMVCs and stroke or whether AMVCs are markers of an increased risk of stroke based on their association with atherosclerotic vascular disease. As noted above, both MACs and AVCs seem to be associated not only with carotid atherosclerosis but

also with aortic atherosclerotic plaques, especially complex lesions, which are a well-known risk factor for ischemic cerebral events.^{10,11,21-23} Moreover, carotid atherosclerosis has been reported to be associated with a higher prevalence of atherosclerotic plaques in the aortic arch; therefore, it constitutes an additional potential source of cerebral embolization.²⁴ However, our results show that only MACs are independent predictors of carotid wall thickening. We did not confirm the association between MACs or AVCs and cerebrovascular episodes.

There are some arguments to support the hypothesis of the cause and effect relationship between MAVCs and stroke.^{20,25,26} Mitral annular calcification has been suggested as a possible source of cerebral embolism in the Framingham Study independently of traditional risk factors for stroke, even of atrial fibrillation²⁷; however, MAC found on echocardiography is not considered to be an indication for treatment with anticoagulants.²⁸ A number of case reports have provided evidence for brain ischemic lesions, overt or clinically silent, resulting from calcific emboli from the aortic valve.²⁹⁻³³ Rodriguez et al.³⁴ demonstrated an association between mitral and aortic annular or valvular calcifications and asymptomatic brain infarcts. They showed that embolization from the left-sided cardiac annular or valvular calcifications can vary from friable calcium deposits to noncalcified (thrombotic) material. However, a causal relationship has not been clarified so far.

Although all heart valves have the same basic histological structure, there are differences between atrioventricular and ventricular outflow valves; for example, the semilunar leaflets of the aortic valve are thinner and operate under conditions of high-velocity blood flow, resulting in higher shear stress compared with the mitral valve.³⁵ Structural differences overlapping with other risk factors may result in the differences in pathological processes occurring on mitral and aortic leaflets. Thus, we hypothesized that the presence of calcifications on both valves may be associated with higher risk of pathological processes and clinical outcomes.

To our knowledge, we have been the first to evaluate the combined presence of valvular calcifications in relation to cerebrovascular episodes or cardiovascular risk factors and disease. Interestingly, we demonstrated that only the simultaneous presence of valvular calcifications was associated with increased CIMT in univariate analysis. However, this factor was no longer an independent predictor of increased CIMT in multivariate analysis, which might be due to a limited number of patients in the AMVC group.

Our study has several limitations. First, the sample size was small and the study had a retrospective design. Patients were not examined by magnetic resonance imaging and silent stroke could not be analyzed. However, symptomatic stroke is more important from a clinical point

of view. Moreover, we did not evaluate the presence of aortic atheroma by transesophageal echocardiography and its potential influence on neurological episodes. Additionally, we did not use multislice computed tomography to confirm that increased echogenicity on echocardiography is caused by valve calcification and not fibrosis.

In conclusion, we demonstrated a relationship between MACs and increased CIMT but not TIA or stroke in patients with angiographically confirmed coronary artery disease. There is no association between the presence of calcifications both on mitral and aortic valves and carotid atherosclerosis.

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Zwapnienia pierścienia zastawki mitralnej i aortalnej a epizody niedokrwienia mózgu u pacjentów z chorobą niedokrwienną serca

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SŁOWA KLUCZOWE

choroba
niedokrwienna serca,
miażdżycza tętnic
szyjnych, mózgowe
epizody
niedokrwienia,
zwapnienia zastawki
aortalnej, zwapnienia
zastawki mitralnej

STRESZCZENIE

WPROWADZENIE Miażdżycza jest ogólnoustrojowym procesem patologicznym, obejmującym całe łożysko naczyń tętniczych. Zwapnienia zastawkowe są związane z czynnikami ryzyka sercowo-naczyniowego. Istotne zwężenie tętnic dogłowych odpowiada za ok. 20% epizodów niedokrwienia mózgu.

CELE Celem pracy była ocena związku pomiędzy występowaniem zwapnień w pierścieniach zastawki mitralnej i aortalnej, grubością błony wewnętrznej i środkowej tętnic szyjnych (*carotid intima-media thickness* – IMT) oraz mózgowych epizodów niedokrwienych.

PACJENCI I METODY 127 pacjentów z angiograficznie potwierdzoną chorobą wieńcową (68 mężczyzn i 59 kobiet; wiek 74 [33–87] lat) podzielono na 4 grupy: ze zwapnieniami zastawki aortalnej (*aortic valve calcifications* – AVC), ze zwapnieniami zastawki mitralnej (*mitral annular calcifications* – MAC), ze zwapnieniami obu zastawek (*aortic valve and mitral annular calcifications* – AMVC) oraz bez zwapnień na zastawkach (*no valvular calcifications* – no-VC), na podstawie oceny echokardiograficznej. CIMT oraz obecność blaszek miażdżycowych w tętnicach domózgowych oceniano ultrasonograficznie. Uzyskano dane z wywiadu dotyczące występowania mózgowych epizodów niedokrwienych.

WYNIKI Łączna częstość występowania zwapnień na zastawce mitralnej i aortalnej wynosiła 59% (z czego AVC – 55%, MAC – 24%, AMVC – 21%). CIMT była znamienne większa w grupie MAC i AMVC ($p < 0,05$ dla MAC; $p < 0,01$ dla AMVC). Udar niedokrwienno występował częściej w grupie AVC ($p < 0,05$), a w grupie MAC częściej obserwowano obecność blaszek miażdżycowych w tętnicach szyjnych ($p < 0,05$), przejściowych epizodów niedokrwienych (*transient ischemic attack* – TIA; $p < 0,05$) oraz udarów niedokrwienych ($p < 0,05$) w porównaniu z grupą no-VC. W modelu wieloczynnikowym tylko MAC pozostały niezależnie związane ze zwiększoną CIMT.

WNIOSKI U pacjentów z chorobą wieńcową MAC są niezależnie związane ze zwiększoną CIMT, ale nie TIA ani udarami niedokrwienymi mózgu. Nie obserwuje się związku między jednoczesną obecnością zwapnień na zastawkach mitralnej i aortalnej a występowaniem miażdżycy tętnic szyjnych.

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