Original article

Femoral rather than carotid artery ultrasound imaging predicts extent and severity of coronary artery disease

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Abstract

Background: Pathological, epidemiological and clinical studies indicate that there is coexistence between peripheral vascular disease and coronary artery disease (CAD). B-mode ultrasound of superficial arteries is a non-invasive, valid and reproducible method of directly visualising and assessing carotid and femoral intima-media thickness (IMT) and focal atherosclerosis.

Aim: To evaluate the prognostic importance of carotid and femoral vascular changes in predicting CAD severity.

Methods: 410 patients – 300 (73.2%) males and 110 (26.8%) females – aged 29-75 years (mean age 55.9±9.5 years), referred for elective coronary arteriography, were studied. Clinical examination and laboratory tests were performed, and ultrasound assessments of IMT and atherosclerotic plaque thickness in the common carotid arteries and common femoral arteries were evaluated.

Results: Coronary angiography revealed CAD in 81% of patients (85% of males, 70% of females). Cox multiple hazards regression analyses showed a significant relationship between size of atherosclerotic plaques in peripheral arteries and CAD. Odds ratio of CAD associated with every 1-mm plaque thickening ranged from 1.7 to 3.0 ($p < 0.001$) depending on examined artery. Using multiple stepwise regression analysis, the following parameters were found to be independent predictors of one-vessel CAD: myocardial infarction (MI) in anamnesis (OR=22.3; 95% CI 4.0-122.9), typical chest pain (OR=6.4; 95% CI 1.2-34.2), femoral IMT (OR=5.0; 95% CI 1.4-18.4), ex-smoking (OR=5.6; 95% CI 1.1-28.7), and pulse pressure (OR=1.8; 95% CI 1.0-3.2). Independent predictors of multi-vessel CAD were: MI (OR=3.7; 95% CI 1.8-7.5), typical angina (OR=3.3; 95% CI 1.7-6.5), age (OR=1.05; 95% CI 1.01-1.08), number of cigarettes smoked (OR=0.8; 95% CI 0.6-0.9), total cholesterol level (OR=1.1; 95% CI 1.0-1.2), and left femoral plaque thickness (OR=1.4; 95% CI 1.0-2.0).

Conclusion: Femoral IMT is an independent predictor of a single-vessel disease, whereas femoral atherosclerotic plaque presence indicates advanced CAD.

Key words: coronary artery disease, coronary angiography, atherosclerotic plaque, intima-media thickness

Kardiol Pol 2007; 65: 760-766

Introduction

Many clinical, epidemiological and pathological studies indicate that atherosclerosis develops simultaneously in the central and peripheral arterial system [1, 2]. Ultrasonography using high resolution probes enables noninvasive, precise and repetitive visualisation of the superficial large arteries. Assessment of parameters such as intima-media thickness (IMT) or the presence of atherosclerotic plaques in the carotid arteries and/or, less often, femoral arteries is an established method used to estimate with high probability the chance of coronary artery atherosclerosis in selected groups of patients [3-10]. The purpose of this study was to determine whether prediction of coronary artery disease (CAD) severity was possible on the basis of ultrasound imaging of peripheral arteries.

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Received: 13 January 2007. **Accepted:** 21 March 2007.

Methods

The study involved 410 consecutive patients (300 males) at the age of 29-75 years (mean 55.9±9.5) admitted to hospital to perform coronary angiography. Patients after percutaneous and/or surgical revascularisation procedures of either peripheral or coronary arteries and those who did not express consent to participate in the study were excluded. The data on the following parameters were collected: gender, age, diagnosis of arterial hypertension and/or diabetes mellitus, history of MI, smoking status, type of chest pain according to ESC (European Society of Cardiology) classification [11], body mass index (BMI), current arterial pressure and lipidogram. Study group characteristic is presented in Table I.

Prior to coronary angiography every patient underwent ultrasound imaging of the peripheral arteries using a 7.5 MHz probe. The distal 15 mm of the common carotid arteries and their bulbs as well as the common femoral arteries below the inguinal ligament were visualised. The IMT of the distal 10-15 mm segments of the common carotid and common femoral arteries free from atherosclerotic lesions were calculated using Image-Pro Plus computer software. Each measurement was taken for the distal vascular wall after an image of longitudinal artery section was frozen at the beginning of the QRS complex (in order to eliminate possible differences due to cardiac cycle phase). Segments of 10 mm length of proximal edge of the first line (lumen – intima border) as well as proximal edge of the second echo line (intima – adventitia border) were contoured and then mean distance between them was calculated out of three measurements [12]. Atherosclerotic plaque was defined as echo-dense structure protruding into the arterial lumen by at least 50% in relation to the inner contour of IMT of the adjacent vascular wall. Calculation of atherosclerotic plaque thickness was performed on the frozen image measuring maximal distance between lumen – plaque and media – adventitia borders. Dimension of the most thickened lesion in a given artery was used for further analysis.

Coronary angiography was performed using the Judkins technique. Coronary artery disease was defined as the presence of at least one stenosis exceeding 50% of the coronary artery diameter. Lesion severity was assessed by means of Gensini angiographic score calculation [13] and according to the number of atherosclerotic arteries. Division of coronary vascular bed into three arteries – right coronary, left anterior descending and circumflex – was adopted. The criterion to distinguish one-, two- and three-vessel disease was lesion location rather than the total number of stenotic lesions. The left main artery stenosis was classified as two-vessel disease. Two- or three-vessel disease and/or Gensini index >10 were considered to define severe (multi-vessel) CAD.

Statistical analysis

Statistical analysis was carried out by means of logistic regression analysis that enabled searching for risk

Table I. Selected parameters of physical and biochemical examinations as well as ultrasound imaging of the peripheral arteries in the study group

Variable	Value
Age [years]	55.9±9.5
BMI [kg/m^2]	27.4±3.6
Coronary artery disease familiar burden [%]	19.9
Diabetes mellitus* [%]	9.4
History of myocardial infarction [%]	56.3
Hypertension** [%]	47.3
Hypercholesterolaemia [%]	43.7
Active smoking [%]	34.9
History of smoking [%]	38.5
Systolic blood pressure [mmHg]	129.9±18.7
Diastolic blood pressure [mmHg]	79.8±11.0
Total cholesterol concentration [mg/dl]	210.6±40.9
HDL-cholesterol concentration [mg/dl]	44.0±12.2
LDL-cholesterol concentration [mg/dl]	133.0±36.2
Triglyceride concentration [mg/dl]	165.5±90.9
Parameters of peripheral arteries ultrasound imaging:	
IMT of the right common coronary artery [mm]	0.65±0.19
Atherosclerotic plaque thickness in the right common coronary artery [mm]	0 (0.00, 1.25)
IMT of the left common coronary artery [mm]	0.69±0.20
Atherosclerotic plaque thickness in the left common coronary artery [mm]	0 (0.00, 1.18)
Average IMT of both common coronary arteries [mm]	0.67±0.17
IMT of the right femoral artery [mm]	0.80±0.35
Atherosclerotic plaque thickness in the right femoral artery [mm]	1.1 (0.00, 1.68)
IMT of the left femoral artery [mm]	0.78±0.32
Atherosclerotic plaque thickness in the left femoral artery [mm]	1.04 (0.00, 1.60)
Average IMT of both common femoral arteries [mm]	0.78±0.28

Values expressed as mean±SD or median (Q_1, Q_3)
**defined as fasting glucose ≥110.0 mg/dl and/or antidiabetic treatment*
***defined as systolic arterial pressure ≥140 mmHg and/or diastolic one ≥90 mmHg*

Table II. Prevalence of significant atherosclerotic lesions in males and females

Number of affected coronary arteries	Males		Females		p	Total	
	n	%	n	%		n	%
0	45	15.0	33	30.0	<0.0001	78	19.0
1	55	18.3	18	16.4	NS	73	17.8
2	77	25.7	33	30.0	NS	110	26.8
3	123	41.0	26	23.6	<0.0001	149	36.4
Sum	300	100.0	110	100.0		410	100.0

factors associated with diagnosis of one- and multi-vessel CAD. Search for the risk factors of significant stenosis was performed step by step. First, all factors were grouped according to the relations between one another, known from available literature or taking into consideration their natural associations. In each distinguished group of potential CAD risk factors, their correlation with the risk of coronary artery lesions was estimated.

All promising variables found this way were entered again into the selection procedure and eventually a final group of risk factors promoting development of significant lesions in the coronary arteries was chosen. Correlation power between individual variables and risk of atherosclerotic lesions coexistence in the coronary arteries was expressed as the odds ratio (OR). In the case of qualitative variables it indicated whether a person exposed to a given factor was at higher/lower risk of developing coronary lesions. In the case of quantitative variables it indicated how much a person in whom level of a given risk factor increased by a certain value, e.g. age by 1 year, smoking period – 10 years, number of cigarettes – 10 per day, BMI – 1 kg/m^2, total cholesterol and its fraction concentrations – 10 mg/dl, systolic, diastolic and pulse pressure – 10 mmHg, IMT – 0.5 mm, atherosclerotic plaque lesion – 1.0 mm, was at higher/lower risk of CAD.

Precision of ORs estimation was expressed as 95% confidence interval (95% CI) and p value.

In the subsequent statistical analysis relationships between all collected results (even if they were not found to be risk factors for coronary lesions coexistence in the multivariate regression analysis) and predictors of coronary atherosclerosis were examined. The aim of such an approach was to reveal risk factors for coronary lesions coexistences that being associated with predictors were not able to be found independent risk factors. This part of the statistical analysis was based on evaluation of correlation in the case of quantitative parameters and on Student's t-test as well as Wilcoxon test if a given predictor or studied factors were qualitative (e.g. coexistence of arterial hypertension, typical pain, etc.). The results for quantitative variables were presented as arithmetic means and standard deviations or medians and quartiles.

Calculations were performed using SAS 9.0 and S-Plus system 6.1.

Results

On coronary angiography significant lesions of coronary arteries were found in 332 (81%) patients including 255 (85%) males and 77 (70%) females. The extent and severity of CAD are presented in Table II. The study group characteristics regarding parameters both of clinical examination and assessed on peripheral arteries ultrasonography, particularly the prevalence of atherosclerotic plaques in the peripheral arteries in relation to presence of CAD and significance of atherosclerotic plaque visualisation in the peripheral arteries for CAD diagnosis, have been presented in detail elsewhere [3, 10].

The mean value of the Gensini score in the study group was 19 (9, 29), lower in females – 5 (5, 23) than in males – 20 (10, 30). Its value showed a positive correlation with age (r=0.24, p <0.0001), systolic blood pressure (r=0.12, p <0.02), pulse pressure (r=0.14, p <0.007), total cholesterol concentration (r=0.17, p <0.0004), HDL-C (r=–0.10, p <0.04), LDL-C (r=0.21, p <0.0001), thickness of atherosclerotic plaques in the left common carotid artery (r=0.24, p <0.0001) as well as in the right common carotid artery (r=0.26, p <0.0001), left common femoral artery (r=0.26, p <0.0001) and right common femoral artery (r=0.25, p <0.0001).

Multivariate analysis of regression showed that the most powerful risk factors for the presence of significant atherosclerotic lesions in the coronary arteries were as follows: history of MI, presence of typical anginal pain, smoking, presence of atherosclerotic plaques in femoral arteries and in the left common carotid artery (Table III). The risk of CAD increased together with increased thickness of the atherosclerotic plaque seen in the peripheral artery (Table IV).

The best model of the independent risk factors of one- and multi-vessel CAD is presented in Tables V and VI.

Discussion

The results of our study indicate that the presence and extent of atherosclerotic plaques in the peripheral arteries rather than thickening of intima-media complex predict significant CAD. This is consistent with the findings of previously published reports showing that the presence of atherosclerotic plaques in the peripheral arteries more precisely than IMT predicted acute coronary syndromes and cardiac-related sudden deaths [4, 5, 14, 15], and that such risk increases together with plaque size [14-16]. Others suggested that in patients with typical angina the presence of atherosclerotic lesions in the carotid arteries proves the existence of severe CAD (three-vessel disease or left main stenosis) [6, 7]. Kallikazaros et al. also believe that the absence of atherosclerotic lesions in the peripheral arteries in patients with normal systolic performance of the left ventricle indicates low probability of severe CAD [7]. In the author's opinion, visualisation of atherosclerotic plaques, unlike measuring IMT, does not require special training or additional software and is much easier, more repetitive and faster to perform. The method may then be more commonly used in clinical practice.

Thickness of atherosclerotic plaques in the peripheral arteries was found in our study to be the most important risk factor of CAD. In the available literature there are only a few reports suggesting a close relationship between the extent of atherosclerotic plaques in the peripheral arteries and CAD presence or progression. Spence et al. found in individuals with suspected CAD that total area of the atherosclerotic plaque measured in the common, internal as well as external carotid arteries was a more appropriate risk index of acute coronary syndromes than IMT [16].

Our original finding is that in subjects with suspected CAD, ultrasonography of the common femoral arteries is more informative than of the carotid arteries. It allows to estimate not only the probability of coronary artery atherosclerosis but its severity as well. It seems that atherosclerosis develops mainly at the sites of vessel bifurcation and their bends [17]. It suggests that local factors, including blood flow, have a pronounced impact on its development. It may be so that similar underlying local haemodynamic factors (e.g. flow disturbances) as a result of the bent course of both arteries lead to simultaneous development of atherosclerosis in the coronary and femoral arteries [18]. This concept may be confirmed by the fact that atherosclerotic plaques in bent femoral arteries occur earlier and more frequently than in common carotid arteries with a straight course [14, 19-21]. Contrary to our results, findings from autopsy studies show a weaker correlation between coronary and femoral artery

Table III. Risk factors of significant coronary artery stenosis – odds ratios (OR) and p values

Risk factor	OR	95% CI	p
Previous myocardial infarction	21.5	6.0-76.7	<0.0001
Typical anginal pain	9.5	2.9-31.7	0.0002
Active smoking	0.3	0.09-1.0	<0.05
Pulse pressure	1.6	1.0-2.5	<0.05
Atherosclerotic plaque in the left femoral artery	2.0	1.1-3.8	<0.03
Atherosclerotic plaque in the right femoral artery	2.3	1.0-5.3	<0.04
Atherosclerotic plaque in the left carotid artery	9.3	2.1-39.7	<0.003

Table IV. Risk of coronary artery disease associated with increased thickness of atherosclerotic plaque in the peripheral artery by 1 mm

Artery	OR	95% CI	p
Left carotid	3.0	1.6-7.5	<0.001
Right carotid	1.7	1.5-4.8	0.0007
Left femoral	2.0	1.7-4.2	<0.001
Right femoral	2.9	2.2-6.3	<0.001

Table V. Risk factors of single-vessel disease, odds ratios and p values

Risk factor	OR	95% CI	p
Previous myocardial infarction	22.3	4.0-122.9	0.0004
Typical anginal pain	6.4	1.2-34.2	<0.03
Intima-media thickness of the right femoral artery	5.0	1.4-18.4	<0.02
Smoking in history	5.6	1.1-28.7	<0.04
Elevated pulse pressure	1.8	1.0-3.2	<0.05

Table VI. Risk factors of severe coronary artery disease, odds ratios and p values

Risk factor	OR	95% CI	p
Previous myocardial infarction	3.7	1.8-7.5	0.0004
Typical anginal pain	3.3	1.7-6.5	0.0006
Age	1.05	1.01-1.08	<0.01
Number of cigarettes per day	0.8	0.6-0.9	<0.02
Increased serum concentration of total cholesterol	1.1	1.05-1.2	<0.03
Atherosclerotic plaque thickness in the left femoral artery	1.4	1.0-2.0	<0.05

atherosclerosis as compared to the relationship between carotid and coronary arteries [1, 2, 17]. It is of note that both internal and external carotid arteries were also examined. Data collected by J. Salonen and R. Salonen suggest that different factors contribute to atherosclerosis development in the femoral arteries compared to either the carotid or the coronary arteries [4]. Some investigators believe that due to vessels' structure and their course, local haemodynamic abnormalities have a more expressed impact on atherosclerosis development in the common carotid than the common femoral arteries. Thus, systemic risk factors having an impact on atherosclerotic lesions development should have played a more pronounced role in the case of femoral arteries [22].

Ultrasound imaging of the internal and external carotid arteries is more demanding due to anatomical circumstances and is not always possible to be carried out in each patient. Their assessment is less accurate and reproducible as compared to the common carotid and femoral arteries. Held et al. observed that patients with atherosclerotic plaques found in the femoral arteries more frequently required coronary artery revascularisation. Moreover, no correlation between the presence of atherosclerotic lesions in the femoral arteries and elevated risk of either MI or sudden cardiac death was noted in their study. They suggest that the presence of atherosclerotic plaques in the carotid arteries may reflect coexistence of unstable plaques in the coronary arteries while atherosclerotic lesions in the femoral arteries may be a marker of slow and progressive CAD (i.e. stable plaques) [14].

Some authors believe that the intima-media thickening may precede atherosclerosis development [23] or simply may represent its early stage [4, 12]. This concept seems to be supported by our own results suggesting that intima-media thickening in the common femoral arteries reflects benign coronary artery atherosclerosis. At the same time, the presence of more advanced atherosclerosis in the peripheral arteries, i.e. plaques, markedly increases the risk of severe CAD.

The discordance of our results and findings of other reports regarding a correlation between carotid artery IMT and coronary artery atherosclerosis [8, 9] is likely caused by methodological differences. Contrary to those authors, our IMT measurements were taken only at sites free from atherosclerotic lesions in the medial and distal segments of the common carotid arteries. Atherosclerosis in these segments is a rare finding [24]. Other authors evaluated IMT independently of the presence of atherosclerotic plaques or calculated maximal thickness of intima-media complex. In our study IMT exceeding 1.0 mm was thought to represent extensive (diffused) atherosclerosis.

Conclusions

Ultrasound visualisation of common femoral arteries enables better assessment of coronary artery atherosclerosis than imaging of the common carotid arteries. Thickening of the intima-media complex of femoral arteries is an independent risk factor for one-vessel CAD development while presence of atherosclerotic lesions in the common femoral arteries strongly indicates coexistence of severe CAD.

References

1. Mitchell JR, Schwartz CJ. Relationship between arterial disease in different sites. A study of the aorta and coronary, carotid, and iliac arteries. *Bt Med J* 1962; 1: 1293-301.
2. Young W, Gofman JW, Tandy R, et al. The quantitation of atherosclerosis III: the extent of correlation of degrees of atherosclerosis within and between the coronary and cerebral vascular beds. *Am J Cardiol* 1960; 6: 300-8.
3. Pasierski T, Sosnowski C, Szulczyk A, et al. Czy ocena zmian miażdżycowych w tętnicach obwodowych może odegrać rolę w rozpoznawaniu choroby wieńcowej? *Pol Arch Med Wewn* 2004; 111: 21-5.
4. Salonen JT, Salonen R. Ultrasound B-mode imaging in observational studies of atherosclerotic progression. *Circulation* 1993; 87 (3 Suppl): II56-65.
5. Salonen R, Salonen JT. Determinants of carotid intima-media thickness: a population-based ultrasonography study in eastern Finnish men. *J Intern Med* 1991; 229: 225-31.
6. Craven TE, Ryu JE, Espeland MA, et al. Evaluation of the associations between carotid artery atherosclerosis and coronary artery stenosis. A case-control study. *Circulation* 1990; 82: 1230-42.
7. Kallikazaros I, Tsioufis C, Sideris S, et al. Carotid artery disease as a marker for the presence of severe coronary artery disease in patients evaluated for chest pain. *Stroke* 1999; 30: 1002-7.
8. Kablak-Ziembicka A, Tracz W, Przewlocki T, et al. Association of increased carotid intima-media thickness with the extent of coronary artery disease. *Heart* 2004; 90: 1286-90.
9. Kotsis VT, Pitiriga VCh, Stabouli SV, et al. Carotid artery intima-media thickness could predict the presence of coronary artery lesions. *Am J Hypertens* 2005; 18: 601-6.
10. Sosnowski C, Pasierski T, Janeczko-Sosnowska E, et al. Przydatność ultrasonograficznej oceny miażdżycy tętnic szyjnych i udowych w ocenie ryzyka choroby wieńcowej. *Pol Przegl Kardiol* 2005; 7: 389-95.
11. Guidelines for cardiac exercise testing. ESC Working Group on Exercise Physiology, Physiopathology and Electrocardiography. *Eur Heart J* 1993; 14: 969-78.
12. Pignoly P, Tremoli E, Poli A, et al. Intimal plus medial thickness of the arterial wall: a direct measurement with ultrasound imaging. *Circulation* 1986; 74: 1399-406.
13. Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. *Am J Cardiol* 1983; 51: 606.
14. Held C, Hjemdahl P, Eriksson SV, et al. Prognostic implications of intima-media thickness and plaques in the carotid and femoral arteries in patients with stable angina pectoris. *Eur Heart J* 2001; 22: 62-72.

15. Van der Meer IM, Bots ML, Hofman A, et al. Predictive value of noninvasive measures of atherosclerosis for incident myocardial infarction: the Rotterdam Study. *Circulation* 2004; 109: 1089-94.
16. Spence JD, Eliasziw M, DiCicco M, et al. Carotid plaque area: a tool for targeting and evaluating vascular preventive therapy. *Stroke* 2002; 33: 2916-22.
17. Solberg LA, Eggen DA. Localization and sequence of development of atherosclerotic lesions in the carotid and vertebral arteries. *Circulation* 1971; 43: 711-24.
18. Smedby O. Geometrical risk factors for atherosclerosis in the femoral artery: a longitudinal angiographic study. *Ann Biomed Eng* 1998; 26: 391-7.
19. Solberg LA, McGarry PA, Moossy J, et al. Severity of atherosclerosis in cerebral arteries, coronary arteries, and aortas. *Ann N Y Acad Sci* 1968; 149: 956-73.
20. Kröger K, Kucharczik A, Hirche H, et al. Atherosclerotic lesions are more frequent in femoral arteries than in carotid arteries independent of increasing number of risk factors. *Angiology* 1999; 50: 649-54.
21. Sosnowski C, Pasierski T, Janeczko-Sosnowska E, et al. Miażdżyca dużych tętnic obwodowych u pacjentów z podejrzeniem choroby wieńcowej. *Folia Cardiol* 2005; 12: 635-43.
22. Bonithon-Kopp C, Scarabin PY, Taquet A, et al. Risk factors for early carotid atherosclerosis in middle-aged French women. *Arterioscler Thromb* 1991; 11: 966-72.
23. Hijmering ML, Bots ML, Stroes ES, et al. Non-invasive evaluation of vascular function. *Eur Heart J Supplements* 1999; 1 (Suppl M): M18-M26.
24. Zureik M, Temmar M, Adamopoulos C, et al. Carotid plaques, but not common carotid intima-media thickness, are independently associated with aortic stiffness. *J Hypertens* 2002; 20: 85-93.

Ultrasonograficzna ocena tętnic udowych w porównaniu z oceną tętnic szyjnych pozwala na lepsze przewidywanie obecności i zaawansowania choroby wieńcowej

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Streszczenie

Wstęp: Proces miażdżycowy rozwija się jednocześnie w wielu tętnicach i bardzo rzadko jest ograniczony tylko do jednego łożyska naczyniowego. Wyniki licznych badań autopsyjnych, epidemiologicznych i klinicznych wskazują, że pogrubienie błony wewnętrzno-środkowej tętnic szyjnych i udowych oraz obecność w ich ścianie blaszek miażdżycowych współistnieje z miażdżycą tętnic wieńcowych. Dlatego też ultrasonograficzna ocena tętnic szyjnych i udowych jest uznaną metodą pozwalającą z dużą dokładnością określić prawdopodobieństwo choroby wieńcowej (CAD) w wybranych grupach chorych.

Cel: Ocena przydatności badania ultrasonograficznego dużych tętnic obwodowych w przewidywaniu zaawansowania miażdżycy tętnic wieńcowych.

Metodyka: Badaniami objęto kohortę 410 kolejnych chorych (73,2% mężczyzn) w wieku 29–75 lat, średnio 55,9±9,5, kierowanych do inwazyjnej diagnostyki CAD zgodnie z wytycznymi PTK. Przed koronarografią u każdego chorego przeprowadzono badania kliniczne i laboratoryjne dotyczące czynników ryzyka miażdżycy, a następnie za pomocą sondy 7,5 MHz sprzężonej z aparatem Hewlett Packard wykonano badanie ultrasonograficzne tętnic szyjnych wspólnych i udowych wspólnych z oceną grubości błony wewnętrzno-środkowej oraz obecności i grubości blaszek miażdżycowych. Do pomiaru grubości błony wewnętrzno-środkowej używano konwencji zweryfikowanej histopatologicznie przez Pignoli i wsp., w której błona wewnętrzno-środkowa odpowiada pierwszej warstwie hipoechogenicznej ściany dalszej. Blaszka miażdżycowa została zdefiniowana jako pogrubienie błony wewnętrznej i środkowej o ponad 50%, wpuklające się do światła tętnicy. Pomiaru grubości dokonywano w miejscu jej maksymalnego wychylenia się do wnętrza naczynia. Chorobę wieńcową zdefiniowano jako obecność co najmniej jednego zwężenia >50%, jej zaawansowanie oceniono w zależności od liczby zwężonych tętnic jako jedno- i wielonaczyniową oraz wyliczając angiograficzny wskaźnik Gensiniego.

Wyniki: Chorobę wieńcową rozpoznano angiograficznie u 81% badanych – u 17,8% chorobę jednonaczyniową, u 63,2% chorobę wielonaczyniową. Wieloczynnikowa analiza regresji wykazała, że blaszki miażdżycowe uwidocznione w tętnicy obwodowej są niezależnym czynnikiem ryzyka obecności CAD. Ryzyko to wzrasta z grubością blaszek i dla 1 mm w zależności od lokalizacji waha się od 1,7 do 3,0 ($p < 0{,}001$). Logistyczna analiza regresji Coksa ujawniła ponadto, że niezależnymi czynnikami ryzyka jednonaczyniowej CAD były: przebyty zawał mięśnia serca (OR=22,3; 95% CI 4,0–122,9; p=0,0004), obecność typowych bólów dławicowych (OR=6,4; 95% CI 1,2–34,2; $p < 0{,}03$), grubość błony wewnętrzno-środkowej w tętnicy udowej prawej (OR=5,0; 95% CI 1,4–18,4; $p < 0{,}02$), palenie tytoniu w przeszłości (OR=5,6; 95% CI 1,1–28,7; $p < 0{,}04$) i podwyższone ciśnienie tętna (OR=1,8; 95% CI 1,0–3,2; $p < 0{,}05$), natomiast niezależnymi czynnikami ryzyka wielonaczyniowej CAD były: przebyty zawał mięśnia serca (OR=3,7; 95% CI 1,8–7,5; p=0,0004), obecność typowych bólów dławicowych (OR=3,3; 95% CI 1,7–6,5; p=0,0006), wiek (OR=1,05; 95% CI 1,01–1,08; $p < 0{,}01$), liczba papierosów wypalanych na dobę (OR=0,8; 95% CI 0,6–0,9; $p < 0{,}02$), podwyższone stężenie cholesterolu całkowitego w osoczu krwi żylnej (OR=1,1; 95% CI 1,0–1,2; $p < 0{,}03$) i grubość blaszki miażdżycowej ujawnionej w tętnicy udowej lewej (OR=1,4; 95% CI 1,0–2,0; $p < 0{,}05$).

Wnioski: Badanie ultrasonograficzne tętnic udowych wspólnych w porównaniu z obrazowaniem tętnic szyjnych wspólnych pozwala na lepszą ocenę prawdopodobieństwa obecności i zaawansowania miażdżycy tętnic wieńcowych. Pogrubienie błony wewnętrzno-środkowej tętnic udowych wspólnych jest niezależnym czynnikiem ryzyka jednonaczyniowej CAD, obecność w nich blaszek miażdżycowych świadczy natomiast o dużym prawdopodobieństwie bardziej zaawansowanej CAD.

Słowa kluczowe: choroba wieńcowa, koronarografia, blaszka miażdżycowa, błona wewnętrzno-środkowa

Kardiol Pol 2007; 65: 760-766

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Praca wpłynęła: 13.01.2007. **Zaakceptowana do druku:** 21.03.2007.