Original article

The prevalence of potentially unstable coronary lesions in patients with coronary artery disease – virtual histology study

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Abstract

Background: Histopathological studies indicate that coronary artery lesions with a thin fibrous cap and large necrotic core (thin-cap fibroatheromas, TCFA) are characterised by a high risk of rupture and can potentially trigger acute coronary syndrome (ACS). Atherosclerotic lesions with a well preserved fibrous cap (fibroatheromas, FA) are considered to be more stable ones. Intravascular ultrasound virtual histology (IVUS-VH) enables identification of FA and TCFA in vivo. There are no published data regarding IVUS-VH derived occurrence of both FA and TCFA in patients with different clinical presentation.

Aim: To determine IVUS-VH derived occurrence of FA and TCFA in coronary arteries of patients with chronic stable angina in comparison with recent or acute ACS subjects.

Methods: Intravascular ultrasound examination was performed in 60 patients, who were prospectively enrolled to three groups: group A – chronic stable angina, without a history of ACS within the previous 12 months; group B – recent ACS (4 weeks – 3 months); group C – acute phase of ACS.

Results: The final analysis included 75 non-culprit lesions (group A: n=29 lesions; group B: n=22; group C: n=24). There were no significant differences in lesions' angiographic and ultrasound characteristics between the studied groups. There was no significant difference in the occurrence of FA lesions between the studied groups (20.7 vs. 22.7 vs. 4.2, respectively; NS). There was a significant difference in TCFA occurrence between the studied groups (31.0 vs. 50.0 vs. 79.2%; $p < 0.01$).

Conclusions: The present study confirms higher occurrence of thin-cap fibroatheromas in patients with clinically confirmed coronary instability. It also indicates that IVUS-VH can be a valuable tool for rupture prone lesion identification, which might help in better risk stratification in coronary artery disease patients.

Key words: acute coronary syndromes, thin-cap fibroatheroma, virtual histology

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Introduction

Atherosclerotic plaque rupture is known to be a major cause of acute coronary syndromes (ACS) [1]. On the basis of post mortem examinations of subjects who died of ACS it has been demonstrated that so-called atherosclerotic plaque with a thin fibrous cap (thin-cap fibroatheroma, TCFA) is the lesion morphologically most similar to the ruptured plaque. It may be characterised by the presence of a well developed necrotic core, which size in most cases exceeds 10% of the total surface area of the lesion. Another morphological element enabling the recognition of TCFA is a thin strand of fibrous tissue, which separates the thrombogenic area inside the plaque from the lumen of the vessel and which width does not exceed 65 μm in 95% of cases. Another atherosclerotic lesion which contains a well developed necrotic core with a fibrous cap (fibroatheroma, FA) is considered to have a lower risk of rupture [2].

The virtual histology system that has recently been introduced into clinical practice (intravascular ultrasound virtual histology, IVUS-VH) is the first diagnostic tool allowing *in situ* assessment of the composition of atherosclerotic changes. This system is a modification of intracoronary ultrasound equipment used so far and enables the identification of all four components of plaque: fibrous tissue, fibrous tissue with adipose elements, calcifications, and necrotic core. Virtual

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histology is also the first imaging method that allows *in vivo* recognition of TCFA and FA changes in coronary arteries of patients with various forms of ischaemic heart disease. So far there have been no published reports comparing the prevalence of these lesions in a population of subjects with stable angina, subjects after ACS, and patients in the acute phase of ACS.

The aim of this study was to assess the prevalence of atherosclerotic plaques of FA and TCFA morphology in the above-mentioned groups of patients.

Methods

Examined groups of subjects

The study included 60 consecutive patients who were prospectively allocated to one of the three groups.

Group A (stable angina) included patients with stable angina diagnosed on the basis of a typical history of chest pain provoked by exertion, class 2 and 3 CCS (Canadian Cardiovascular Society). In all patients the diagnosis was confirmed with electrocardiographic treadmill exercise test according to the standard protocol. Patients who were diagnosed with ACS within 12 months prior to enrolment were excluded from this group.

Group B (history of ACS) included patients who had ACS within 4 weeks to 3 months before the study, who still presented with typical symptoms of angina and in whom signs of ischaemia were observed in non-invasive exercise testing. ACS was defined as a resting chest pain lasting more than 20 minutes, accompanied by an increase of serum myocardial necrosis marker concentrations above the threshold values valid in the centre where the patient was hospitalised.

Group C (acute phase of ACS) included patients diagnosed with non-ST elevation ACS who were qualified for the invasive treatment strategy and underwent coronary angiography within the first 48 hours of hospitalisation. ACS with no ST elevation was defined as a resting chest pain, lasting >20 minutes, accompanied by changes in resting 12-lead electrocardiogram, defined as ST-segment depression of at least 1 mm in at least two contiguous leads.

Patients with acute ST-segment elevation myocardial infarction (STEMI) were not qualified for the study. Other excluding criteria were as follows: cardiogenic shock, angiographically significant stenosis of the left main coronary artery, multivessel coronary artery disease (CAD) requiring surgical treatment, anatomical variations of the coronary arteries or morphology of atherosclerotic changes that made the insertion of an ultrasound probe through the lesion impossible, and the inability to obtain consent from the patient planned to participate in the study.

Coronary angiography

In all patients coronary angiography was performed according to the standard procedure. Immediately after angiography the IVUS examination was conducted. The analysis included lesions that, according to the examiners, were not directly responsible for ischaemia (non-culprit lesions). Mostly borderline lesions according to angiography (40-70% occlusion of the vessel) were evaluated. In some patients the evaluation also included significant lesions, when the degree of stenosis allowed safe administration of the IVUS probe. The quantitative coronary angiography (QCA) digital system was used for the measurement of the degree of vessel obstruction.

Ultrasound analysis

An electronic IVUS system was used for the ultrasound analysis (Volcano In-Vision Gold, version 4.3). After intracoronary administration of 200 μg of nitroglycerin, the ultrasound catheter (EagleEye Gold 20 MHz; Volcano Corp.) was introduced to the examined vessel and placed at least 10 mm distally to the examined lesion. The ultrasound probe was then pulled back in the vessel at a constant speed of 0.5 mm/s, using a standardised device (R-100 Pullback Device; Volcano Corp.). The ultrasound analysis was conducted according to the guidelines of the American College of Cardiology [3]. In view of the limitations of the IVUS-VH system to identify and properly classify intravascular thrombi, atherosclerotic changes containing a thrombus were excluded from the analysis.

The assessment of the atherosclerotic changes conducted using the Virtual Histology system (Virtual Histology 1.3; Volcano Corp.), similarly to the assessment with the use of 'classical' IVUS analysis, was started by establishing the proximal and distal reference points. Images obtained from grey-scale ultrasonography were used so that the reference points in IVUS-VH were compatible with previously identified reference points in classical IVUS. The lesions of FA and TCFA morphology were recognised based on criteria described elsewhere [4, 5]. In the case of TCFA they included a well organised necrotic core constituting >10% of the plaque surface area, direct communication between the core and the lumen of the vessel, and no ultrasound features of a strand of fibrous tissue. The criteria of FA recognition included: a well organised necrotic core constituting more than 10% of the plaque surface area, the presence of a visible strand of fibrous tissue between the necrotic core and the lumen of the vessel. These criteria had to be fulfilled in three subsequent cross-sections of the vessel. Examples of lesions of TCFA and FA morphology are presented in Figure 1.

Ethics

The protocol of the study was approved by the Ethic Committee of the Medical Academy in Warsaw. All patients expressed their written consent to participate in the study.

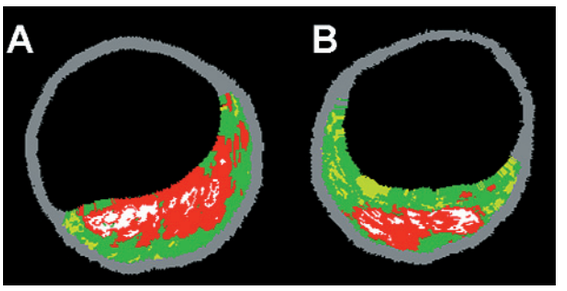

Figure 1. Example of atherosclerotic lesion of TCFA morphology (**A**) and of lesion with a well developed necrotic core and a fibrous cap – FA (**B**)

Statistical analysis

The statistical analysis was conducted using the SPSS program (v. 11.5; SPSS Inc., Chicago, Illinois, USA).

Data are presented as mean values ± standard deviation. The analysis of mean values was performed with Student's t-test. Discrete data are presented as numbers and percentages, and differences were compared using χ^2 test. Correlations were assessed with Pearson test. The value of $p < 0.05$ was regarded as significant.

Results

The study included 60 patients, in whom 83 atherosclerotic lesions in coronary arteries were identified using IVUS. Eight atherosclerotic plaques were excluded from the analysis because of the presence of massive calcifications or intravascular thrombus. The final analysis included 75 lesions in 56 subjects. There were 24 patients (29 lesions) in group A, and 16 patients (22 lesions) were qualified to group B. Because of the difficulties in documenting previous diagnosis of unstable angina, it was decided that group B would include only patients with documented MI. This group eventually included 9 subjects who had undergone STEMI (7 subjects treated with primary coronary angioplasty, 2 subjects receiving thrombolysis) and 7 patients after non-ST-elevation MI (NSTEMI) (3 of whom were treated with angioplasty in the acute phase and 4 who received pharmacological treatment). The mean time from ACS was 55 days. Group C included 16 patients (24 lesions); in 10 subjects NSTEMI was recognised, and 6 patients underwent coronary angiography due to unstable angina.

There were no significant differences in the prevalence of coronary artery disease risk factors or comorbidities between groups. The mean age of patients in group B was significantly lower compared with two other subpopulations. Patients from group B were also more often found to have undergone MI and coronary angioplasty (Table I).

Patients from group B received the most aggressive treatment. The rate of typical medications use in the prevention of secondary cardiovascular events was found to be the highest in this group of subjects. Patients from group C were found to have the lowest rate of administration of statins and aspirin. This observation might be explained by the fact that in many patients from this population the analysed episode of ACS was the first symptom of CAD (Table II).

Angiographic analysis did not reveal any significant differences in the distribution of atherosclerotic changes in coronary arteries between the groups. No significant differences in the prevalence of angiographically significant changes were found and the mean degree of stenosis calculated using digital quantitative angiography was comparable between examined groups. There were also no significant differences in the mean values of classic ultrasound parameters, such as the lumen area of the vessel, minimal lumen area, and proportional area of the vessel covered with atherosclerotic plaque (plaque burden). Furthermore, calculated values of the volume of artery components,

Table I. Clinical characteristics of the examined groups of patients

Parameter	Group A	Group B	Group C	p
Age [years]	61.2±8.6	54.2±10.3	65.5±11.37	<0.05*
Males	16 (66.7%)	10 (62.5%)	11 (68.8%)	NS
Hypertension	19 (79.2%)	10 (62.5%)	13 (81.3%)	NS
Diabetes mellitus	6 (25.0%)	3 (18.8%)	2 (12.5%)	NS
Stroke/transient ischemic attack	4 (16.7%)	0 (0.0%)	1 (6.3%)	NS
Hyperlipidaemia	20 (83.3%)	12 (75.0%)	12 (75.0%)	NS
Smoking	8 (33.3%)	11 (68.8%)	7 (43.8%)	NS
Past myocardial infarction	3 (12.5%)	16 (100%)	4 (25.0%)	<0.01*
History of percutaneous coronary intervention	7 (29.2%)	10 (62.5%)	3 (18.8%)	<0.05*

** A vs. B, B vs. C, A vs. C – NS*

Table II. The use of medication in examined groups of patients

Drug	Group A	Group B	Group C	p
Aspirin	23 (95.8%)	16 (100%)	9 (56.3%)	<0.01*
Beta-blockers	21 (87.5%)	15 (93.8%)	8 (50.0%)	<0.01*
ACE inhibitors	18 (75.0%)	12 (75.0%)	4 (25.0%)	<0.01*
Statins	22 (91.7%)	16 (100%)	7 (43.0%)	<0.01*
Thienopyridine derivatives	5 (20.8%)	10 (62.5%)	0 (0.0%)	<0.01**
Calcium channel blockers	8 (33.3%)	2 (12.5%)	3 (18.8%)	NS

** A vs. C, B vs. C, A vs. B – NS; ** B vs. C, A vs. C – p <0.05; A vs. B – p <0.05*

Table III. Angiographic and ultrasonographic characteristics of examined lesions

Parameter	Group A	Group B	Group C	p
Localisation of examined lesions				
left main coronary artery	2 (6.9%)	1 (4.6%)	2 (8.3%)	NS
left anterior descending	18 (62.1%)	16 (72.7%)	15 (62.5%)	NS
circumflex artery	1 (3.5%)	0 (0.0%)	2 (8.3%)	NS
right coronary artery	8 (27.6%)	5 (22.7%)	5 (20.8%)	NS
Angiographically borderline lesion	28 (96.6%)	18 (81.8%)	21 (87.5%)	NS
Angiographically significant lesion	1 (3.5%)	4 (18.2%)	3 (12.5%)	NS
Lumen area of the vessel [mm^2]	5.23±2.65	4.95±2.02	4.98±1.64	NS
Surface area of the vessel [mm^2]	15.79±5.39	16.67±5.76	17.34±4.64	NS
Atherosclerotic plaque size [%]	66.50±11.18	69.90±8.10	70.31±9.31	NS
Volume of the vessel [mm^3]	227.18±161.05	224.69±129.58	286.38±154.25	NS
Volume of the plaque [mm^3]	128.63±107.08	133.03±75.94	165.09±90.66	NS
Length of the lesion [mm]	13.99±4.31	14.54±5.09	16.37±5.05	NS

Table IV. Prevalence of FA and TCFA lesions

	Group A	Group B	Group C	p
Fibroatheromas	6 (20.7%)	5 (22.7%)	1 (4.2%)	NS
Thin-cap fibroatheromas	9 (31.0%)	11 (50.0%)	19 (79.2%)	<0.01*

** p value for χ^2 for the whole group; A vs. B – NS; B vs. C – p <0.05; A vs. C – p <0.01*

such as the lumen, atherosclerotic plaque and the vessel, did not significantly differ between examined populations (Table III).

A significant correlation was found between the clinical characteristics of subjects and the prevalence of TCFA lesions. The lowest frequency was demonstrated in group A, the highest in group C. Moreover, subjects from group C had a tendency to a lower prevalence of FA lesions; the difference was not, however, significant (Table IV; Figure 2).

Because of the significant difference in the mean age between patients from group B and the other two groups, additional analysis of the correlation between the content of the necrotic core and the clinical characteristics of the patients was also performed. No significant correlation was found between the age of examined patients and the content of a necrotic core of the lesion (R=0.003; NS).

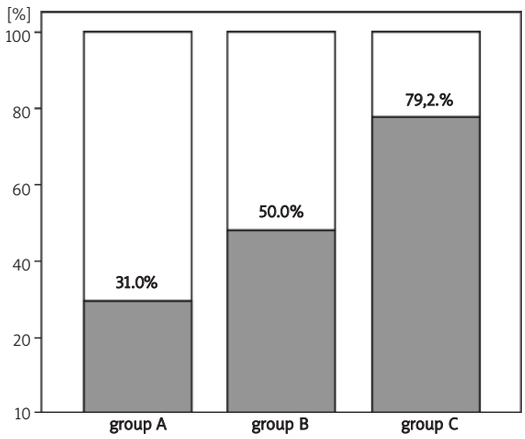

Figure 2. Lesions (in %) of TCFA morphology in examined groups of patients (p <0.01)

Discussion

Histopathological studies have demonstrated that TCFA lesions are responsible for the majority of ACS events. So far the only way to establish the prevalence of such lesions has been the microscopic analysis of tissues obtained during the autopsy of subjects who died of cardiovascular disease. Virtual histology is the first diagnostic tool allowing *in situ* detection of lesions of TCFA morphology. According to published data, such plaques may be present even in the absence of detectable abnormalities in coronary angiography.

In one of the first studies of Sano et al., conducted using ultrasonographic systems for tissue identification, the structure of atherosclerotic changes not visible in coronary angiography was analysed. In a group of 30 patients with confirmed CAD, 120 segments with stenosis not exceeding 10% were identified. In the case of 78 (65%) segments, an atherosclerotic lesion with a well developed necrotic core was observed, and in 5% of segments plaques of TCFA morphology were found [4]. The results of other studies demonstrated a variable prevalence of TCFA in patients with various forms of ischaemic heart disease. Rodriguez-Granillo et al. analysed a group of 55 patients with stable angina or ACS. They revealed that in subjects with ACS (either with ST-segment elevation or non-ST-segment elevation) lesions of TCFA morphology were observed three times more often than in patients with stable angina. In this report the diagnosed condition (stable angina vs. ACS) was the only significant predictor of TCFA occurrence [5].

In our study significant differences in the prevalence of TCFA lesion occurrence between examined groups of patients were seen. Thirty-one percent of all atherosclerotic changes identified in the group of subjects with stable angina could have been characterised as TCFA. The highest prevalence of such lesions was observed in subjects with ACS. Patients with a history of ACS, receiving aggressive pharmacotherapy for secondary prevention from the time of the ischaemic event, presented with medium prevalence of TCFA lesions. In the examined groups, however, there were no significant differences in the prevalence of FA lesions with well developed necrotic core and a fibrous cap.

Our analysis for the first time ever demonstrated differences of atherosclerotic plaque structure between patients with ACS and those with a history of ACS. One of the possible explanations of this observation may be a significantly higher rate of aggressive pharmacotherapy for the secondary prevention of cardiovascular events observed in patients with a history of recent ACS.

The results of multicentre randomised clinical trials indicate that intensive pharmacological treatment of patients after ACS leads to a significant decrease in the incidence of serious cardiovascular events in long-term follow-up. Of all medications used, HMG-CoA reductase inhibitors (statins) have been documented to have a significant efficacy; their administration reduces the incidence of MI and total mortality [6, 7]. So-called stabilisation of atherosclerotic plaque is considered to be one of the basic mechanisms of the positive effects of HMG-CoA reductase inhibitors in this population. Statins with strong lipid-lowering action can directly influence the progression and morphology of atherosclerotic lesions. Observations based on digital quantitative angiography indicate that the use of statins in patients with symptomatic CAD may lead to a significant decrease of plaque progression [8].

Most recent reports based on IVUS have demonstrated that statins used in high doses can result in almost total inhibition of progression and in some cases regression of atherosclerotic plaques [9, 10]. The studies using the IVUS technique indicated that lipid-lowering therapy can possibly result in alteration of the morphology of atherosclerotic plaque. Schartl et al. have demonstrated that the use of atorvastatin leads to an increase of the echogenicity of atherosclerotic lesions, which indirectly reflects the intense fibrotic process resulting in decreased plaque susceptibility to rupture and destabilisation [11].

Published reports provide data on the influence of lipid-lowering therapy on the structure of atherosclerotic lesions assessed in vivo. This issue has been analysed in two studies using the measurement of dispersed echoes intensity (integrated backscatter, IB-IVUS) which is an intracoronary ultrasonographic system fairly similar to IVUS-VH. Kawasaki et al. conducted IVUS analysis with histological assessment in a group of 52 patients who were subsequently randomised to therapy with pravastatin (20 mg/day), atorvastatin (20 mg/day) or dietary intervention only. The follow-up IVUS examination conducted after 6 months revealed a significant decrease of the amount of lipid content of atherosclerotic plaques in patients treated with both pravastatin and atorvastatin compared with atherosclerotic lesions in patients from the control group. Lipid-lowering therapy resulted in a significant increase of fibrous tissue content within plaques. It did not, however, significantly alter the size of the lesion or the degree of vessel occlusion [12]. A similar influence of therapy with atorvastatin on atherosclerotic lesion parameters was observed in Yokoyama et al.'s study of 50 patients. The lipid-lowering treatment resulted in additional decrease of the total volume of atherosclerotic plaque [13].

The presented data do not provide an answer to the question whether the difference in the prevalence of TCFA between patients who had undergone ACS and patients in the acute phase of ACS, observed in the presented study, resulted only from the use of intensive lipid--lowering treatment. Another possible explanation of this

observation may be the process of spontaneous healing of unstable atherosclerotic plaques, that has been observed in some studies using IVUS examination [14].

Because of the limited number of examined patients, lack of randomisation to prospectively defined therapeutic strategies and, most importantly, the inability to conduct follow-up ultrasonographic assessment in our study patients, finding answers to these questions was not possible. Furthermore, in the majority of randomised trials the reduction of the incidence of adverse cardiovascular events was observed only after 6 months of treatment. In the study presented here the mean time since ACS was around 2 months. It should be assumed that, in most cases, statin therapy was initiated at the time of ACS. The question is whether 2 months are sufficient to see the effect of beneficial remodelling of atherosclerotic plaque, which would be detected with the methods used in the study. Another factor with a potential influence on the results of the study was age difference observed between examined groups of patients. Statistical analysis did not confirm a direct influence of the age of examined subjects on the contents of the necrotic core within analysed lesions. Such an influence, however, cannot be completely excluded.

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Częstość występowania potencjalnie niestabilnych zmian miażdżycowych w populacji pacjentów z chorobą wieńcową – badanie z zastosowaniem wirtualnej histologii

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Streszczenie

Wstęp: Jak wynika z badań histopatologicznych, pęknięcie blaszki miażdżycowej jest najczęstszą przyczyną występowania ostrych zespołów wieńcowych (ACS). Blaszka miażdżycowa z cienką czapeczką łącznotkankową (ang. *thin-cap fibroatheroma*, TCFA) została zidentyfikowana jako zmiana charakteryzująca się wysokim ryzykiem destabilizacji, podczas gdy blaszka z zachowaną czapeczką łącznotkankową (ang. *fibroatheroma*, FA) uważana jest za zmianę o mniejszym ryzyku pęknięcia. Wirtualna histologia (ang. *intravascular ultrasound – virtual histology*, IVUS-VH) jest metodą diagnostyczną pozwalającą na identyfikację zmian o morfologii TCFA i FA w warunkach przyżyciowych. Dotychczas nie ma jednoznacznych danych dotyczących występowania tego typu zmian w tętnicach pacjentów z różnymi postaciami choroby wieńcowej.

Cel: Ustalenie częstości występowania zmian o morfologii TCFA i FA w tętnicach wieńcowych pacjentów ze stabilną chorobą wieńcową, chorych z ACS w wywiadzie i chorych w fazie ostrej ACS.

Metodyka: Badaniu metodą IVUS-VH poddano 60 chorych. Grupę A stanowiły osoby z rozpoznaną stabilną chorobą wieńcową bez wywiadu ACS w czasie ostatnich 12 mies. Do grupy B kwalifikowano chorych z przebytym ACS, który wystąpił w czasie ostatnich 4 tygodni do 3 mies. poprzedzających badanie. W grupie C znalazły się osoby poddawane badaniu w ostrej fazie ACS.

Wyniki: Ostatecznej analizie poddano 75 zmian miażdżycowych w tętnicach wieńcowych (grupa A n=29 zmian; grupa B n=22; grupa C n=24). Pomiędzy badanymi grupami nie stwierdzono różnic w charakterystyce angiograficznej i ultrasonograficznej. Nie odnotowano także istotnych różnic w częstości występowania zmian o morfologii FA (20,7 *vs* 7 *vs* 4,2%, odpowiednio grupa A, B i C; NS). Obserwowano istotną zależność pomiędzy rozpoznaniem klinicznym a występowaniem zmian o morfologii TCFA, największą częstość tego typu zmian stwierdzono w grupie C (31,0 *vs* 50,0 *vs* 79,2%, odpowiednio; p <0,01).

Wnioski: Częstość występowania zmian o morfologii TCFA różni się istotnie pomiędzy grupami chorych z różnymi postaciami choroby wieńcowej. W grupie osób po przebytym ACS stwierdza się znacząco mniejszą częstość występowania zmian o morfologii TCFA w porównaniu z grupą chorych w fazie ostrej ACS. Przyżyciowa identyfikacja zmian potencjalnie niestabilnych w grupie pacjentów po przebytym ACS może pozwolić na lepszą stratyfikację ryzyka wystąpienia niepożądanych zdarzeń sercowo-naczyniowych.

Słowa kluczowe: ostry zespół wieńcowy, blaszka z cienką czapeczką łącznotkankową, wirtualna histologia

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