

Myocardial ischaemia in systemic lupus erythematosus: detection and clinical relevance

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Abstract

Background: Severe cardiovascular complications are among the most important causes of mortality in systemic lupus erythematosus (SLE) patients.

Aim: To assess the usefulness of echocardiography, ECG, and coronary artery calcium scoring (CACS) in the detection of myocardial ischaemia in SLE patients compared to single photon emission computerised tomography (SPECT) and to assess their five-year follow-up.

Methods: In 50 consecutive SLE patients (mean age 39.2 ± 12.9 years, 90% female), clinical assessment, resting and exercise ECG and echocardiography, multidetector computed tomography — based CACS and SPECT studies (Tc-99m sestamibi) were performed. Patients were then followed for five years.

Results: SPECT revealed perfusion defects in 25 (50%) patients; persistent defects in 18 (36%) and exercise-induced defects in seven (14%) subjects. No typical ischaemic heart disease clinical symptoms, signs of ischaemia in resting ECG, or left ventricular contractility impairment in echocardiography were observed. Signs of ischaemia in exercise ECG were found in 17 (34%) patients. The CACS ranged from 1 to 843.2 (median 23.15), and coronary calcifications were observed in 12 (24%) patients. Compared to the SPECT study, exercise ECG had 68% sensitivity and 100% specificity in detecting myocardial ischaemia, while CACS had only 28% sensitivity and 58% specificity. During follow-up, one patient who showed myocardial perfusion defects and the highest calcium score (843.2) at baseline, developed CCS II class symptoms of myocardial ischaemia. Coronary angiography was not performed because of severe anaemia; the patient died three months later. In two other patients with perfusion defects and calcium deposits at baseline, CCS I class symptoms were observed; coronary angiography showed only thin calcified coronary plaques that were haemodynamically insignificant.

Conclusions: In about half of relatively young, mostly female, SLE patients, SPECT shows myocardial perfusion defects, with coronary calcifications present in one quarter of them. While ECG and echocardiography may not reveal any pathology, ECG exercise test can identify these patients with high specificity. In patients with a negative SPECT, the short-term prognosis is good, while in patients with perfusion defects and coronary calcifications, the clinical symptoms of myocardial ischaemia could occur. However, at a low calcium score (< 150), the short-term risk of significant atherosclerosis progression is low.

Key words: systemic lupus erythematosus, autoimmune diseases, coronary calcification, atherosclerosis, MDCT, CACS, SPECT

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a generalised autoimmune disease, in which diffuse, chronic inflammatory reaction plays an important pathogenic role. At the same time, the mortality of SLE patients is influenced mainly by an increased occurrence of severe cardiovascular (CV) complications, in which atherosclerosis plays a major role [1].

Non-invasive diagnostic techniques used to detect ischaemic heart disease include resting and exercise ECG, echocardiography, perfusion scintigraphy and coronary artery calcium scoring (CACS).

While resting ECG mainly documents previous myocardial infarction (Q waves), and resting echocardiography shows segmental left ventricular (LV) wall motion abnormalities only when $\geq 20\%$ of muscle thickness is not viable, stress tests have much higher sensitivity in detecting transient ischaemia in stable coronary artery disease (CAD). The ECG exercise test and exercise perfusion scintigraphy are the most commonly used. The sensitivity of ECG exercise test in detecting myocardial ischaemia is estimated at approximately 68% and specificity at 77% [2]. Technetium-99m sestamibi perfusion scintigraphy performed at rest and during exercise is judged to be the most sensitive (91–100%) and specific (71–100%) non-invasive method of myocardial ischaemia detection [3, 4]. In addition, perfusion scintigraphy might show microcirculation disturbances that are not evident in exercise ECG. Such pathology in microcirculation may occur in SLE together with classical atherosclerosis of the epicardial arteries. Coronary calcium score is a new way of assessing the presence of calcified atherosclerotic plaques [5, 6]. Increased coronary calcium score identifies subjects at high long-term cardiac risk [7].

An increased occurrence of CV complications in SLE [1] substantiates the need for early diagnostic evaluation of premature atherosclerosis and myocardial ischaemia in this group of patients. Therefore, this study was conducted to assess the usefulness of resting echocardiography, resting and exercise ECG, and CACS in the detection of myocardial ischaemia in SLE patients compared to single photon emission computerised tomography (SPECT) and to assess their five year follow-up.

METHODS

Patients

The study was performed in 50 consecutive out-patients treated for systemic SLE in our department. All patients fulfilled at least four classification criteria for SLE [8, 9] and all were in stable clinical condition (no need for immunosuppressive therapy intensification, i.e. current immunosuppressive drug dose increase or introduction of an additional immunosuppressive drug within the last three months). Clinical examination, rest ECG and echocardiography, ECG exercise test, multidetector computed tomography (MDCT) — based CACS and SPECT

studies were performed in all the patients. They were then observed for any clinical symptoms of ischaemic heart disease during five years of follow-up.

Echocardiography

The following parameters were determined by echocardiography (Toshiba Aplio SSA-770 Ultrasound System, Toshiba, Japan): diastolic and systolic dimensions; its segmental systolic function; LV ejection fraction (LVEF) (Simpson method); wall thickness in diastole; left atrium diameter; right ventricular diastolic diameter; ascending aorta diameter; E and A mitral inflow velocities; valvular pressure gradients; and regurgitation assessment. The thickness of the pericardium and the presence of pericardial effusion were also assessed.

SPECT

The SPECT study (ECAM Gamma Camera, Siemens, Germany) was performed at rest and during exercise in a two-day protocol. On the first day, at submaximal stress, a 25–40 mCi dose of Tc-99m sestamibi was injected (the individual dose was modified taking into account the patient's weight) and exercise continued for one additional minute after injection. The Tc-99m sestamibi SPECT imaging started 15–30 min later. On the second day, rest examinations were performed. The SPECT was performed using a circular 180° acquisition for 60 projections at 20 s per projection. Myocardial perfusion was assessed in 17 LV myocardial segments. The number of segments with persistent or exercise-induced perfusion defects was assessed by visual semiquantitative interpretation by two experienced observers, each unaware of the other's examination results. Each segment was scored using a five-point scoring system (0 = normal, 1 = equivocal, 2 = moderate, 3 = severe reduction of radioisotope uptake, 4 = apparent absence of detectable tracer uptake in a segment). A combined stress score (SSS) was obtained by adding the scores of the 17 segments of the stress images. Perfusion with SSS < 4 was considered normal. During exercise SPECT study, ECG was recorded continuously and inspected for the classical signs of ischaemia (horizontal or down-slope ST depression of ≥ 0.1 mV or T wave inversion).

Coronary calcium scoring

Coronary calcium scoring was performed using a MDCT imager (Somatom Definition, Siemens, Germany). The images were ECG triggered. The 3 mm-thick sections were obtained covering the whole heart. Coronary artery calcifications were defined as lesions with attenuation greater than 130 HU in more than four adjacent pixels. For quantification of coronary calcium, 3D Leonardo application (Siemens, Germany) was used. The number of atherosclerotic plaques, in particular coronary arteries and their volume, were assessed. An Agatston calcium score was calculated [10].

Table 1. Presence and types of autoantibodies and other laboratory parameters

	Range (mean \pm SD)	Patients with out-of-range levels
aCL IgG [RU/mL]	0.68–121.56 (15.6 \pm 25.9)	16 (32.0%)
aCL IgM [RU/mL]	1.62–52.93 (12.9 \pm 12.7)	21 (42.0%)
anti β 2GPI IgG [RU/mL]	0.16–95.33 (5.7 \pm 20.5)	6 (12.0%)
anti β 2GPI IgM [RU/mL]	0.21–21.66 (3.2 \pm 4.7)	19 (38.0%)
Lupus anticoagulant	Yes/No	14 (28.0%)
ANA [titer]	0–1/20480	45 (90.0%)
CRP [mg/L]	0.10–41.70 (4.0 \pm 8.9)	9 (18.0%)
C3c [g/L]	0.43–1.39 (0.92 \pm 0.23)	25 (50.0%)
C4 [g/L]	0.02–0.26 (0.13 \pm 0.07)	13 (26.0%)
Total cholesterol [mmol/L]	3.10–6.60 (4.56 \pm 0.87)	12 (24.0%)
LDL cholesterol [mmol/L]	1.12–4.23 (2.62 \pm 0.83)	12 (24.0%)
HDL cholesterol [mmol/L]	0.6–1.98 (1.39 \pm 0.32)	5 (10.0%)
Triglycerides [mmol/L]	0.35–2.56 (1.22 \pm 0.52)	7 (14%)
Glucose [mmol/L]	3.5–5.5 (4.44 \pm 0.47)	0 (0%)

Elevated value for CRP > 5 mg/L; decreased values for C3c < 0.9 g/L, for C4 < 0.1 g/L; elevated values for total cholesterol > 5.0 mmol/L, LDL cholesterol > 3.0 mmol/L, triglycerides > 1.7 mmol/L, glucose > 6.1 mmol/L; decrease value for HDL cholesterol < 1.0 mmol/L for males, < 1.2 mmol/L for females; aCL — anticardiolipin antibodies (cut-off value for IgG > 20 RU/mL, for IgM > 30 RU/mL; see methods); anti β 2GPI — anti β 2-glycoprotein I antibodies (cut-off value for IgG > 3 RU/mL, for IgM > 2.6 RU/mL; see methods); ANA — antinuclear antibodies; CRP — C-reactive protein

Laboratory tests

Beside standard tests, laboratory evaluation included serum concentrations of high-sensitivity C-reactive protein and complementary C3c and C4 components by nephelometry (Siemens, Germany). Serum levels of anticardiolipin (aCL) and anti β 2GPI antibodies (of both IgG and IgM classes) were measured using a home-made ELISA with Sapporo standard for anti β 2GPI antibody measurements (HCAL for IgG, EY2C9 for IgM) [11]. Values exceeding the 99th percentile of a healthy population sample were considered positive. Lupus anticoagulant was determined in accordance with the three-step procedure recommended by the International Society on Thrombosis and Haemostasis [12].

Statistical analysis

Statistical analysis was performed using Statistica 6.0 Sigma software. All numerical data are expressed as mean values \pm SD or as proportions. Continuous variables were compared by the use of Student *t*-test. The χ^2 test was used to examine differences in proportions. The level for statistical significance was predetermined at *p* value < 0.05.

Before the study, informed consent was obtained from each patient. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki. The study was approved by the Ethical Committee of the Jagiellonian University in Krakow.

RESULTS

Of 50 examined patients, 45 (90%) were female, aged 18–67 years (mean age 39.2 years). Duration of the disease at the

time of examination varied between one and 11 years (mean 6.0 years). Three patients had been previously diagnosed with antiphospholipid syndrome (APS) based on the revised APS classification criteria [13]. One of these three suffered from an objectively confirmed pulmonary embolism. There were two tobacco smokers; none of the patients was obese. History revealed arterial hypertension in four subjects. The ECG recordings were normal in all patients.

The results of laboratory tests are shown in Table 1. Peripheral blood count, serum sodium, potassium, glucose, creatinine and urinalysis were all normal. Hypercholesterolaemia was found in 12 (24%) patients. The SLEDAI score [14] ranged from 0 to 12 (median 4). Immunosuppressive treatment included methylprednisolone in 26 (52.0%) subjects, prednisone in two (4.0%), chloroquine derivate in four (8.0%), azathioprine in three (6.0%), cyclophosphamide in two (4.0%), and methotrexate in two (4.0%). The remaining patients had not used any immunosuppressive drugs during the last 12 months of observation. Other treatments included angiotensin converting enzyme inhibitors in three (6.0%) subjects, beta-blockers in two (4.0%), and calcium channel blockers in two (4.0%).

Echocardiographic examination revealed normal LVEF in all patients, and normal LV diameters in all but two patients. Segmental wall motion of LV was also normal in all patients. However, mitral inflow E/A ratio showed diastolic LV dysfunction in 13 (26.0%) patients: in four, pseudonormal inflow pattern was observed, indicating advanced diastolic dysfunction, while nine patients showed a significantly decreased E/A ratio (*E/A* < 1), reflecting relaxation impairment. Diastolic LV dysfunction was present despite normal LV mu-

Table 2. Echocardiographic data

	Value (mean \pm SD)	Patients with pathology
LVEDD [mm]	39–57 (45.4 \pm 4.7)	2 (4.0%)
LVESD [mm]	23–47 (32.0 \pm 5.2)	0 (0%)
LVEF [%]	58–75 (64.5 \pm 4.4)	0 (0%)
E/A	0.63–2.17	13 (26.0%)
IVS [mm]	6–10 (8.8 \pm 1.1)	0 (0%)
PW [mm]	7–11 (8.8 \pm 1.2)	0 (0%)
LA [mm]	26–48 (32 \pm 5.4)	6 (12%)
AA [mm]	22–36 (31 \pm 4.3)	0 (0%)
RV [mm]	19–35 (24.8 \pm 3.8)	8 (16%)
RVSP > 30 mm Hg	35–55 (39.3 \pm 7.1)*	8 (16%)

*In patients with RVSP > 30 mm Hg; out-of-range values: LVEDD > 56 mm, IVS or PW > 12 mm, LA > 40 mm, AA > 36 mm, RV > 30 mm; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; LVEF — left ventricular ejection fraction; E — early diastolic mitral inflow; A — mitral inflow during atrial contraction; IVS — interventricular septum; PW — posterior wall of the left ventricle; LA — left atrium; AA — ascending aorta; RV — right ventricle; RVSP — RV systolic pressure

sple thickness in all patients. In spite of the absence of clinically significant valvular stenosis, mitral and/or aortic leaflet thickening was observed in 31 (62.0%) patients. Clinically relevant mitral regurgitation ($\geq 2^{\text{nd}}$ degree) was present in only one patient. Moreover, pericardial thickening with pericardial effusion (2–12 mm) was frequent in this group (43.3% of patients). Left ventricular diastolic dysfunction and mitral insufficiency resulted in the dilation of the left atrium in six (12.0%) patients. Aortic diameters were within the normal range. Right ventricular systolic pressure was elevated (> 30 mm Hg) and right ventricles dilated in eight (16.0%) patients. All numerical data are shown in Table 2.

The SPECT or CACS studies showed abnormalities in 30 (60.0%) patients. The ECG exercise test revealed horizontal or down-slope ST depression of ≥ 0.1 mV in 17 (34%) patients. The patients with SPECT, CACS or exercise ECG abnormalities did not differ from subjects without these pathologic findings in terms of their age, gender or disease duration. No significant correlation was found between LV function assessed by rest echocardiography and SPECT, CACS or ECG exercise test results.

The SPECT study revealed myocardial perfusion abnormalities in 25 (50.0%) patients: persistent defects in 18 (36.0%) patients, exercise-induced defects in seven (14.0%). In patients with abnormal perfusion (SSS ≥ 4), SSS ranged from 4 to 10 (median 6). Mild perfusion defects (SSS 4–8) were observed in 22 (88%) patients, moderately abnormal defects (SSS 9–13) in three (12%). The distribution of the numbers of persistently or exercise-induced underperfused LV myocardial segments is shown in Table 3.

Coronary calcifications were present in 12 (24%) patients. The number of atherosclerotic calcified plaques ranged from 1 to 23 (median 4), its volume 2 — 761.8 (median 23.6) mm³. Calcium scores ranged from 1 to 843.2 (median 23.15).

Table 3. Distribution of numbers of underperfused left ventricular myocardial segments in systemic lupus erythematosus patients studied (SPECT study)

No. of underperfused myocardial segments	Patients with perfusion defects	
	Persistent	Exercise-induced
0	32 (64%)	43 (86%)
1	—	2 (4%)
2	6 (12%)	—
3	11 (22%)	4 (8%)
4	—	1 (2%)
5	1 (2%)	—

Table 4. Distribution of perfusion abnormalities (SPECT) and coronary artery calcifications (CACS) in individual coronary arteries

Coronary artery	Patients with perfusion abnormalities (SPECT)	Patients with coronary artery calcifications (CACS)
LAD	17 (68%)	6 (49%)
RCA	3 (12%)	2 (17%)
LAD + RCA	5 (20%)	2 (17%)
LAD + Cx + RCA	—	2 (17%)

LAD — left anterior descending artery; RCA — right coronary artery; Cx — circumflex artery

The distribution of perfusion abnormalities and coronary calcifications among the particular coronary arteries is shown in Table 4.

All patients with positive ECG exercise tests showed myocardial perfusion defects in SPECT study. Myocardial perfusion abnormalities and coronary calcifications were both present in seven (14%) patients. In 18 (36%) patients, SPECT abnormalities were isolated (calcium score = 0). On the other hand, in five (10%) patients with mild calcium deposits (1–3 plaques, calcium score 4.4–35.1, median 11), no SPECT abnormalities were detected.

The SPECT results correlated with SLE activity: mean SLEDAI score in patients positive in SPECT was 6.16 ± 5.03 as compared with patients with normal perfusion (SLEDAI 3.79 ± 2.06 , $p = 0.04$).

The number of patients with elevated levels of total and LDL cholesterol was comparable in the group with pathologic results of SPECT or CACS ($n = 6$ out of 30; 20%) and in the group with normal SPECT or CACS results ($n = 6$ out of 20; 30%, NS).

Compared to the SPECT study, the ECG exercise test had 68% sensitivity and 100% specificity in the diagnosis of myocardial ischaemia, while CACS showed only 28% sensitivity and 58% specificity.

During five years of follow-up, clinical symptoms of myocardial ischaemia developed in three (6%) patients (SLEDAI score at inclusion 6–10, median 8, APS diagnosed in one of them). In one patient who initially showed myocardial perfusion defects (in three myocardial segments, SSS = 9) and highest observed calcium score (843.2), the CCS II class symptoms of myocardial ischaemia were observed. This patient was referred for coronary angiography, however the examination was not performed due to the onset of severe anaemia. The patient died three months later. In the two other patients who also previously showed perfusion defects and calcium deposits, CCS I class symptoms developed; coronary angiography was performed and showed haemodynamically insignificant thin wall calcified plaques. All the remaining 47 patients showed no clinical signs of myocardial ischaemia during the five-year follow-up.

Mean value of the SLEDAI score remained unchanged during the follow-up period. The SLE flare (SLEDAI increase ≥ 3) was observed in five patients: in three due to the onset of the clinical symptoms of myocardial ischaemia, and in two due to the onset of haematuria.

DISCUSSION

In the general population, the detection of subclinical CAD helps to identify individuals at high risk of future CV events [15]. However, the best diagnostic method of subclinical CAD detection in SLE patients has not been yet identified. Coronary angiography is an invasive method and cannot be used for screening. The ECG exercise test gives insufficient data due to low sensitivity (68%) and specificity (77%): approximately 32% of subjects with ischaemia are missed by this method [2]. Accordingly, ECG exercise test was found to be

insufficient to diagnose CAD in 22 SLE females [16]. Similarly, LV wall motion abnormalities are visible in resting echocardiography only in survivors of myocardial infarction if at least 20% of muscle thickness is not viable.

Previous studies have demonstrated the high prognostic value of myocardial perfusion scintigraphy in patients with CAD [3, 17]. This non-invasive tool shows excellent sensitivity (91–100%) for the detection of ischaemia [3, 4]. For this reason, we compared several non-invasive diagnostic techniques for coronary disease detection in SLE patients with SPECT as a reference method.

Our study supports previously published data showing high frequency of myocardial perfusion defects detected by SPECT in SLE patients [18–21]. We saw these defects in 50% of patients, despite normal resting ECG recordings and the lack of any myocardial ischaemia clinical symptoms or LV wall motion abnormalities. Perfusion abnormalities were predominantly observed both at rest and during exercise, indicating persistent myocardial injury. In most of the patients, the number of underperfused LV segments was low (Table 3) and showed mildly abnormal perfusion (SSS = 4–8). It is, however, well established that the presence of even small perfusion defects by SPECT strongly affects prognosis [22, 23]. Of our 25 patients with perfusion defects, only in 17 (68%) did ECG exercise test show myocardial ischaemia, thereby confirming the low sensitivity of exercise ECG for CAD detection. Rest ECG and LV segmental wall motion evaluation by echocardiography were even less sensitive, showing no pathology at all.

Besides the presence of myocardial perfusion defects, 24% of our asymptomatic SLE patients manifested atherosclerosis in coronary arteries by MDCT. Coronary vessels are most frequently affected by calcifications. In a recently published study of 50 SLE patients [24], the frequency of atherosclerotic plaques observed in MDCT was highest in the coronary arteries (42% of patients with calcifications), followed by the carotid arteries (24% of patients with calcifications). Calcium scoring has been studied extensively over the past decade for predicting outcome in generally asymptomatic subjects at intermediate clinical risk for CAD [5, 6]. It has been shown that coronary calcium deposits provide independent prediction for short- and long-term cardiac events [7, 25, 26].

Even in patients with normal SPECT results, increased CACS identifies subjects at high long-term cardiac risk [7]. A CACS of 0 identifies a very low risk cohort, even among clinically high-risk groups, such as diabetic patients [27].

The factors that cause myocardial perfusion defects and premature atherosclerosis in SLE patients are not clearly defined. Interestingly, in our study hypercholesterolaemia did not differentiate SLE patients with and without coronary calcifications or myocardial perfusion defects. It has been suggested that antiphospholipid autoantibodies accompanying

SLE may promote thrombotic events in coronary circulation and foster the development of atherosclerosis [1]. Thrombi formed in the coronary microcirculation might lead to perfusion defects in small regions of the myocardium. Such small defects, localised predominantly in the segments supplied by the left anterior descending artery, were observed in our study. On the other hand, we never observed a pattern characteristic for classical CAD (closed epicardial artery), manifested by larger, sharp-edged defects in the supplied region.

Coronary circulation disturbances due to microthrombosis would also be in line with the differences between our SPECT and CACS results. Of 25 patients with myocardial perfusion defects, coronary calcified plaques were present in only seven (28%). Thus, most of the perfusion defects observed in the SPECT study (18 [72%] patients) were not related to classical atherosclerotic lesions. Interestingly, a single study in SLE patients after myocardial infarction showed in most cases angiographically normal coronary arteries [28]. While an intracoronary thrombus may dissolve in time, classical coronary calcified plaques persist and should be detected during coronary angiography. Thus, it is possible that SPECT shows myocardial ischaemia caused rather by SLE-specific factors, such as thrombosis, that cannot be detected by CACS. Besides microthrombosis, the other factor that may influence myocardial perfusion abnormalities in SLE is vasculitis caused by circulating immunologic complexes.

During five years of follow-up, three (6%) patients manifested clinical symptoms of myocardial ischaemia. Such a low incidence of clinically significant myocardial ischaemia may be explained by the low CACS seen in our group. Significant worsening of prognosis related to increased number of cardiac events (up to > 50% of patients during a 12-years observation) has been described in subjects with a calcium score > 400 [7], while the median calcium score in our group was 23.15. Still, in all three patients with the onset of clinical symptoms during follow-up, SPECT and CACS performed at inclusion already showed perfusion defects and calcium deposits in coronary arteries. The single patient with CCS II class symptoms who died during the observation period had the highest observed calcium score (843.2) and SPECT perfusion defects in three myocardial segments with SSS = 9. In both patients with the CCS I class symptoms, coronary angiography showed thin wall calcified lesions. In keeping with our findings, data from the American LUMINA cohort [29] identified the presence of CV disease (endocarditis, angina, conduction defects, congestive heart failure) in only 6.8% of SLE patients.

Despite the low incidence of symptomatic CAD in our study, the prognostic importance of SPECT and CACS examinations must be emphasised. Although in one study of eight SLE subjects, positive in SPECT, coronary angiography showed normal epicardial vessels [20], in another study atherosclerotic plaques were documented in 38% of patients with

myocardial perfusion defects [4]. Results obtained in a group of 1,126 apparently healthy subjects showed that SPECT and CACS examinations are independent and complementary predictors of short- and long-term prognosis, and that the integration of CACS results with those of SPECT could improve risk prediction in a group of generally asymptomatic patients without clinically apparent CAD [7]. To some extent, our study corroborates those results showing that ischaemic heart disease symptoms occur in patients with both SPECT and CACS abnormalities.

CONCLUSIONS

In about half of relatively young, mostly female, SLE patients SPECT shows myocardial perfusion defects, with coronary calcifications present in one quarter of them. While resting ECG and echocardiography may not reveal any pathology, ECG exercise test is able to identify patients with myocardial perfusion defects with 68% sensitivity and 100% specificity. In patients with negative SPECT, the short-term prognosis is good, while in patients with perfusion defects and coronary calcifications, the clinical symptoms of myocardial ischaemia could appear. However, at low calcium score (< 150), the short-term risk of significant atherosclerosis progression is low.

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Niedokrwienie miokardium u chorych z toczniem rumieniowatym układowym: metody detekcji i znaczenie kliniczne

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Streszczenie

Wstęp: Powikłania sercowo-naczyniowe stanowią obecnie główną przyczynę śmiertelności u chorych z toczniem rumieniowatym układowym (SLE).

Cel: Celem pracy była ocena przydatności i znaczenia rokowniczego spoczynkowego badania echokardiograficznego, spoczynkowego i wysiłkowego badania EKG oraz wskaźnika uwapnienia tętnic wieńcowych (CACS) metodą wielorzędowej tomografii komputerowej w wykrywaniu niedokrwienia mięśnia sercowego u osób z SLE w porównaniu z wynikami scyntygrafii perfuzyjnej serca (SPECT) w obserwacji 5-letniej.

Metody: U 50 chorych z SLE (średni wiek $39,2 \pm 12,9$ roku; 90% kobiet) wykonano badanie kliniczne, spoczynkowe i wysiłkowe badanie EKG, CACS i SPECT. Chorych poddano 5-letniej obserwacji.

Wyniki: Badanie SPECT uwidoczniło zaburzenia perfuzji miokardium u 25 (50%) pacjentów: trwale ubytki perfuzji u 18 (36%), a wysiłkowe u 7 (14%) chorych. U żadnego pacjenta nie stwierdzono objawów choroby niedokrwiennej serca, zmian niedokrwienych w spoczynkowym EKG lub zaburzeń kurczliwości odcinkowej lewej komory. Podczas elektrokardiograficznego testu wysiłkowego zmiany niedokrwienne zaobserwowano u 17 (34%) chorych. Wartość CACS wynosiła 1–843,2 (mediana 23,15), zwapnienia w tętnicach wieńcowych były obecne u 12 (24%) chorych. W porównaniu ze SPECT elektrokardiograficzny test wysiłkowy charakteryzował się 68-procentową czułością i 100-procentową swoistością w wykrywaniu niedokrwienia miokardium, podczas gdy CACS jedynie 28-procentową czułością i 58-procentową swoistością. Podczas 5-letniej obserwacji u 1 chorego, u którego wyjściowo obserwowano zaburzenia perfuzji mięśnia sercowego oraz najwyższy wskaźnik uwapnienia (843,2), wystąpiły objawy choroby niedokrwiennej serca w II klasie wg CCS. Ze względu na ciężką anemię nie wykonywano koronarografii; pacjent zmarł 3 miesiące później. U innych 2 osób z zaburzeniami perfuzji i zwapnieniami tętnic wieńcowych wystąpiły objawy niedokrwienia w I klasie wg CCS. Badania koronarograficzne wykonane u tych chorych wykazały obecność przyściennych zmian miażdżycowych nieistotnych hemodynamicznie.

Wnioski: W grupie młodych bezobjawowych pacjentów z SLE badanie SPECT wykazuje zaburzenia perfuzji miokardium w 50% przypadków. Podczas gdy spoczynkowe badanie EKG i spoczynkowe badanie echokardiograficzne mogą nie wykazać żadnej patologii, elektrokardiograficzny test wysiłkowy identyfikuje tych chorych z 68-procentową czułością i 100-procentową swoistością. Zwapnienia tętnic wieńcowych uwidoczniono u 1/4 chorych z SLE. Pięcioletnie rokowanie u pacjentów z prawidłowym wynikiem badania SPECT jest dobre, natomiast u osób z zaburzeniami perfuzji i zwapnieniami tętnic wieńcowych mogą wystąpić objawy choroby wieńcowej. U pacjentów z niskim wskaźnikiem uwapnienia tętnic (< 150) ryzyko istotnej klinicznie progresji miażdżycy w 5-letniej obserwacji jest jednak małe.

Słowa kluczowe: toczń rumieniowat układowy, autoimmunizacja, zwapnienia tętnic wieńcowych, miażdżycy, MDCT, CACS, SPECT

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