Comparison of the exercise treadmill test and 24-hour ECG Holter monitoring in patients with syndrome X or coronary atherosclerosis

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Abstract

Background: Typical chest pain and ECG changes suggest the presence of myocardial ischaemia in cardiac syndrome X (SX) patients and resemble the symptoms observed in subjects with atherosclerotic coronary artery disease (CAD).

Aim: To compare the results of exercise treadmill tests (ETT), 24-hour ECG recordings and echocardiography in SX and CAD patients without previous myocardial infarction with the presence of significant lumen stenosis in one (CA1), two (CA2) or three (CA3) coronary arteries.

Methods: Two hundred six patients were included in the study: 43 SX (28 female), 49 CA1 (11 female), 51 CA2 (7 female) and 63 CA3 patients (8 female) all of whom underwent ETT according to the Bruce protocol, 24-hour ECG recordings and echocardiography.

Results: SX patients had median ST-segment depression during ETT comparable to that in CA1 and CA2 patients but significantly less than the CA3 subjects (p=0.024). Median time to ST depression of at least 1 mm, as well as median time of exercise, was significantly longer in SX individuals than in all CAD patients. The post-exercise recovery time of ST-segment changes was significantly longer in SX patients than in the CA1 group (p=0.006), comparable to that in CA2 subjects and shorter than that in CA3 individuals (p=0.003). Both the maximal ST-segment depression and the duration of significant ST-segment depression in Holter ECG recordings were significantly higher in SX patients than in CA1 subjects, were comparable to the values observed in the CA2 group and significantly lower than in CA3 individuals. The heart rate variability parameters (SDNN and pNN50) were significantly higher in SX patients than in CAD subjects. Patients with SX had a significantly thinner interventricular septum and smaller left ventricular end-diastolic cavity dimension than individuals from the CA1, CA2 and CA3 groups. There were no significant differences in the left ventricular ejection fraction or the thickness of the left ventricular posterior wall between SX patients and CAD patients.

Conclusions: Analysis of the ST segment in SX patients suggests the presence of advanced CAD. However, SX patients have better heart rate variability and exercise performance than patients with CAD.

Key words: syndrome X, coronary artery disease, exercise test, ECG Holter monitoring

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Introduction

Syndrome X (SX) is a condition encompassing angina, or angina-like, discomfort with ST-segment depression on exercise testing, suggestive of myocardial ischaemia despite normal coronary arteriograms [1-3]. Patients with SX experience chest pain similar to that found in individuals with atherosclerotic coronary artery disease (CAD). The presence of ST-segment changes and negative T-waves is reported at rest and/or exercise in both SX and CAD patients. These changes are usually very similar and are of comparable depth and duration during, and after an angina attack, and are present in many ECG leads [2-4].

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Although SX is predominantly seen in postmenopausal women, it also affects male patients [2]. There are many conflicting theories regarding the origin of the clinical findings in SX subjects, including augmented sympathetic drive and/or decreased parasympathetic activity, generalised vascular and endothelial dysfunction, gastroesophageal reflux or altered somatic and visceral pain perception [5-12]. Other causes of the symptoms include an abnormal myocardial sensitivity to adenosine and/or increased potassium release after exercise [13, 14]. True myocardial ischaemia has also been proposed as a trigger of the symptoms, whereas some data show that it is not a common phenomenon in SX patients [2-4, 15-18].

In contrast to CAD patients, the clinical course and prognosis in SX subjects is usually good [2, 3] although not as benign as commonly thought [15]. Although considerable similarity in the clinical symptoms and the results of non-invasive tests have been reported in both groups, SX individuals may vary in their clinical presentation when compared with patients with atherosclerotic CAD [2, 3, 7, 15, 19].

In this study, we compared the results of exercise treadmill tests (ETT), 24-hour ECG recordings and echocardiography in SX individuals and in CAD patients having significant lumen stenosis in one (CA1), two (CA2) or three (CA3) coronary arteries and no previous myocardial infarction (MI).

Methods

Patients

This is a retrospective study of consecutive patients admitted for elective coronary angiography and fulfilling the following criteria:

1. No previous MI.
2. Stable angina pectoris.
3. Not taking statin or fibrate agents.
4. Having performed an ETT according to the Bruce protocol (see details below) before coronary angiography.
5. Having performed a 24-hour ECG Holter recording (see details below) before coronary angiography.
6. Having performed resting trans-thoracic echocardiography before coronary angiography.

Cardiac syndrome X

From a group of patients with typical effort angina, at least 0.1 mV ST segment depression in at least 2 consecutive leads during ETT and normal coronary angiograms, we excluded those with diabetes mellitus, thyroid dysfunction, systemic hypertension, coronary artery spasm (excluded by the use of a hyperventilation test [20]), myocardial bridging, left ventricular (LV) hypertrophy, mitral valve prolapse, any structural heart disease and LV wall motion abnormality detected by echocardiography, and those with an abnormal resting ECG interfering with the interpretation of ST segments during ETT or ECG Holter analysis (pre-excitation syndrome, bundle branch block or hemiblock). The final SX group therefore consisted of 43 patients fulfilling all the criteria listed above.

Atherosclerotic coronary artery disease

Only patients with ≥50% lumen stenosis of any coronary artery were accepted into the CAD group. Those with thyroid dysfunction, coronary artery spasm, myocardial bridging and an abnormal resting ECG interfering with the interpretation of ST segments during ETT or ECG Holter analysis (pre-excitation syndrome, bundle branch block or hemiblock) were excluded. The final CAD group consisted of 163 subjects of whom 49 were CA1 subjects, 51 – CA2 and 63 – CA3.

Exercise Treadmill Test

The ETT was performed according to the Bruce protocol, with continuous ECG monitoring (Track Master--Mortara, USA). A resting 12-lead ECG was printed before starting the ETT and then continuously recorded. An ECG print-out was obtained whenever any abnormality was detected and always at the peak of exercise and after the ETT was finished. Finally, automatically generated ECG contained the full recording, together with printouts of each minute during exercise and recovery, plus the data when significant ST-segment depression of 1 mm (0.1 mV) in ≥2 adjacent leads and the maximal ST-segment depression occurred. In addition, an ECG was printed whenever chest pain occurred. Recovery time of ≥2 minutes was usually allowed if no ST-segment changes were present or exercise-evoked ST changes disappeared during this time. In other cases the recovery phase was terminated when ST-segment normalisation was achieved and/or the heart rate slowed to <100 beats/minute.

Blood pressure (BP) was measured by the Korotkoff method using a mercury cuff sphygmomanometer. The BP measurements were taken at rest and during the last minute of each exercise stage, including recovery. The ETT was terminated either because of the patient's request to stop, because of moderate to severe angina limiting exercise continuation, deep ST-segment depression (≥2 mm in at least 2 leads), complex ventricular arrhythmia (ventricular bigeminy, ventricular runs or ventricular tachycardia), new bundle branch block, physical exhaustion or any other disabling symptom (intermittent claudication, dizziness or dyspnoea). The ETT was also discontinued if no abnormal signs and symptoms were present but the patient's heart rate (HR) exceeded 95% of the age-adjusted limit.

The ETT was defined as positive when ≥1 mm ST-segment depression was observed in ≥2 leads corresponding to any myocardial wall. During the ETT, the following parameters were monitored and measured: time to 1 mm ST-segment depression, duration of exercise, total obtained workload, time to ST normalisation, systolic and diastolic BP, and HR before and during the test and following recovery. No drugs, except short lasting nitrates for pain relief, were allowed during the 24 hours before the ETT and patients were instructed not to eat or drink for at least two hours prior to the test. The ETT was performed from 1 day to 3 months before coronary angiography.

24-hour ECG Holter monitoring

A 24-hour continuous ECG monitoring was performed with a three-channel cassette recorder (Premier 3.3, DRG, USA). Heart rate variability (HRV) was measured as standard deviations of normal-to-normal RR intervals (SDNN) and percentage of successive RR intervals varying >50 ms (pNN50) according to published data [21]. Additionally, the mean values of 24-hour heart rate (HR24), the maximal depth of ST-segment depression (ST_{24max}) and the total duration of significant ST-segment depression (ST_{24max}) were analysed. The ECG Holter recording was performed from 4 days to 3 months before coronary angiography.

Echocardiography

Echocardiography was performed with a commercially available ultrasound system (Sonos 2500, Hawlett Packard, Andover, Massachusetts, USA) equipped with a 2.5 MHz transducer. Left ventricular end-diastolic (LVEDd) and end-systolic (LVEDs) cavity dimensions, as well as interventricular septal wall thickness (IVSd) and LV posterior wall thickness (LVPWTd) were all measured. The LV ejection fraction (LVEF) was calculated using the Teichholz formula.

Bioethics

The study protocol was approved by the Local Ethical Committee and informed consent was obtained from all participating patients.

Statistical analysis

The Kolmogorov-Smirnoff test revealed that some of the analysed variables did not have a normal distribution. Values of continuous data are given as median and interquartile range (IQR), whereas categorised variables are presented as numbers of patients. To compare the SX patients with each subgroup of CAD patients, the nonparametric Mann-Whitney test for unpaired data was used for analysis of continuous data. The exact Fisher test was used for the analysis of dichotomised data. A p value <0.05 was considered significant. All statistical analyses were performed using SPSS 7.0 for Windows (SPSS, USA) and the figures were made using GraphPad Prism 4.0 for Windows (GraphPad Software, USA).

Results

The clinical characteristics of SX and CAD patients with one-, two- or three-vessel disease are presented in Table I.

Table I. Clinical characteristics of patients with SX and CAD

Parameter	SX patients n=43	CA1 n=49	p value SX vs. CA1	CA2 n=51	p value SX vs. CA2	CA3 n=63	p value SX vs. CA3
Female gender [n (%)]	28 (65.1)	11 (224)	NS	7 (13.7)	0.026	8 (12.7)	0.009
Smokers [n (%)]	17 (39.5)	30 (61.2)	NS	37 (72.5)	0.002	40 (63.5)	0.018
Contractile dysfunction [n (%)]	0 (0)	3 (6.1)	NA	6 (11.8)	NA	16 (25.4)	NA
Diabetes mellitus [n (%)]	0 (0)	5 (10.2)	NA	3 (5.9)	NA	9 (14.3)	NA
Arterial hypertension [n (%)]	0 (0)	22 (44.9)	NA	31 (60.8)	NA	32 (50.8)	NA
Age [years]	44 (41-49)	54 (47-59)	<0.001	53 (50-59)	<0.001	54 (48-61)	<0.001
BMI [kg/m^2]	25 (23-28)	26 (25-28)	NS	28 (26-29)	0.005	28 (26-31)	0.001
Total cholesterol [mg/dl]	210 (192-235)	238 (218-264)	<0.001	248 (227-277)	<0.001	256 (235-291)	<0.001
HDL cholesterol [mg/dl]	42 (36-52)	40 (35-47)	NS	39 (34-44)	0.036	39 (33-44)	0.021
LDL cholesterol [mg/dl]	137 (120-157)	167 (157-189)	<0.001	186 (159-206)	<0.001	194 (167-227)	<0.001
Triglycerides [mg/dl]	126 (84-179)	179 (149-205)	<0.001	172 (146-205)	<0.001	192 (149-258)	<0.001

Abbreviations: SX – syndrome X, CA1 – one vessel coronary disease, CA2 – two vessel coronary disease, CA3 – three vessel coronary disease, BMI – body mass index, HDL – high-density lipoprotein, LD – low-density lipoprotein, NA – results of Fisher exact test not available as there are no cases in SX group

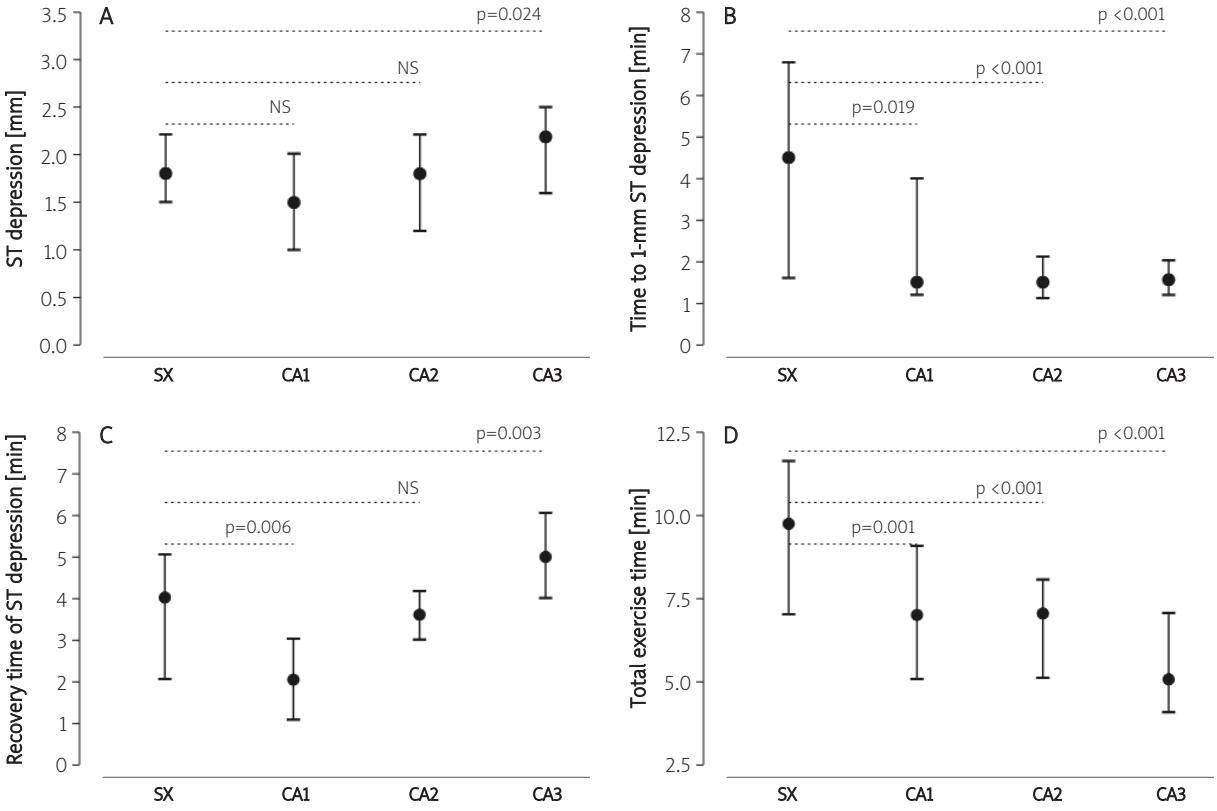

Figure 1. Results of exercise treadmill test. Panel **A** shows maximal ST-segment depression observed during ETT. Panel **B** presents the time to significant 1 mm ST-segment depression during ETT. Panel **C** shows the post-exercise recovery time of ST-segment changes. Panel **D** presents the total exercise time during ETT. For abbreviations see text. Median and interquartile ranges are shown in all figures

There were no significant differences between the SX and CA1 patients in terms of gender and smoking behaviour. However, SX patients were more commonly female and non-smokers than CA2 and CA3 individuals. Due to the exclusion criteria used in the selection of SX patients, there was no contractile dysfunction, diabetes or hypertension among them in contrast to CAD subjects. The SX patients were younger and had lower total and LDL cholesterol and triglyceride levels than all CAD patients. Although there was no significant difference in BMI or HDL cholesterol between SX and CA1 patients, SX individuals had lower BMI and higher HDL cholesterol concentrations than CA2 and CA3 patients, which appears to be secondary to the female prevalence in the SX group.

The results of ETT and HRV from the 24-hour ECG recordings are shown in Table II and Figure 1. During ETT, patients with SX presented significantly higher resting and peak exercise heart rates, lower resting and peak exercise systolic BP readings, longer time to ST-segment depression of 1 mm in at least 2 leads and longer total ETT time than all CAD subjects. The total obtained workload during exercise was significantly higher in SX individuals than in all CAD patients. The maximal depth of ST-segment depression was significantly lower in SX than in CA3 patients but did not differ significantly from that in the CA1 and CA2 groups. The time needed to normalise ST-segment depression during post-exercise recovery was significantly longer in SX patients than in CA1 subjects, did not differ from that in the CA2 group and was significantly shorter than in CA3 individuals.

There was no significant difference in the mean HR in 24-hour ECG recordings between the analysed groups. Both SDNN and pNN50 were significantly higher in SX patients than in all those with CAD (Table II). Both maximal ST-segment depression and duration of significant ST-segment depression in Holter ECG recordings were significantly higher in SC patients than in CA1 subjects, were comparable to the values observed in the CA2 group and significantly lower than in CA3 individuals. Besides, significant ST-segment depression was found in 32 (74.4%) SX patients, 19 (38.8%) CA1

Table II. Results of ETT and 24-hour ECG recordings in SX and CAD patients with disease involving one- (CA2), two- (CA2) and three- (CA3) vessels

	SX	CA1	p for SX vs. CA1	CA2	p for SX vs. CA2	CA3	p for SX vs. CA3
HR_{rest} [beats/min]	96 (86-107)	80 (73-84)	<0.001	82 (76-91)	<0.001	82 (77-88)	<0.001
HR_{peak} [beats/min]	148 (141-157)	134 (125-144)	<0.001	132 (120-142)	<0.001	124 (118-134)	<0.001
SBP_{rest} [mm Hg]	115 (110-120)	120 (115-140)	0.002	125 (115-140)	0.001	130 (115-140)	<0.001
SBP_{peak} [mm Hg]	145 (140-160)	175 (160-190)	<0.001	170 (160-185)	<0.001	170 (155-180)	<0.001
ST_{max} [mm]	1.8 (1.5-2.2)	1.5 (1.0-2.0)	NS	1.8 (1.2-2.2)	NS	2.2 (1.6-2.5)	0.024
ST_1 [min]	4.5 (1.5-6.8)	1.5 (1.2-4.0)	0.019	1.5 (1.1-2.1)	<0.001	1.5 (1.2-2.0)	<0.001
ST_{recov} [min]	4.0 (2.0-5.0)	2.0 (2.0-3.0)	0.006	3.5 (3.0-5.0)	NS	5.0 (4.0-6.0)	0.003
Etime [min]	9.8 (7.0-11.6)	7.1 (5.1-9.0)	0.001	6.9 (5.0-8.1)	<0.001	5.0 (4.2-7.0)	<0.001
Load [METs]	11.0 (8.0-13.4)	7.0 (6.0-9.1)	<0.001	6.8 (4.8-7.1)	<0.001	5.8 (4.6-6.8)	<0.001
SDNN [ms]	140 (122-167)	98 (89-123)	<0.001	100 (78-115)	<0.001	74 (62-110)	<0.001
pNN50 [%]	9 (4-23)	4 (1-5)	<0.001	4 (2-7)	0.001	4 (2-6)	<0.001

Abbreviations: SX – syndrome X, CA1 – one vessel coronary disease, CA2 – two vessel coronary disease, CA3 – three vessel coronary disease, HR_{rest} – heart rate at rest before exercise, HR_{peak} – heart rate at peak exercise, SBP_{rest} – systolic blood pressure at rest, SBP_{peak} – systolic blood pressure at peak exercise, ST_{max} – the maximal ST depression during exercise, ST_1 – time to significant ST depression of at least 1 mm in at least two ECG leads, ST_{recov} – time to ST-segment normalisation during post-exercise recovery, Etime – time of exercise during ETT, Load – total obtained workload during exercise, SDNN – standard deviations of normal-to-normal RR intervals during Holter ECG recording, pNN50 – percentage of successive RR intervals varying >50 ms during Holter ECG recording

individuals, 37 (72.5%) CA2 subjects and 62 (98.4%) CA3 subjects. Again, the presence of ST-segment changes was comparable between SX and CA2 patients.

Patients with SX had significantly thinner interventricular septum and smaller LVEDd than individuals from the CA1, CA2 and CA3 groups. There were no significant differences in the LVEF or the LVPWTd between SX patients and subjects from the CA1, CA2 and CA3 groups. Minor contractile dysfunction (hypokinesia and akinesia of 1 segment in 1 patient) was present only in CAD patients: 3 (6.1%) from the CA1 group, 6 (11.8%) in CA2 individuals and 16 (25.4%) in CA3 subjects.

Discussion

The main findings of this study are that SX individuals compared with all CAD patients have a faster HR and lower systolic BP, both at rest and peak exercise. They also have longer duration of exercise with better exercise performance, later occurrence of significant, 1 mm ST-segment depression during ETT, and higher HRV in 24-hour ECG recording, thinner interventricular septum, smaller end-diastolic LV cavity dimension and no contractile abnormalities. However, SX patients have comparable ST-segment depression during exercise to that in CA1 and CA2 patients but it is significantly lower than that occurring in CA3 patients.

Additionally, in SX individuals post-exercise ST-segment normalisation takes longer than in CA1, is similar in CA2 and is shorter than that in CA3 patients. Moreover, SX patients have significantly higher values of maximal ST-segment depression and duration of significant ST-segment depression in Holter ECG recordings than CA1 subjects. These values are comparable in SC individuals and patients from the CA2 group, and significantly lower than in CA3 patients.

Patients with SX and CAD have much in common, i.e. chronic chest pain (angina pectoris or angina-like pain) and significant ST-segment changes. It is not easy to distinguish between these groups of patients without coronary arteriography. Detailed analysis of symptoms reveals that SX subjects have at least comparable severity and similar triggering factors of angina pain, i.e. anxiety or exercise, and these symptoms may also occur at rest. However, the pain may be prolonged, and is relieved by sublingual nitrates in no more than 50% of SX individuals [2, 19].

Our study shows that some results of ECG-based non-invasive diagnostic procedures are similar in SX and CAD patients, whereas some other results are different. Selected variables derived from ST-segment analysis show that patients with SX present results analogous to those in CAD subjects with two-vessel disease. This applies to maximal ST-segment depression on exercise, time needed for its normalisation during recovery, the maximal ST-segment depression and total time of significant ST-segment depression found in 24-hour ambulatory ECG.

Nonetheless, the analysis of HR and systolic BP, before and during exercise, time to significant ST depression, total exercise time and circadian HRV and

ST-segment changes in 24-hour ECG recordings as well as some echocardiographic parameters demonstrated significant differences between SX and CAD subjects. The heart rate is faster and the systolic BP lower, both at rest and at peak exercise, in SX subjects. The IVSd and the LVEDd are smaller in SX patients. Their SDNN and pNN50 values are normal, suggesting a low risk of future cardiovascular events, which confirms the results of other studies [19, 20].

The increased HR in SX patients, pre- and during ETT, may be due to female prevalence and/or some propensity of these patients to abnormal autonomic regulation of cardiovascular function [2, 5, 6]. Although several studies [2, 5, 6, 22] have indicated that a predominance of sympathetic activity may contribute to the pathogenesis of SX, another [23] shows, as do our HRV results, no apparent defect in cardiac autonomic function.

The lower systolic BP, before and at peak exercise, is a consequence of our selection criteria, as no hypertensive patients were included in the SX group. However, the question of what causes ST-segment depression in SX patients is still open. Patients with SX are heterogeneous and therefore it is not surprising that ST-segment depression can be evoked by the many varied factors mentioned earlier [2-17]. Severe ST changes can be due to abnormal myocardial potassium metabolism, which may be caused by decreased activity of the ATP-ase dependent sodium pump and enhanced Na/Li counter transport in SX patients [13-15, 23]. The ischaemic-like ST-segment changes may also be attributed to increased coronary microvascular tone and its overshooting contractile reaction to various stimuli, e.g. adenosine, catecholamines or other vasodepressor agents producing significant ST changes. In addition, anxiety and reduced pain threshold can trigger the ST-segment depression accompanying angina attacks in SX patients [2, 8, 14, 16, 17, 24, 25].

True myocardial ischaemia remains the most common trigger of ST-segment depression independently of all the potential non-cardiac, as well as cardiac, causes, which are usually excluded in SX patients (e.g. valve disease, cardiomyopathies or hypertension) [2, 4]. A recent study using cardiovascular magnetic resonance revealed the presence of subendocardial hypoperfusion associated with intense chest pain during intravenous adenosine administration in SX patients [17]. Another study [26], which involved the use of coronary intravascular ultrasound, displayed three morphological groups within SX patients: the first with normal coronary arteries, the second with atheromatous stenosis <50% (mean area stenosis 38%), and the last with marked intimal thickening of coronary arteries. The patients with normal coronary arteries presented a vasodilator response whilst those with atherosclerotic changes (intra- or extraluminal) showed a vasoconstrictor response to exercise. In one report [27], double-helical computed tomography identified coronary calcification, which was associated with the presence of atherosclerotic changes, in 63% of women with SX compared to only 22% of normal females. These data imply that atherosclerosis and myocardial ischaemia, whether occult or apparent, are present in some SX patients and may be responsible for the ST-segment changes observed. However, only very sophisticated methods are able to reveal the true level of myocardial ischaemia during angina attacks in SX individuals.

Limitations of the study

There are significant differences in clinical characteristics between SX and CAD patients. Generally, this is due to the rigid criteria for SX, as patients with co-existing diabetes, hypertension and any cardiac disease were excluded from this group. The mean age of approximately 45 years and female predominance of our SX patients reflects the common finding that usually post-menopausal women can be met in this group [2, 10, 19]. By contrast, the CAD patients, who were recruited into the study consecutively, represent a wider age range and thus older patients are included in this group. A lower BMI, a better lipid profile and a lower ratio of smokers to non-smokers seem to be secondary to both female predominance and the lower age of SX patients.

Therefore, it is plausible to suggest that the differences in clinical characteristics observed between the SX and CAD patients may be the reason for the results of ETT, 24-hour ambulatory ECG recordings and echocardiography, but appear to be of minor relevance. We are also aware that some of the results, such as HR changes during the ETT, might be affected by medications taken by our patients. Although no drugs (except nitrates for angina) were allowed during the 24 hours before the ETT, this time might be too short to eliminate some pharmacological agents and their active derivatives (e.g. beta-blockers).

Conclusions

The characteristics of ST-segment depression evoked by exercise are similar in patients with SX and advanced CAD, but we are unable to offer an explanation for this. However, SX patients have preserved HRV, a faster HR and a lower systolic BP while exercising, can tolerate longer exercise and have less pronounced interventricular septum thickness and end-diastolic LV cavity dimension than CAD patients.

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Porównanie wyników próby wysiłkowej EKG i 24-godzinnej rejestracji EKG metodą Holtera u pacjentów z kardiologicznym zespołem X i miażdżycą tętnic wieńcowych

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Streszczenie

Wstęp: Chorzy z kardiologicznym zespołem X (SX) zgłaszają incydenty bólu w klatce piersiowej przypominające swoim charakterem dolegliwości dławicowe występujące u osób z chorobą wieńcową (CAD). Podczas diagnostycznych prób wysiłkowych EKG chorzy z SX, podobnie jak chorzy z CAD, prezentują zależne od wielkości obciążenia wysiłkiem objawy bólowe z towarzyszącymi obniżeniami odcinka ST w EKG. Podobieństwo objawów klinicznych i zmian elektrokardiograficznych jest przyczyną dużych trudności w różnicowaniu SX i CAD.

Cel: Porównanie wyników testów wysiłkowych EKG, 24-godzinnego monitorowania EKG metodą Holtera oraz badania echokardiograficznego przeprowadzonych u pacjentów z SX i chorych bez przebytego zawału serca, ale z potwierdzoną w badaniu koronarograficznym CAD, tj. istotnym zwężeniem w jednej tętnicy (CA1), dwóch tętnicach (CA2) lub trzech tętnicach wieńcowych (CA3).

Metodyka: Badania przeprowadzono u 206 pacjentów: 43 osób (28 kobiet) z SX, 49 osób z CA1 (11 kobiet), 51 osób z CA2 (7 kobiet) oraz 63 osób z CA3 (8 kobiet). U wszystkich badanych przeprowadzono test wysiłkowy EKG na bieżni ruchomej wg protokołu Bruce'a, 24-godzinne monitorowanie EKG metodą Holtera z oceną dobowej zmienności czasu trwania kolejnych odstępów RR rytmu zatokowego oraz badanie echokardiograficzne.

Wyniki: Pacjenci z SX w porównaniu z pacjentami z CAD mieli szybszą czynność serca i niższe ciśnienie tętnicze, zarówno w spoczynku, jak i na szczycie wysiłku. Próba wysiłkowa EKG wykazała podobny stopień obniżenia odcinka ST w grupie pacjentów z SX i pacjentów z CA1 oraz CA2, natomiast pacjenci z CA3 prezentowali istotnie większe obniżenie odcinka ST. Czas do wystąpienia obniżenia odcinka ST o co najmniej 1 mm oraz całkowity czas trwania wysiłku były istotnie dłuższe w grupie osób z SX w porównaniu z wszystkimi pacjentami z CAD. Powysiłkowy czas powrotu odcinka ST do wartości wyjściowych był znamiennie dłuższy w grupie osób z SX w porównaniu z pacjentami z CA1 (p=0,006), natomiast był podobny do stwierdzonego w grupie pacjentów z CA2 oraz istotnie krótszy w porównaniu z pacjentami z CA3 (p=0,003). W porównaniu ze wszystkimi pacjentami z CAD 24-godzinna rejestracja EKG metodą Holtera wykazała w grupie chorych z SX znamiennie większe, prawidłowe wartości zarówno SDNN (odchylenie standardowe czasów trwania wszystkich odstępów RR rytmu zatokowego), jak i pNN50 (odsetek odstępów RR różniących się o >50 ms od sąsiedniego odstępu RR, obliczony w odniesieniu do wszystkich odstępów RR). Grubość rozkurczowa przegrody międzykomorowej oraz wymiar późnorozkurczowy lewej komory były istotnie mniejsze w grupie osób z SX, podczas gdy frakcja wyrzutowa lewej komory i grubość rozkurczowa tylnej ściany lewej komory były podobne we wszystkich badanych grupach.

Wnioski: Zakres zmian odcinka ST stwierdzany w wysiłkowych EKG osób z SX sugeruje obecność zaawansowanej choroby wieńcowej. Pacjenci z SX w porównaniu z chorymi z CAD mają jednak większą tolerancję wysiłku oraz lepsze wyniki parametrów zmienności rytmu zatokowego.

Słowa kluczowe: zespół X, choroba wieńcowa, test wysiłkowy, monitorowanie EKG metodą Holtera

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