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QT dispersion in patients with Churg-Strauss syndrome

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Abstract

Background: Churg-Strauss syndrome (CSS) is a rare, systemic necrotising small and middle-sized vessel vasculitis, accompanied by blood eosinophilia, eosinophil infiltration of various tissues and bronchial asthma. Cardiac injury caused by myocardial eosinophilic infiltration and/or vasculitis in CSS seems to be very common. Active inflammatory process accompanied by myocardial fibrosis has been described in this population even despite disease remission. Nevertheless, little is known about the possible myocardial repolarisation abnormalities in CSS which may lead to life-threatening ventricular arrhythmias.

Aim: To evaluate myocardial repolarisation in CSS patients at the time of initial diagnosis and during the last disease remission.

Methods: In 20 CSS patients (8 male, 12 female) QT dispersion (QTd) and QTc dispersion (QTcd) calculated from heart rate corrected QT (QTc) from the surface 12-lead electrocardiograms were measured at the time of initial diagnosis and during the last disease remission. As a control group, 20 sex- and age-matched healthy volunteers were studied. Transthoracic echocardiography was performed in all CSS patients at remission and in the control group.

Results: QTcd was higher in CSS (n = 20) than in healthy controls (n = 20) in each period of time: at the time of initial diagnosis (45.4 ± 14.2 *vs* 26.1 ± 6.5, p < 0.0001) and at the remission (38.6 ± 13.4 *vs* 26.1 ± 6.5, p = 0.002). At the time of initial diagnosis in CSS patients with heart involvement (n = 13), when compared to patients without heart involvement, (n = 7), both QTcd (52.2 ± 12.1 *vs* 34.7 ± 10.7, p = 0.007) and QTd (37.7 ± 12.7 *vs* 24 ± 11.4, p = 0.008) were higher, and this difference remained significant at remission only for QTcd (46.7 ± 13.2 *vs* 33.1 ± 10.8, p = 0.03). No significant correlation was observed between QTcd/QTd and disease activity (measured using the Birmingham Vasculitis Activity Score — BVAS), eosinophil blood count, presence of ANCA, nor the duration of the disease.

Conclusions: The most pronounced increased QTcd was detected in the CSS patients with cardiac involvement at the time of initial diagnosis and remained higher at remission in all CSS patients when compared to healthy controls. Nevertheless, in the CSS patients, QTcd remains within the normal ranges, which may explain the relatively small number of ventricular arrhythmias in these patients.

Key words: QT dispersion, Churg-Strauss syndrome, cardiac involvement, vasculitis

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INTRODUCTION

Churg-Strauss syndrome (CSS) is a rare, systemic necrotising small and middle-sized vessel vasculitis, accompanied by blood eosinophilia, eosinophil infiltration of various tissues and bronchial asthma [1]. Cardiac involvement, depending on the report and the disease's activity, has been documented in 16–92% of CSS patients and is associated with a poor prognosis and high mortality, if untreated [2]. Myocarditis with cardiomyopathy, pericarditis (25% of patients), pericardial effusion (up to 22%), heart failure (HF, 18%), ventricular arr-

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hythmias, and sudden cardiac death (SCD) have been documented [2]. Ventricular arrhythmias are relatively rare and their mechanism has not yet been explained [3, 4].

We hypothesised that CSS-related heart injury may cause myocardial repolarisation abnormalities. The QT dispersion (QTd) measured on a surface ECG is a simple, low-cost and non-invasive technique, reflecting the heterogeneity of ventricular repolarisation [5]. An increased QTd has been reported as an arrhythmogenic risk factor in various clinical groups [5], but has not yet been studied in CSS patients.

The aim of this study was to determine QTd in a standard 12-lead ECG in CSS patients at the time of initial diagnosis and during the last disease remission.

METHODS

Subjects

We analysed retrospectively medical records of CSS patients who were hospitalised and diagnosed between July 1999 and August 2010 in our department and later followed in the outpatient clinic. The patients were evaluated twice: at the time of the initial CSS diagnosis, and during the last clinical remission. The diagnosis of CSS was reassessed and confirmed according to the current American College of Rheumatology (ACR) criteria, which distinguish six key features [6]. The six are: asthma, peripheral blood eosinophilia > 10%, mono- or polyneuropathy, chronic paranasal sinusitis, extravascular eosinophils revealed on biopsy, and migratory infiltrates in lungs. Only patients who fulfilled at least four of the six ACR criteria and had technically good quality ECG available from each of the two studied time points were included in the study. Excluded from the study were patients who prior to the CSS had: coronary artery disease (CAD), arterial hypertension, HF, long QT syndrome, diabetes mellitus or were on medications known to influence the ECG or heart rate. The medical documentation was evaluated for clinical manifestations of CSS, laboratory data and medication used. The presence of cardiac involvement at the time of initial CSS diagnosis was based on clinical features, elevated troponin I level, ECG abnormalities, and pathological ECHO findings such as new wall motion abnormalities, pericarditis, endocarditis or myocarditis, simultaneously excluding CAD and other possibly underlying conditions. The Birmingham Vasculitis Activity Score (BVAS, range: 0–63 patients) was used to assess activity of CSS at initial diagnosis and in remission. Remission of CSS was defined as the absence of disease symptoms (BVAS ≤ 1) and blood eosinophilia in normal ranges for at least three months. Additionally in all CSS patients in remission, standard 12-lead ECGs and transthoracic echocardiography (TTE) were performed.

Control group

Sex- and age-matched healthy volunteers were selected randomly for the control group. All participants gave an informed consent to participate. The study protocol complied with the Helsinki Declaration and was approved by the Jagiellonian University Ethics Committee.

Electrocardiography

A standard 12-lead ECG was recorded at a paper speed of 25 mm/s in CSS patients (at the time of initial disease diagnosis and during the last remission) and in a healthy control group.

The QT intervals were measured in each lead from the beginning of depolarisation of the QRS complex to the end of the T wave. The end of the T wave was defined as the point of return to baseline, and in the presence of a U wave, the U wave was not included and the T wave was extrapolated to baseline. When there was no definite end of the T wave, or where premature complexes obscured the T wave end, the lead was not analysed. For each lead, three consecutive cycles were measured and the arithmetic mean of the QT interval for that lead was used in all future calculations for QT dispersion. The QT intervals were additionally corrected (QTc) according to the heart rate using Bazett's formula. The QTd was calculated as the difference between the minimum and maximum QT intervals for any of the 12 leads, and QTc dispersion (QTcd) as the difference between the minimum and maximum QTc intervals for any of the 12 leads.

All measurements were made from paper recordings using hand calipers by two investigators unaware of clinical data. If conflicting results occurred, the ECG was discussed and differences in measurements were resolved by consensus.

Transthoracic echocardiography

All CSS patients in remission, and the control group, underwent a TTE (M-mode, two-dimensional and Doppler echocardiography with Vivid 7 ultrasound systems; GE Vingmed Ultrasound A/S, Horten, Norway) for the evaluation of systolic and diastolic function, in accordance with the current recommendation of the American Society of Echocardiography. Systolic dysfunction was defined as left ventricular ejection fraction (LVEF) < 50%. The following criteria were used to classify abnormal diastolic function: (a) impaired relaxation pattern: E/A ratio < 1.0 and DT > 200 ms; (b) pseudonormal pattern: E/A ratio ranging 1.0–2.0, and at least two of the following: S/D ratio < 1 or Ar ≥ 35 cm/s or e' < a' and E/e' ratio > 10; (c) restrictive pattern: E/A ratio > 2.0 and DT < < 150 ms [7]. The analysis was performed off-line by an independent, experienced investigator, unaware of clinical data.

Statistical analysis

Statistical analysis was performed using StatSoft, Inc. STATISTICA software, version 8.0. (www.statsoft.com). Data were checked for normality and are presented as the mean ± SD or median with interquartile range, when applicable. The comparison between the group means was performed with Student's t test, and Mann-Whitney-U test depending on the data distribution. For repeated measures, Student's t test or

Wilcoxon test were used. Correlation was tested using Spearman rank test. A p value < 0.05 was considered statistically significant.

RESULTS

Clinical findings

Twenty two patients fulfilling at least four ACR diagnostic CSS criteria, hospitalised between 1999 and 2010, were found in medical databases. One patient was excluded from the study because of arterial hypertension, and one because of lack of technically good quality ECG at the time of initial diagnosis. The final study group consisted of 20 patients (12 females, 8 male; aged 44.3 ± 9 years) and 20 sex- and age-matched healthy volunteers as a control. All volunteers were normotensive and free of any cardiac or systemic diseases. Their clinical examinations, ECG and ECHO results were all considered normal. The demographic and clinical characteristics are presented in Table 1.

At the time of initial diagnosis, cardiac involvement was detected in 13 (65%) CSS patients. All these patients with heart involvement in TTE examination showed the presence of pericardial effusion. Six (30%) patients additionally presented myocarditis with wall motion abnormalities and diminished LVEF. Of the 13 patients with primary heart involvement, at remission heart involvement was seen in TTE in eight patients and during follow-up in other patients there were no new cardiac changes detected. Out of the CSS group during disease remission, four (20%) patients were on combined treatment with angiotensin-converting enzyme inhibitors (ACEI), beta-blockers and diuretics due to chronic HF. None of the subjects from the control group took any medications. During the follow-up period in two CSS patients with severe systolic heart dysfunction, clinically important ventricular arrhythmias were documented and no incidence of SCD was observed.

Table 1. Clinical characteristics of the CSS patients (n = 20)

Male/female ratio	8 (40%)/12 (60%)
Age [years]	44.3 ± 9
Age at CSS diagnosis [years]	40.7 ± 9.9
Median of CSS duration [years]	2.5 [1–6]
Criteria for diagnosis of CSS:	
Asthma	20 (100%)
Blood eosinophilia higher than 10% of the WBC differential count	20 (100%)
Chronic paranasal sinusitis	20 (100%)
Migratory or transient lung infiltrates	20 (100%)
Mono-/polyneuropathy	10 (50%)
Extravascular eosinophils revealed on biopsy	6 (30%)
At the time of initial CSS diagnosis:	
p-ANCA	4 (20%)
Median of peripheral blood eosinophilia, $\times 10^3$/L	5,308 [1,931–8,226]
BVAS	22.7 ± 7
Cardiac involvement	13 (65%)
Systolic dysfunction	6 (30%)
Diastolic dysfunction	NA
LVEF [%]	52.2 ± 14.2
Median of maximal peripheral blood eosinophilia during CSS, $\times 10^3$/L	6,162 [2,815–8,226]
In CSS remission (echocardiographic findings):	
Systolic and diastolic dysfunction	4 (20%)
Diastolic dysfunction	4 (20%)
LVEF [%]	55.8 ± 14
Treatment during remission:	
Daily dosage of methylprednisolone in CSS remission [mg]	6.7 ± 2.7
Additional immunosuppressive therapy (cyclophosphamide or azatiopryne)	13 (65%)

Data expressed as mean values ± SD or medians [25–75 interquartile range]; BVAS — Birmingham Vasculitis Activity Score; CSS — Churg-Strauss syndrome; LVEF — left ventricular ejection fraction; NA — not available; p-ANCA — antineutrophil cytoplasmic antibodies with perinuclear pattern; WBC — white blood cells

QT dispersion analysis

In our study, intraobserver variability of QT intervals measurements was calculated at 8% in the control group and at 10% in CSS patients. All QT parameters are presented in Tables 2 and 3.

Both QTcd and QTd were higher in the CSS patients than in healthy controls at the time of initial diagnosis and during remission. Additionally, patients with CSS at the time of diagnosis had a higher heart rate compared to healthy controls; however, this difference disappeared at disease remission.

The QTcd and QTd values were significantly higher at the time of initial diagnosis in the CSS patients with heart involvement compared to CSS patients without heart involvement, and this difference remained significant at remission for QTcd, and disappeared for QTd. The CSS patients had lower QTcd at remission than during the initial diagnosis. This difference was driven by the group of patients with heart involvement, who at remission had lower QTcd than during the initial diagnosis, while the QTcd did not change in patients without heart involvement. No significant correlation was observed between QTcd/QTd and BVAS, eosinophil blood count, presence of ANCA, nor the duration of the disease.

DISCUSSION

Heart injury in the CSS patients is caused by myocardial eosinophilic infiltration and/or vasculitis and seems to be very common, especially during exacerbation of the disease. However, active inflammatory process accompanied by myocardial fibrosis has been described in this population, even despite disease remission [2, 8]. Little is known about the possible myocardial repolarisation abnormalities in CSS, which are

Table 2. QT and QT dispersion values at the time of initial diagnosis and at remission

	CSS patients (n = 20)		P
	At the time of initial diagnosis (n = 20)	At remission (n = 20)	
QT [ms]	376.9 ± 49.4	385.1 ± 14.9	0.16
QTc [ms]	451 ± 44.6	430.1 ± 34.1	0.01
HR [1/min]	97.7 ± 18.4	71.9 ± 9.8	0.0002
QTd [ms]	33.8 ± 13.1	31.4 ± 13.3	0.23
QTcd [ms]	45.4 ± 14.2	38.6 ± 13.4	0.004
	CSS patients at the time of initial diagnosis (n = 20)		P
	With heart involvement (n = 13)	Without heart involvement (n = 7)	
QT [ms]	383.7 ± 59.7	366.3 ± 27.4	0.65
QTc [ms]	459.9 ± 48.6	437 ± 48.6	0.32
HR [1/min]	102.5 ± 18.3	90 ± 17	0.14
QTd [ms]	37.7 ± 12.7	24 ± 11.4	0.008
QTcd [ms]	52.2 ± 12.1	34.7 ± 10.7	0.007
	CSS patients at remission (n = 20)		P
	With heart involvement (n = 8)	Without heart involvement (n = 12)	
QT [ms]	380.8 ± 12.6	388 ± 16.1	0.3
QTc [ms]	443.5 ± 32.1	421.3 ± 33.7	0.16
HR [1/min]	76.9 ± 10.3	68.5 ± 8.1	0.09
QTd [ms]	38.3 ± 10.6	26.8 ± 13.2	0.07
QTcd [ms]	46.7 ± 13.2	33.1 ± 10.8	0.03

CSS — Churg-Strauss syndrome; HR — heart rate; QT — QT interval; QTc — corrected QT interval; QTd — QT interval dispersion; QTcd — corrected QT interval dispersion

considered to be a risk factor for developing ventricular arrhythmias. Our study is the first to assess myocardial repolarisation processes in CSS patients.

The QTd measurement is a simple, non-invasive and low-cost method of assessing the heterogeneity of myocardial repolarisation [5]. Traditionally higher QTd favours the development of serious and life-threatening ventricular arrhythmias and is present in patients with various cardiac diseases [5, 9–11]. Additionally, increased QTd after myocardial infarction has been found to be a risk factor for SCD [9].

In the present study, QTd was significantly increased in the CSS patients compared to the healthy control group both at the time of initial diagnosis and during remission. The highest QTd was detected in 13 CSS patients with cardiac involvement at the time of initial diagnosis, and this difference diminished when patients entered remission. In the CSS group, two patients (with the highest QTd) had documented serious ventricular arrhythmias over the follow-up period; the first had sudden cardiac arrest (resuscitated successfully) and was qualified for heart transplantation, and the second required cardioverter-defibrillator implantation. Additionally, both these patients had serious HF with very low LVEF (16% and 19% respectively).

In the remaining CSS patients, neither clinically important arrhythmias nor SCD were observed. It is noteworthy that QTc was higher in the CSS patients at the time of initial diagnosis (especially in those with heart involvement) and QTd was higher in all CSS patients even at remission, but there were relatively small numbers of ventricular arrhythmias in these patients [4, 12]. The explanation for this could be that values of QTd in our patients were not very high — maximally 88.8 ms. It has been shown that QTd between 30 and 60 ms are present in healthy subjects [13]. In the Strong Heart Study on 1,839 subjects in multivariate analysis, QTcd > > 58 ms was associated with a 3.2-fold increased risk of cardiovascular mortality. In patients with HF, QTd > 90 ms carried a 2.8 fold greater risk for SCD and non-SCD [13]. Nevertheless, there is a good deal of controversy surrounding prognostic QTd value for arrhythmias, and some authors have assumed only grossly abnormal QTd (of ≥ 100 ms) to be significant [13].

To date, QTd has been investigated in only a few cases of systemic vasculitis. Most of these studies were performed in the Behcet's disease, and found that the patients with this entity had higher QTd, which correlated with the frequency of ventricular arrhythmias, number of premature ventricular complexes, and with diastolic HF [14, 15]. The main postulated mechanisms of repolarisation abnormalities in the Behcet's patients are coronary ischaemia due to vasculitis, myocarditis, myocardial fibrosis and autonomic nerve system dysfunction [14].

Table 3. QT and QT dispersion values of Churg-Strauss patients and of control subjects

	CSS patients at the time of initial diagnosis (n = 20)	Control group (n = 20)	P
QT [ms]	376.9 ± 49.4	375.3 ± 19.5	0.74
QTc [ms]	451 ± 44.6	416.3 ± 14	0.002
HR [1/min]	97.7 ± 18.4	71 ± 6.1	< 0.0001
QTd [ms]	33.8 ± 13.1	21.7 ± 3.2	0.005
QTcd [ms]	45.4 ± 14.2	26.1 ± 6.5	< 0.0001
	CSS patients with heart involvement at the time of initial diagnosis (n = 13)	**Control group (n = 13)**	**P**
QT [ms]	383.7 ± 59.7	373.8 ± 19.6	0.86
QTc [ms]	459.9 ± 48.6	412.6 ± 15	0.0005
HR [1/min]	102.5 ± 18.3	70.6 ± 6.4	< 0.0001
QTd [ms]	37.7 ± 12.7	21 ± 1.5	< 0.0001
QTcd [ms]	52.2 ± 12.1	25 ± 5.2	< 0.0001
	CSS patients without heart involvement at the time of initial diagnosis (n = 7)	**Control group (n = 7)**	**P**
QT [ms]	366.3 ± 27.4	378 ± 19	0.61
QTc [ms]	437 ± 48.6	423.1 ± 9.3	0.9
HR [1/min]	90 ± 17	71.4 ± 6	0.04
QTd [ms]	24 ± 11.4	23.2 ± 5	0.79
QTcd [ms]	34.7 ± 10.7	28.2 ± 8.5	0.16
	CSS patients at remission (n = 20)	**Control group (n = 20)**	**P**
QT [ms]	385.1 ± 14.9	375.3 ± 19.5	0.1
QTc [ms]	430.1 ± 34.1	416.3 ± 14	0.046
HR [1/min]	71.9 ± 9.8	71 ± 6.1	0.74
QTd [ms]	31.4 ± 13.3	21.7 ± 3.2	0.009
QTcd [ms]	38.6 ± 13.4	26.1 ± 6.5	0.002
	CSS patients with heart involvement at remission (n = 8)	**Control group (n = 8)**	**P**
QT [ms]	380.8 ± 12.6	373.4 ± 22.3	0.5
QTc [ms]	443.5 ± 32.1	412.7 ± 16.6	0.04
HR [1/min]	76.9 ± 10.3	72 ± 5.4	0.16
QTd [ms]	38.3 ± 10.6	20.4 ± 1	0.001
QTcd [ms]	46.7 ± 13.2	22.6 ± 1.9	0.007
	CSS patients without heart involvement in remission (n = 12)	**Control group (n = 12)**	**P**
QT [ms]	388 ± 16.1	376.6 ± 17.3	0.11
QTc [ms]	421.3 ± 33.7	418.6 ± 12	0.44
HR [1/min]	68.5 ± 8.1	70.2 ± 6.6	0.51
QTd [ms]	26.8 ± 13.2	22.6 ± 3.9	0.38
QTcd [ms]	33.1 ± 10.8	28.5 ± 7.5	0.26

Abbreviations as in Table 2

Similarly, QTd has been shown to be increased in some studies of Kawasaki disease (KD). Crystal et al. [16] described in a large group of 176 children with KD increased QTd in the acute phase of disease, which did not completely resolve within the first year of recovery. In another work, Ghelani et al. [17] revealed increased QTd in KD patients despite the

convalescent disease phase and without signs of coronary arteries involvement. Patients with KD without CAD are also more prone to ventricular arrhythmias, probably due to myocardial inflammation [18]. Increased QTd has been well documented in patients diagnosed with myocarditis [19] and seems to be associated with ventricular arrhythmias in these cases. One of the explanations of ongoing repolarisation abnormalities in the convalescent phase of KD is thought to be a possible presence of subclinical chronic myocarditis. On the other hand, Kato et al. [20] observed significantly higher baseline QTd in patients with Takayasu's arteritis (without coronary stenosis in angiography) and documented exercise-induced myocardial perfusion abnormalities in scintigraphy in comparison with Takayasu patients with normal scintigraphy results; they suggested QTd measurements as a marker of myocardial involvement in these patients.

There are at least two possible explanations for the higher QTd in the acute phase of CSS: eosinophil myocardial infiltration and the ongoing vasculitis process. Even at the remission of the disease, an active myocarditis process has been described [21, 22] in some CSS patients, and most patients have persistent late gadolinum enhancement abnormalities in magnetic resonance imaging (MRI) [2]. This may indicate fibrosis and has been found to correlate with ventricular arrhythmias [23]. Fibrosis of the myocardium may be responsible for the repolarisation abnormalities in the CSS patients and may help explain why, in our study, patients without cardiac involvement had higher QTd compared to controls. It may simply be that some of the residual myocardial changes might not have been detected in the ECHO examination performed in this study, but would be visible in MRI, or revealed in biopsy [8]. Therefore we speculate that while systolic or diastolic dysfunction may not be evident in echocardiography in CSS patients, subclinical myocardial involvement may be indicated by subtle repolarisation abnormalities in this group.

Limitations of the study

This is a retrospective study, which may create a bias. Also the small number of patients and clinically important cardiac arrhythmias may have influenced the statistical lack of correlation of the QT interval with clinical and laboratory status. Additionally, the CSS patients with heart involvement in the remission period were on ACEI and beta-blockers, which may influence the QT values.

CONCLUSIONS

Patients with CSS, irrespective of heart involvement and disease activity, have increased QTd duration. This points to repolarisation abnormalities and may indicate myocardial involvement in these patients.

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Dyspersja QT u pacjentów z zespołem Churga-Strauss

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Streszczenie

Wstęp: Zespół Churga i Strauss (ZCS) jest rzadkim, układowym, martwiczym zapaleniem małych i średnich naczyń, z towarzyszącą eozynofilią we krwi obwodowej, naciekami eozynofilowymi różnych tkanek i astmą oskrzelową. Uszkodzenie serca na tle nacieków eozynofilowych miokardium ze współistniejącym zapaleniem naczyń serca lub bez niego w ZCS wydaje się częste. Aktywny proces zapalny, oprócz zwłóknień w mięśniu sercowym, opisywano w tym zespole u pacjentów nawet w pełnej klinicznej i laboratoryjnej remisji choroby. Jednak nadal niewiele wiadomo na temat potencjalnych zaburzeń procesów repolaryzacji mięśnia sercowego w ZCS, które mogą leżeć u podłoża poważnych, zagrażających życiu arytmii komorowych.

Cel: Celem badania była ocena procesów repolaryzacji mięśnia sercowego u osób z ZCS w momencie rozpoznania choroby i w czasie ostatniej klinicznej remisji.

Metody: Badano dyspersję odstępu QT (QTd) i dyspersję skorygowanego względem częstości serca odstępu QTc (QTcd) w standardowych 12-odprowadzeniowych elektrokardiogramach u 20 pacjentów z ZCS (8 kobiet, 12 mężczyzn) w momencie rozpoznania choroby i podczas ostatniej klinicznej remisji oraz u 20 dobranych pod względem płci i wieku zdrowych osób. U wszystkich badanych dodatkowo wykonano przezklatkowe badanie echokardiograficzne.

Wyniki: W grupie pacjentów z ZCS (n = 20) w porównaniu z grupą kontrolną (n = 20) stwierdzono wyższe QTcd w obu badanych punktach czasowych, tj. w momencie ustalenia rozpoznania ZCS (45,4 ± 14,2 *v.* 26,1 ± 6,5; p < 0,0001) i w okresie remisji choroby (38,6 ± 13,4 *v.* 26,1 ± 6,5; p = 0,002). W momencie postawienia diagnozy ZCS u osób z zajęciem serca w przebiegu choroby (n = 13) w porównaniu z pacjentami bez zajęcia serca (n = 7) oba parametry były wyższe: QTcd (52,2 ± 12,1 *v.* 34,7 ± 10,7; p = 0,007) i QTd (37,7 ± 12,7 *v.* 24 ± 11,4; p = 0,008), podczas gdy w okresie remisji istotna różnica dotyczyła jedynie QTcd (46,7 ± 13,2 *v.* 33,1 ± 10,8; p = 0,03). U chorych z ZCS nie stwierdzono korelacji między QTcd/QTd a aktywnością choroby mierzoną w skali BVAS (*The Birmingham Vasculitis Activity Score*), liczbą eozynofili we krwi obwodowej, obecnością przeciwciał ANCA (przeciwciała przeciwko składnikom cytoplazmy neutrofilów) ani czasem trwania choroby (p > 0.05).

Wnioski: Najistotniejsze wydłużenie QTcd stwierdzono u chorych z ZCS w momencie rozpoznania choroby; w grupie z pierwotnym zajęciem serca. Wydłużoną QTcd obserwowano w dalszym ciągu u wszystkich pacjentów z ZSC w momencie remisji w porównaniu ze zdrową grupą kontrolną. Niemniej wartości QTcd u chorych z ZCS pozostawały w zakresie normy, co może po części tłumaczyć relatywnie rzadko opisywane komorowe zaburzenia rytmu serca w tej grupie osób.

Słowa kluczowe: dyspersja QT, zespół Churga-Strauss, zajęcie serca, zapalenie naczyń

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