Recurrence of ventricular arrhythmias in patients with non-ischaemic dilated cardiomyopathy: evidence-based predictors

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Abstract

Background: Ventricular arrhythmia (VA) is the most frequent cause of sudden death among patients with non-ischaemic dilated cardiomyopathy (DCM).

Aim: To identify the important VA risk factors in patients with DCM.

Methods and results: Eighty-five DCM patients (73 males, mean age 54 years) with DCM and implantable cardioverter defibrillators (ICD) were followed for 21 ± 19 months after ICD implantation. The mean follow-up was 21 months. Data from 55 patients with VA recorded in the ICD memory and requring ICD intervention during follow-up were compared with 30 patients without arrhythmia. Cox regression analysis identified the following univariate predictors of VA: alcoholic aetiology of DCM (0.05), diuretic treatment (0.003), history of cardiac arrest (0.03), right ventricular diastolic diameter (0.001). Both ACE inhibitor (ACEI) and statin treatments were associated with a tendency towards decreased risk of VA. Multivariate logistic analysis identified four predictors as significantly related to VA: alcoholic aetiology (HR 4.8, p = 0.008), ACEI treatment (HR 0.4, p = 0.01), diuretic treatment (HR 2.6, p = 0.015), and statin treatment (HR 0.1, p = 0.03).

Conclusions: The majority of patients with DCM and ICD have recurrences of VA. Alcoholic aetiology of DCM is associated with an increase in the incidence of arrhythmias. Treatment with ACEI and statins is associated with a reduction of arrhythmias.

Key words: cardiomyopathy, arrhythmia, risk factors, implantable cardioverter defibrillator, sudden death

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Background

Cardiomyopathy is a disease of the heart muscle characterised by cardiac enlargement and impaired systolic function of one or both ventricles [1]. The incidence of dilated cardiomyopathy (DCM) is reported as 5 to 8 cases per 100 000 population per year and appears to be increasing [2]. The natural history of DCM has not been well established [3]. Sudden deaths due to rapid ventricular arrhythmias account for approximately 50-80% of all deaths in patients with idiopathic DCM [4, 5]. The overall long-term prognosis in DCM has improved due to evolving advances in diagnosis and therapy. However, there are still many incidences of sudden cardiac death (SCD) in DCM, which is a first disease manifestation in 4% of all patients with DCM [4].

A variety of clinical predictors of an increased risk of SCD have been identified, including presence of ventricular arrhythmias, advanced age, and specific endomyocardial biopsy features [6]. None of them however, are highly reliable in accurately predicting SCD occurrence. Non-invasive methods are not helpful in predicting arrhythmic events in DCM. Programmed ventricular stimulation has been used to evaluate long-term prognosis, but stratified data are still missing due to deaths occurring in patients without inducible arrhythmias [7].

Implantable cardioverter-defibrillator (ICD) therapy has been shown recently to improve survival in patients at high risk of sudden death due to ventricular tachycardia (VT) or ventricular fibrillation (VF). In DCM patients with a high risk for serious arrhythmic events, ICD therapy prevents

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SCD and therefore improves the long-term survival rate. Applying the arrhythmia risk stratification criteria for the prophylactic ICD implantation treatment in this group remains controversial. The goal of our study was to identify clinically significant criteria which would aid in screening patients for life-threatening arrhythmias.

Methods

Study patients

Data from the four largest centres participating in the Polish ICD Registry were analysed. The study group consisted of 85 patients (73 males, 12 females, average age 54 ± 12 years) with non-ischaemic DCM, out of whom 95% had a history of a previous cardiac arrest and therefore were selected for ICD implantation as a secondary prevention of SCD. The remaining patients received an ICD for primary prevention of SCD. Criteria for the study enrolment were the following: 1) left ventricular (LV) ejection fraction (EF) < 40%, 2) LV systolic diameter (LVSD) > 4.1 cm, 3) left ventricular end-diastolic diameter (LVDD) > 5.7 cm, 4) without > 50% stenosis of coronary artery and no history of myocardial infarction.

In our retrospective and observational study we analysed the value of the following potential arrhythmia risk predictors: demographic and anthropometric factors, comprehensive history and physical examination, electrocardiographic and echocardiographic parameters, 24-hour ambulatory electrocardiography and invasive electrophysiological study (EPS) data.

We analysed recurrences of ventricular arrhythmias and interventions [anti-tachycardia pacing (ATP) or shock] recorded by the ICD memory during the 21 ± 19 months of follow-up. Based on the recorded arrhythmia episode history, we assigned patients to two groups. Data from 55 patients (group 1) with episodes of ventricular arrhythmias such as sustained VT and/or VF were compared with 30 patients (group 2) without life-threatening ventricular arrhythmias. Research protocol met all criteria set by the local Ethics Committee and all patients signed an informed consent form before study participation.

Echocardiography and electrocardiography

Echocardiographic examination was performed according to the recommendations of the American Society of Echocardiography. The following parameters were assessed: LVEF, LVDD, LVSD, right ventricular diastolic diameter (RVDD), pulmonary artery pressure and acceleration time.

The following electrocardiographic (ECG) parameters were assessed in 12-lead ECG: duration of the QRS complex, presence of left or right bundle branch block (LBBB or RBBB respectively), corrected QT (QTc) according to the Bazett formula, and QT dispersion (QTd).

Electrophysiological study

Electrophysiological study was not routinely performed before ICD implantation. Only 45 (53%) out of all patients who additionally consented to this invasive examination underwent EPS. We used the ventricular stimulation protocol including at least basic pacing drive trains at 600 ms and 400 ms with up to 3 extra stimuli performed from the right ventricular apex (RVA) and from the right ventricular outflow tract (RVOT). Induction of sustained VT (VT duration > 30 s) was defined as a positive EPS.

ICD memory data interpretation

Based on the intracardiac electrograms stored in the ICD memory, two independent observers analysed all recorded events and classified them accordingly as: sinus tachycardia, atrial fibrillation or ventricular arrhythmias. Monomorphic VT was defined as a regular tachycardia that differed from the baseline rhythm and was distinguished from atrial fibrillation by: R-R interval variability of < 60 ms, R morphology and A to V ratio in dual chamber ICDs (19 patients received dual chamber ICDs, 66 patients single chamber ICDs). Ventricular fibrillation was defined as an irregular rhythm with polymorphic electrogram morphology and R-R interval shorter than 300 ms. We counted all episodes of arrhythmia which required ICD intervention – independently as ATP or shock. For the purpose of this study the inappropriate ICD therapies were not analysed.

Statistical analysis

All data are presented as a mean ± SD or as numbers and percentages (%). The Shapiro-Wilk test confirmed the normal distribution of the variables; therefore comparison between the groups with or without recurrences of ventricular arrhythmias was made using Student's *t* test for continuous variables and the Fisher test for discrete variables. The effects of relevant variables were evaluated with the Cox proportional hazards model. The curves showing time without arrhythmia recurrences were computed using the Kaplan-Meier method, and the differences between the curves were calculated using the log-rank test. A p value < 0.05 was considered significant. The STATA 06 (StataCorp, USA) software was used.

Results

The clinical characteristics of patients are presented in Table I. Both groups were comparable for all evaluated parameters including demographic and anthropometric factors, clinical symptoms prior to ICD implantation, other coexisting diseases and related treatment. Patients with arrhythmia recurrences more frequently were in NYHA class III prior to implantation than patients without VT/VF (p = 0.03). No significant differences were noted between the groups in ECG parameters.

Among 45 (53%) patients who underwent EPS, 34 (75%) of them had a positive test resulting in induction of sustained VT. There were no significant differences regarding recurrences of arrhythmia detected by ICD in both groups. Patients with arrhythmia recurrences had a higher RVDD (p = 0.008); however other echocardiographic parameters did not differ between groups.

Hospitalisation discharge medications for all patients after their ICD implantation are listed in Table I. A significant difference in prescribing statins at discharge between the two groups of patients was noted. Statins were prescribed to 27% of patients without arrhythmia recurrences compared with only 5% of patients with arrhythmia recurrences (p = 0.008). In addition, angiotensin-converting enzyme inhibitors (ACEI) were administered in 90% of patients without arrhythmia recurrences vs. 54% of patients with recurrences of arrhythmias (p = 0.001). Patients treated with diuretics had VT/VF more frequently than those without this type of treatment (p = 0.04).

Predictors of VT/VF recurrences

After a mean follow-up of 21 ± 19 months, 55 (65%) patients had VT/VF recurrences. The univariate relative risk of arrhythmia recurrences as a function of baseline characteristics in all patients is presented in Table II. The risk of arrhythmia recurrences was almost doubled in patients with alcoholic aetiology of DCM (HR 1.99, p = 0.05). Echocardiographic parameters including LVEF emerged as a weak predictor of VT/VF in univariate analysis. Only RVDD showed a significant correlation with the number of arrhythmic events (HR 1.1, p = 0.001). Moreover, a history of cardiac arrest before ICD implantation was associated with increasing risk of arrhythmias (HR 1.9, p = 0.03).

Selected prescription treatment seemed to play an important role in arrhythmia recurrences. Diuretics significantly increased the risk of arrhythmias (HR 2.5, p = 0.003). The opposite results were observed in the case of ACEI, which significantly reduced the rate of arrhythmic events (HR 0.46, p = 0.008). Treatment with amiodarone and beta-blockers had minimal influence on risk of arrhythmias.

Multivariate analysis was performed in order to establish whether the above listed factors (which were statistically or nearly statistically significant in the univariate analysis) had independent prognostic value for arrhythmic recurrences. Four of these factors – ACEI, statins, diuretic treatment and alcoholic aetiology of cardiomyopathy – remained independent predictors. Alcoholic aetiology of DCM was an independent factor increasing risk of arrhythmias (HR 4.8, p = 0.008). A similar relationship was found for diuretic treatment (HR 2.6, p = 0.015), whereas ACEI (HR 0.4, p = 0.013) and statin treatment (HR 0.14, p = 0.03) were independent factors decreasing the risk of VT/VF recurrences (Figure 1).

Table I. Baseline characteristics of the total group and patients with or without arrhythmia recurrences

Parameters	All patients (n = 85)	Arrhythmia recurrence (n = 55)	No recurrence of arrhythmia (n = 30)	p
Age [years]	53.9	54.4	53	NS
Gender (M/F)	73/12	48/7	25/5	NS
Follow-up [months]	20.8	21.7	18.9	NS
Aetiology, n (%)				
Unknown	60 (71)	38 (69)	22 (74)	NS
Alcoholic	7 (8)	6 (11)	1 (3)	NS
Inflammation	16 (19)	9 (16)	7 (23)	NS
Family	2 (2)	2 (4)	0 (0)	NS
NYHA class, n (%)				
I	6 (7)	2 (4)	4 (13)	NS
II	49 (58)	29 (53)	20 (67)	NS
III	29 (34)	23 (42)	6 (20)	0.03
Concomitant diseases, n (%)				
Hypertension	20 (23)	10 (18)	10 (33)	NS
Diabetes	7 (8)	5 (9)	2 (7)	NS
Hyperthyroidism	10 (12)	7 (13)	3 (10)	NS
Hyperlipidaemia	37 (43)	24 (44)	13 (43)	NS
Concomitant treatment, n (%)				
Amiodarone	45 (53)	29 (53)	16 (53)	NS
Sotalol	8 (9)	5 (9)	3 (10)	NS
Beta-blocker	58 (68)	37 (67)	21 (70)	NS
Diuretics	54 (63)	39 (71)	15 (50)	0.04
Spironol	33 (39)	23 (42)	11 (37)	NS
ACEI	57 (67)	30 (54)	27 (90)	0.001
Statin	11 (13)	3 (5)	8 (27)	0.008
ECG, n (%)				
QRS ≥ 120 ms	44 (52)	29 (53)	15 (50)	NS
QT dispersion ≥ 100 ms	16 (19)	13 (24)	3 (10)	NS
Cycle length of inducible arrhythmia [ms]	266.6	272.6	256.6	NS
QT [ms]	401.9	401.4	403	NS
QTc [ms]	442.6	441.1	444.7	NS
QT dysp [ms]	58	59.7	55	NS
RR [ms]	857.7	872.3	831.7	NS
QRS [ms]	114.8	113.6	117	NS
Echocardiography, n (%)				
RVDD ≥ 30 [mm]	42 (49)	30 (54)	12 (40)	NS
LVDD ≥ 70 [mm]	40 (47)	27 (49)	13 (43)	NS
EF [%]	30.1	30.4	29.5	NS
LVDD [mm]	68	68.8	66.3	NS
LVSD [mm]	53.5	54.1	52.3	NS
RVDD [mm]	28.5	29.9	25.8	0.008
LA [mm]	45.7	46.5	44.3	NS

Abbreviations: ACEI – angiotensin-converting enzyme inhibitor, LVDD – left ventricular diastolic diameter, LVSD – left ventricular systolic diameter, LA – left atrium, VF – ventricular fibrillation, VT – ventricular tachycardia, RVDD – right ventricular diastolic diameter, EF – ejection fraction

Kaplan-Meier analysis

The Kaplan-Meier curves for the occurrence of VT/VF in patients treated with ACEI versus not treated are shown in Figure 2. Patients receiving ACEI treatment had significantly lower incidence of recurrences of arrhythmic events.

Table II. Univariate predictors of ventricular arrhythmia recurrences

Parameters	Hazard ratio	CI	p
RVDD ≥ 30 [mm]	2.2	1.23-3.92	0.007
QT dispersion ≥ 100 [ms]	1.6	0.8-3.09	NS
QRS ≥ 120 [ms]	1.04	0.5-1.8	NS
LVDD ≥ 70 [mm]	1.25	0.7-2.1	NS
Systemic hypertension	0.77	1.58-	NS
Alcoholic aetiology	1.99	0.94-4.2	0.05
Sustained VT	1.19	0.68-2.09	NS
Diuretic treatment	2.5	1.3-4.5	0.003
ACEI treatment	0.46	0.26-0.8	0.008
Statin treatment	0.42	0.13-1.35	NS
LVDD [mm]	1	0.9-1.04	NS
RVDD [mm]	1.1	1.03-1.16	0.001

Abbreviations: ACEI – angiotensin-converting enzyme inhibitor, LVDD – left ventricular diastolic diameter, RVDD – right ventricular diastolic diameter

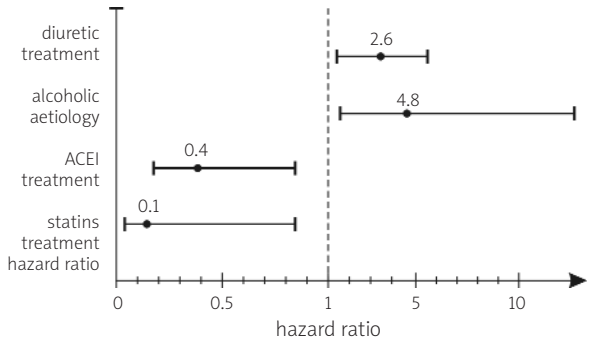

Figure 1. Multivariate predictors of arrhythmia recurrences

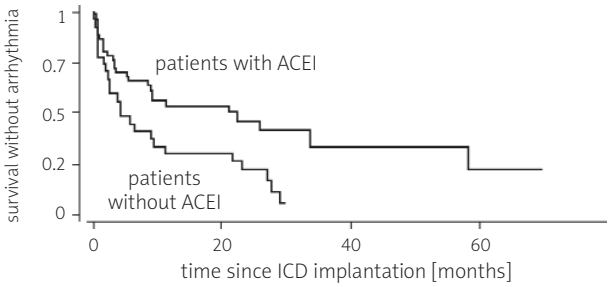

Figure 2. Curves for survival without arrhythmia recurrences in patients treated with ACEI (n = 57) and without ACEI (n = 28)

Discussion

It has been shown recently that prophylactic ICD therapy improved overall survival in selected high-risk post-infarction patients with decreased LVEF. In the setting of DCM, however, arrhythmia risk stratification with regard to prophylactic ICD implantation is still a matter of discussion [8]. The present study found that the majority of patients (65%) with non-ischaemic DCM had recurrences of VT/VF during the mean of 21 months of follow-up. Additional treatment and aetiology of DCM plays an important role in the recurrence of ventricular arrhythmias.

Comparison with previous studies

The incidence of appropriate ICD intervention in our study group was similar to the results of Fazio et al. [9], who found that 62% of patients with DCM received appropriate shocks during a 30-month follow-up. The high rate of appropriate ICD therapy provides indirect evidence that ventricular arrhythmias are a frequent cause of SCD in the natural history of patients with DCM.

Aetiology of DCM

About one fourth of all cases of congestive heart failure in the United States are due to idiopathic DCM [10]. It is likely that idiopathic DCM represents a common expression of myocardial damage that has been produced by a variety of factors such as toxic, immunological or viral ones [11]. Similarly, patients in our study differed in aetiology of DCM, although in over 60% of patients the aetiology of DCM remained unknown. Only alcoholic aetiology of DCM in the present study was strongly associated with recurrences of ventricular arrhythmias. The consumption of excessive amounts of alcoholic beverages can result in toxic effects on the myocardium [12]. This fact is related to the increased prevalence of cardiomyopathy and cardiac arrhythmias. Alcoholic cardiomyopathy is a common diagnosis in chronic alcoholics and is present in 50% of this population [12]. Faris et al. documented a relationship between alcohol intake and cardiac mortality in women but not in men [13]. Greenspon et al. [14] and Rosenquist [15] in their studies suggested a potential association between alcohol and arrhythmias. Alcohol may influence the heart by interaction with mitochondrial metabolism, lipid and protein metabolism and electro-mechanical conjugation modification [15].

Electrophysiological study

Even though EPS has been shown to play a role in identifying high-risk patients with ischaemic cardiomyopathy, programmed ventricular stimulation has not been demonstrated to be useful for SCD risk

stratification in DCM [16]. Knight et al. assessed a group of patients with non-ischaemic DCM, unexplained syncope and negative EPS who were treated with an ICD. Fifty percent of patients had ventricular arrhythmia recurrences, which suggests that the results of EPS have low predictive value in patients with DCM [16]. Also in our study EPS was not useful for prediction of arrhythmia recurrences.

Echocardiographic parameters

One of the major observations is the demonstration of RVDD as an independent predictor of arrhythmia. This parameter emerged as a predictor of arrhythmias in univariate analysis. Similar results were noted by Sun et al. [17], who evaluated the effects of right RV dilation on the progression of LV dysfunction and survival in patients with idiopathic DCM. The authors concluded that patients with significant RV dilation had three times higher mortality rate over four years and more rapidly deteriorating LV function than patients with less initial RV dilation. The most likely explanation for that finding is that patients with RV dilation had more severe heart failure with pulmonary hypertension and RV overload.

Importantly, the present study did not find any relationship between LVEF and intensity of arrhythmic events. This is consistent with data presented by Mehta et al., who reported that long-term outcome in patients with and without recurrent ventricular arrhythmias is similar to patients with moderate to severe LV dysfunction [18]. Also in the CAT study, LVEF did not correlate with the risk of ventricular arrhythmias [19]. Similar results were obtained by Borggrefe et al. [20]. In that study, LVEF was not a predictor of SCD, but was a predictor of total mortality.

Treatment

In our study the use of ACEI was associated with a 60% significant reduction in the relative hazard for ventricular arrhythmia recurrences. This confirmed the results of earlier reports which documented the influence of ACEI treatment on the risk of SCD. Two large trials, AIRE and TRACE, also showed a significant reduction of SCD rate in patients treated with ACEI [21, 22].

A meta-analysis of randomised clinical trials showed a 20% reduction in SCD with the use of ACEI [23]. Our findings are consistent with those of AIRE and TRACE, which showed a reduction of arrhythmia events with ACEI for patients with reduced LVEF.

In our study, diuretic treatment, except spironolactone, was associated with more frequent VT/VF recurrences in a multivariate analysis. Our results are in agreement with previous studies [24, 25], although placebo-controlled randomised studies on the effectiveness of diuretic therapy in patients with ventricular arrhythmias and ICDs are not available. In the retrospective analysis of the SOLVD study, non-potassium-sparing diuretics treatment was significantly associated with fatal arrhythmias in patients with LV dysfunction [24]. In the GESICA study, diuretic treatment was associated with ventricular ectopic activity, which was shown to be an independent factor of SCD in that study [25]. However, it is also possible that diuretic treatment might be just a surrogate for more advanced heart failure.

Only a few investigators have examined the effects of lipid-lowering drugs on cardiac arrhythmias in DCM patients [26, 27]. As in our patients, these studies showed that lipid-lowering drugs decreased the risk of ventricular arrhythmias [26, 27]. De Sutter et al. reported an observational study on 78 patients with VT/VF who were treated with an ICD and found that patients who received lipid-lowering therapy had significantly fewer episodes of recurrent VT/VF than patients who did not receive lipid-lowering therapy [28]. In AVID patients with an ICD, lipid-lowering therapy was associated with a reduction of VT/VF recurrence, suggesting that part of the benefit of statin therapy may be due to an antiarrhythmic effect [29]. Insights from the SCD-HeFT trial showed that the use of statin was associated with reduced all-cause mortality in patients with heart failure [26]. Statins appeared to benefit patients with non-ischaemic and ischaemic cardiomyopathy similarly in ICD and non-ICD patients [26]. Similarly, subanalysis of the MADIT II study revealed an association between the use of statins and reduction of ventricular tachyarrhythmias [30]. Statin use in the DEFINITE study was associated with a 78% reduction in mortality [27]. This reduction was caused, in part, by a reduction of arrhythmic sudden death [27]. These observations support the likelihood that direct or indirect antiarrhythmic effects of statin therapy may contribute to a greater benefit in patients with VT/VF. We observed a 90% reduction of ventricular arrhythmias. However, only 13% of all study patients were on statin therapy, so the effects of these agents could have been overestimated in our study.

Amiodarone is a preferred antiarrhythmic drug in patients with DCM. However, the results of studies evaluating the effects of amiodarone in patients with systolic heart failure showed somewhat conflicting results [31, 32]. The SCD-HeFT study revealed no differences in mortality between amiodarone and placebo in patients with NYHA class II or III. Moreover, in a subgroup of patients with NYHA III amiodarone even worsened survival [32]. Only ICD therapy was associated with a decreased risk of death (by 23% in that study) [32]. Our findings are similar to the SCD-HeFT study: amiodarone treatment revealed only a weak tendency to decrease the risk of arrhythmic events, whereas other arrhythmic drug therapies (sotalol, beta-blockers) have been disappointing.

Study limitations

The major limitation of our study is the fact that it was an observational study and data were collected from a registry. Therefore, there are no data regarding mortality,

heart transplant and other treatment-related outcomes. Similarly, the prescribed drug therapy was based on attending physicians' individual preferences rather than on a standardised treatment protocol. Also, the statin treatment group was small, which could have affected the statistical analysis.

Conclusions

Most patients with non-ischaemic DCM have recurrences of VT/VF. Alcoholic aetiology and diuretic treatment increase the risk of ventricular arrhythmia episodes. Therapy with ACEI and statins decreases the risk of ventricular arrhythmia recurrences.

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Czynniki predysponujące do wystąpienia arytmii komorowych u chorych z kardiomiopatią rozstrzeniową o etologii innej niż niedokrwienna

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Streszczenie

Wstęp: Arytmia komorowa (VA) jest najczęstszą przyczyną nagłej śmierci sercowej (SCD) wśród chorych z nieniedokrwienną kardiomiopatią rozstrzeniową (DCM).

Cel: Ocena czynników ryzyka VA u chorych z DCM.

Metody: Na podstawie danych z Polskiego Rejestru ICD włączono do badania 85 chorych z DCM (73 mężczyzn, średnia wieku 54 lata) z wszczepionymi kardiowerterami-defibrylatorami serca (ICD). Średni okres obserwacji wyniósł 21 ± 19 miesięcy. W grupie badanej 95% osób otrzymało ICD w ramach prewencji wtórnej SCD. Kryteria włączenia do badania obejmowały: dysfunkcję skurczową lewej komory z frakcją wyrzutową (EF) < 40%, wymiar końcowoskurczowy lewej komory (LVSD) > 4,1 cm, wymiar końcoworozkurczowy lewej komory (LVDD) > 5,7 cm oraz negatywny wywiad w kierunku przebytego zawału serca i brak zwężeń > 50% w koronarografii.

Wyniki: Grupa 55 chorych, u których w pamięci ICD zarejestrowano VA, została porównana z grupą 30 chorych bez incydentów arytmii. Obie grupy nie różniły się statystycznie pod względem czynników demograficznych, antropometrycznych, chorób współistniejących ani parametrów elektrokardiograficznych (częstotliwość rytmu serca, szerokość QRS, QT, QTc, dyspersja QT). Osoby, u których wielokrotnie wystąpiły epizody arytmii, znamiennie częściej były w III klasie wg NYHA przed implantacją (p = 0,03). U 44 (53%) badanych wykonano inwazyjne badanie elektrofizjologiczne, podczas którego u 75% indukowano utrwalone częstoskurcze komorowe. W czasie obserwacji nie było znamiennych różnic w występowaniu VA rejestrowanych przez ICD w grupie z indukowalnymi i z nieindukowalnymi arytmiami. Analiza regresji Coksa wykazała, że niezależnymi czynnikami ryzyka VA były: alkoholowa etiologia DCM (p = 0,05), leczenie diuretykiem (p = 0,003), wywiad zatrzymania krążenia (p = 0,03), wymiar końcoworozkurczowy prawej komory (p = 0,001). Stosowanie w leczeniu inhibitorów konwertazy angiotensyny (ACEI) lub statyn wiązało się z tendencją do obniżania ryzyka arytmii. W analizie wieloczynnikowej 4 czynniki uzyskały znamienność statystyczną: etiologia alkoholowa (HR 4,8; p = 0,008), stosowanie ACEI (HR 0,4; p = 0,01), stosowanie diuretyków (HR 2,6; p = 0,015) i leczenie statyną (HR 0,1; p = 0,03).

Wnioski: U większości chorych z DCM występują incydenty VA. Etiologia alkoholowa DCM jest związana ze zwiększeniem ryzyka arytmii. Stosowanie w leczeniu ACEI i statyn zmniejszało ryzyko arytmii.

Słowa kluczowe: kardiomiopatia, arytmia, czynniki ryzyka, wszczepialny kardiowerter-defibrylator, nagła śmierć sercowa

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