Postol A.S.[1], Neminushchiy N.M.[2], Antipov G.N.[1],
Ivanchenko A.V.[1], Lyashenko V.V.[1], Vygovsky A.B.[1], Shnejder Yu.B.[1]

[1] Federal centers of High Medical Technologies, Kaliningrad, Russia

[2] Sechenov First Moscow State Medical University, Moscow, Russia

The Necessity of an ICD-Therapy in Patients with Indications for Primary Prevention of Sudden Cardiac Death. One Center Experience

Aim Analysis of responses of cardioverter-defibrillators implanted in patients with cardiomyopathies (CMPs) of various origins and a high risk of sudden cardiac death (SCD) to assess the effectiveness of a modern strategy for primary prevention of SCD.

Material and methods In the Federal Center for High Medical Technologies in Kaliningrad from 2014 through 2018, implantable cardioverter-defibrillators (ICD) and cardiac resynchronization therapy defibrillators (CRT-D) were installed in 165 patients. Major indications for device implantation in these patients included left ventricular (LV) systolic dysfunction with ejection fraction (EF) ≤35%; chronic heart failure (CHF) consistent with the New York Heart Association (NYHA) functional class (FC) II-III (IV for CRT-D) without previous episodes of life-threatening ventricular arrhythmias, circulatory arrest and resuscitation, which was consistent with the current international strategy for primary prevention of SCD. The study patients were divided into two groups based on the CMP origin; group 1 included 101 (61.2%) patients with CMP of ischemic origin (ICMP) and group 2 consisted of 64 (38.8%) patients with CMP of non-ischemic origin (NCMP). Information about arrhythmic episodes and device activation was retrieved from the device electronic memory during visits of patients to the clinic and was also transmitted to the clinic by a remote monitoring system. This information was studied and evaluated for the validity and effectiveness of the device triggering. If necessary, the parameters of detection and treatment were adjusted taking into account the obtained information. Information was analyzed and statistically processed with the SPSS Statistics 20.0 software.

Results The patients were followed up for 28.3 ± 15.6 months, during which the devices delivered therapy to 55 (33.3%) patients of the entire group. In the ICMP group, the devices were activated in 44 (26.7%) patients and in the NCMP group, the devices were activated in 11 (6.7%) patients. In group 1 (ICMP), appropriate triggering was observed in 33 (20.0%) patients and inappropriate triggering was observed in 11 (6.7%) patients. In group 2 (NCMP), appropriate triggering was observed in 2 (1.2%) patients and inappropriate triggering was observed in 9 (5.5%) patients. The main cause of inappropriate triggering was atrial fibrillation (AF). 17 (10.3%) patients with ICMP had sustained ventricular tachycardia (VT), which did not reach the detection frequency for ICD therapy; these VTs were only detected by devices and terminated spontaneously. Intragroup differences in the number of patients who received an appropriate treatment were statistically significant: 33 (32.6%) in the ICMP group vs. 2 (3.1%) in the NCMP group ($p<0.006$). Differences in the number of patients who received an inappropriate treatment were not statistically significant although their number was greater in the NCMP group than in the ICMP group (9 (14.1%) vs. 11 (10.9%), $p>0.05$).

Conclusion A higher requirement for the ICD treatment was revealed in patients with ICMP compared to patients with NCMP. The low demand for the ICD treatment in patients with NCMP and the more frequent inappropriate actuation of the devices in this patient group due to AF allow a conclusion that the criteria for primary prevention of SCD with ICD (LV EF ≤35% and clinically significant CHF) are not equally effective indications for ICD implantation in patients with ICMP and NCMP. It can be assumed that life-threatening ventricular arrhythmias are evident in patients with NCMP before the development of hemodynamically significant LV dysfunction and CHF, which warrants further research in this direction.

Keywords Ischemic cardiomyopathy; nonischemic cardiomyopathy; sudden cardiac death; implantable cardioverter-defibrillator; cardiac resynchronization therapy defibrillator; appropriate shock; inappropriate shock; atrial fibrillation

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Corresponding author Postol A.S. E-mail: postol-75@mail.ru

Introduction

Sudden cardiac death (SCD) is a death that occurs suddenly and unexpectedly (unpredictable death of a patient) within an hour after the onset of the first clinical symptoms. If death is unwitnessed, the definition allows using a 24-hour time span from the moment when the deceased was in good health before death [1]. Since 2005, there has been a primary prevention strategy for SCD, which, following current clinical guidelines, requires using implantable cardioverter defibrillators (ICDs) in patients without a history of life-threatening ventricular arrhythmias (VA; ventricular tachycardia (VT) with impaired hemodynamics or ventricular fibrillation (VF)), but at risk of developing similar episodes and SCD [1, 2]. Left ventricular (LV) dysfunction (ejection fraction (LVEF ≤35%) and clinically significant chronic heart failure (CHF; functional class (FC) II-III, for cardiac resynchronization therapy defibrillators (CRT-D) FC II-IV according to the New York Heart Association (NYHA) classification) are the main predictors of life-threatening arrhythmias and SCD risk criteria. In this case, ventricular tachyarrhythmias (VT/VF) are the leading mechanism of blood stasis, which in most cases leads to SCD [3, 4]. Coronary artery disease (CAD; up to 80%), various cardiomyopathies (15%) are the most common nosological causes of SCD.Primary electrical disorders of the heart (channelopathies) occur less often (5%) [5–7]. The particular paradox is that the high prevalence of SCD is caused by CAD and usually its stable forms, while diseases associated with a high risk of SCD, such as channelopathies and some cardiomyopathies, are significantly less frequent in the population. This then significantly complicates the identification of individuals at increased risk of SCD.

ICDs are the main means of preventing SCD. The term ICD therapy is used now to mean all the operations (activations) of ICD triggered to interrupt life-threatening ventricular tachyarrhythmias and restore sinus rhythm. According to the existing Russian and international clinical guidelines, ICD therapy has currently class I indications for primary prevention of SCD under the previously mentioned criteria: LVEF ≤35%, CHF NYHA FC II–III in patients with ischemic cardiomyopathy (ICM), and nonischemic cardiomyopathy (NICM) [2]. The question remains whether ICDs are required for primary SCD prevention in patients with NICM [8-10]. This question arose at the beginning of the development of the method, after several studies (CAT [11], AMIOVIRT [12], DEFINITE [9]) to assess the efficacy of ICD therapy in patients with NICM and LV dysfunction. They did not demonstrate a significant reduction in all-cause mortality in patients with implanted ICDs when compared to the control group. In 2016, the DANISH study [13], also did not confirm a significant decrease in all-cause mortality due to the use of ICDs. The groups of patients with NICM are largely heterogeneous in terms of several indicators and, primarily, their arrhythmogenicity and the severity of the risk of SCD. However, in accordance with established international practice, we divided patients into the ICM and NICM groups, in order to assess triggered operations and compare the efficacy of ICD in the groups. We tried to identify other factors which may influence the likelihood of developing life-threatening arrhythmias. For example, we took into account gadolinium-enhanced magnetic resonance imaging (MRI) findings. This method has a high prognostic value in terms of life-threatening VAs and SCD [14–18].

Objective

To analyze triggered ICD operations in patients with ICM of different origins and high risk of SCD, in order to assess the efficacy of the modern strategy of SCD primary prevention.

Material and methods

This study is a retrospective comparison of ICDs and CRT-Ds in 165 patients. The study was conducted in the Federal Center of High Medical Technologies (Kaliningrad, Russia).

Inclusion criteria: CHF NYHA FC II–III (FC II-IV for CRT-D), LVEF ≤35%, ICD or CRT-D, best possible drug treatment of CHF.

Exclusion criteria: history of cerebrovascular accident, transient ischemic attack within 3 months prior to the inclusion, autoimmune and active inflammatory diseases of the myocardium, thyrotoxicosis, life-limiting diseases of up to 1 year, and history myocardial infarction within <40 days.

The patients were divided into two groups: Group 1 included 101 patients (61.2%) with ICM and myocardial

scarring after a heart attack(s); Group 2 comprised 64 (38.8%) patients with NICM.

Electrocardiography and coronary angiography (CAG) were the main methods used in the study performed in all patients to divide them into groups. The presence or absence of specific electrocardiographic post-infarction changes and the presence or absence of specific coronary changes according to CAG allowed to group the patients. All patients underwent echocardiography, in order to assess cardiac dimensions, global and regional wall motion, and assess dyssynchrony of myocardial contractions. Most patients (121 (73.3%)) underwent delayed gadolinium-enhancement MRI of the heart. Clinical characteristics of patients are presented in Table 1.

ICDs (including CRT-Ds) were implanted according to the indications defined in the 2017 clinical guideline [2]. The following devices were implanted: Lumax 340 VR – 45 (27.3%), Lumax CRTD – 1 (0.6%) and Lumax 540 VR – 3 (1.8%), Teligen-100 – 14 (8.4%); Protecta CRTD – 57 (34.5%), Protecta DR – 22 (13.3%), Protecta VR – 10 (6.0%), Maximo II CRTD – 5 (3.0%), Maximo II DR – 4 (2.4%), Maximo II VR – 4 (2.4%). The devices were programmed in the standard manner following manufacturers' recommendations and the 2019 Expert Consensus Statement on Optimal Implantable Cardioverter-Defibrillator Programming and Testing [19, 20]. The heart rhythm monitoring zone was activated (from 100–130 bpm to the VF detection rate). The lower limit of the VF detection and treatment was 188–200 bpm. In most cases, anti-tachycardia pacing (ATP) could be used in the VF detection zone in case of the detection of monomorphic VT. Special algorithms-discriminators were programmed to allow the devices to distinguish VT from supraventricular tachycardia, in order to reduce the probability of unsuitable device activation. The analysis of QRS complex morphology was most commonly used.

The follow-up period was 28.3±15.6 months, during which patients were monitored using a combination of patient visits to hospital and remote monitoring in 100 (60.6%) patients and personal visits only without remote monitoring in 65 (39.4%) patients. The information on arrhythmia episodes and ICD/CRT-D activation were retrieved from the device memory. The stored endograms were used to evaluate the efficacy of ICD therapy and, if necessary, change the parameters of ICD.

The data obtained was statistically processed using SPSS Statistics 20.0. The results are presented as the mean (± standard deviation) or the absolute number and the

Table 1. Clinical characteristics of patients and types of implanted devices

Indicator	All patients (n=165)	Patients with ICM (n=101)	Patients with NICM (n=64)	p
Age, years	63.5±9.8	65.67±8.0	60.30±11.4	0.480
Male/female	135 (81.9)/30 (18.1)	95 (94.0)/6 (6)	43 (67.2)/21 (32.8)	0.070
Non-sustained VT	68 (40.4)	62 (61.9)	29 (48.4)	0.122
VPB	132 (80.0)	85 (84.1)	44 (68.7)	0.720
AF	51 (30.9)	45 (44.6)	29 (45.3)	0.251
CHF	140 (84.8)	84 (84.8)	56 (87.5)	0.657
NYHA FC II	32 (19.4)	25 (78.1*)	7 (21.9*)	0.150
NYHA FC III	78 (47.2*)	39 (50*)	39 (50*)	0.511
NYHA FC IV	30 (18.3*)	12 (40*)	18 (60*)	0.307
MRI performed	121 (73.3)	78 (77.2*)	43 (67*)	0.970
Arrhythmogenic substrate	79 (47.9)	74 (96.1)	5 (11.6)	0.004
Coronary angiography	165 (100)	101 (100)	64 (100)	0.002
CABG	63 (38.0)	63 (100*)	0	0.284
Types of implantable devices				
CRT-D	63 (38.1)	22 (35*)	41 (65.0*)	0.011
ICD DR	40 (24.8)	33 (82.5*)	7 (17.5*)	0.004
ICD VR	62 (38.7)	45 (72.3*)	17 (27.7*)	0.006

The data is expressed as the absolute number of patients (%), unless otherwise is specified. ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy; VT, ventricular tachycardia; VPB, ventricular premature beats; AF, atrial fibrillation; CHF, chronic heart failure; FC, functional class; NYHA, New York Heart Association; MRI, magnetic resonance imaging; CABG, coronary artery bypass grafting; CRT-D, cardiac resynchronization therapy defibrillator; ICD, implantable cardioverter-defibrillator. ICD-DR, dual-chamber implantable cardioverter-defibrillator; ICD-VR, single-chamber implantable cardioverter-defibrillator.
*, the percentage of patients in the ICM or NICM group, but not the percentage of the total number of patients included in the study.

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Table 2. Device activations, arrhythmia episodes, and antiarrhythmic therapy over the follow-up period

Indicator	All patients (n=165)	Patients with ICM (n=101)	Patients with NICM (n=64)	p
Patients with adequate pacing	35 (21.2)	33 (20.0)	2 (1.2)	0.006
Patients with inadequate pacing	20 (12.1)	11 (6.6)	9 (5.5)	0.247
Patients with SMVT	52 (31.5)	50 (30.3)	2 (1.2)	0.005
Patients with detected VT	35 (21.2)	33 (20.0)	2 (2.1)	0.005
SMVT resolved spontaneously	17 (10.3)	17 (10.3)	–	0.001
Required significant changes in the device configuration	45 (27.2)	36 (21.8)	9 (5.5)	0.051
Beta-blockers	155 (93.9)	95 (57.6)	60 (36.3)	0.105
Amiodarone	26 (15,7)	24 (14.5)	2 (1.2)	0.051
Total activations	55 (33.3)	44 (26.7)	11 (6.7)	0.053
Effective ATP	13 (7.9)	11 (6.7)	2 (1.2)	–
Electric shocks	22 (13.3)	22 (13.3)	–	0.001
Inadequate activations	20 (12.1)	11 (6.7)	9 (5.5)	0.247

ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy; SMVT, sustained monomorphic ventricular tachycardia; VT, ventricular tachycardia; VF, ventricular fibrillation; AF, atrial fibrillation; ATP, anti-tachycardia pacing.

percentage (n (%)). The univariate analysis of variance (ANOVA) and the Scheffe test were used to assess the differences between groups in continuous metrics. The chi-squared test and Fisher's exact test were used to analyze the categorical variables. Pearson's correlation coefficient was used to assess correlations between the quantitative variables. The differences were statistically significant at $p<0.05$. The cumulative intergroup differences in device activations over time were assessed using the Kaplan–Meier test to evaluate the cumulative survival function and construct the survival curves.

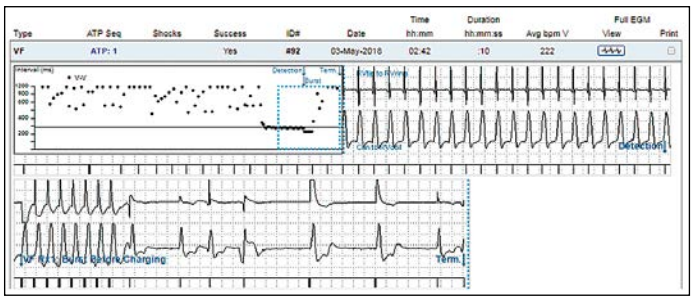
Results

The follow-up period was 28.3 ± 15.6 months, during which 9 (5.5%) patients died: 7 (4.2%) patients in the ICM group and 2 (1.2%) patients in the NICM group. There were no SCDs in either group. The causes of death were the progression of CHF in 6 (3.63) cases and non-cardiac causes in 3 (1.8%) cases. During the follow-up period, 55 (33.3%) patients in the entire group experienced the activation of the implanted devices (Table 2). Device activation was reported in 44 (26.7%) patients with ICM and 11 (6.7%) patients with NICM. In Group 1, adequate and inadequate activations were reported in 33 (20.0%) and 11 (6.7%) patients, respectively. In Group 2 (NICM), adequate and inadequate activations were observed in 2 (1.2%) and 9 (5.5%) patients, respectively. The main cause of the inadequate activations was atrial fibrillation (AF) ($p<0.001$). Seventeen (10.3%) patients had sustained VT below the ICD detection rate which was only recorded by the devices and resolved spontaneously. All 17 (10.3%) patients had ICMs. The differences in the number of patients who received adequate pacing are statistically significant ($p<0.006$) in both groups. The differences in the number of patients who received inadequate pacing are statistically insignificant in both groups.

Analysis of the device activations and arrhythmia episodes in the ICM and NICM groups

In the ICM group (n=101), 50 (30.3%) ICDs detected VAs, of which 33 (20.0%) were life-threatening VAs in the VF detection area: VTs with a less than 270 ms (more than 220 per minute) cycle in 8 (4.8%) patients; VT with a lower rate and cycle duration of 330 to 270 ms (180 to 220 per minute) in 25 (15.1%) patients. All these episodes ended with effective device activation (Figure 1 and Figure 2). Seventeen 17 (10.3%) patients with ICD had sustained VTs in the monitor area with cycle duration of more than 330 ms (less than 180 per minute) with spontaneous resolution of paroxysms. Another 41 (24.8%) patients had unstable VAs in the monitor area for up to 8 seconds. All patients with VAs detected by cardiac MRI had myocardial fibrosis in a potentially arrhythmogenic structure and a history of myocardial infarction (n=50, 100%). Eleven (6.7%) patients had non-life-threatening supraventricular arrhythmias in the VF detection area due to atrial fibrillation and flutter (Figure 3). In the ICM group, 22 (13.3%) patients received adequate pacing to correct VAs (n=102 episodes). In eleven (6.7%) patients, monomorphic VTs were corrected by applying ATP during or before charging the defibrillator (n=31 episodes). Eleven (6.7%) patients received inadequate pacing (n=30 exposures). AF was the most common cause of inadequate pacing. Two (1.2%) patients reported multiple activations: 8 and 10 shocks per day. Clinically significant differences between patients with ICM who received and did not receive ICD pacing were the presence of high-grade ventricular

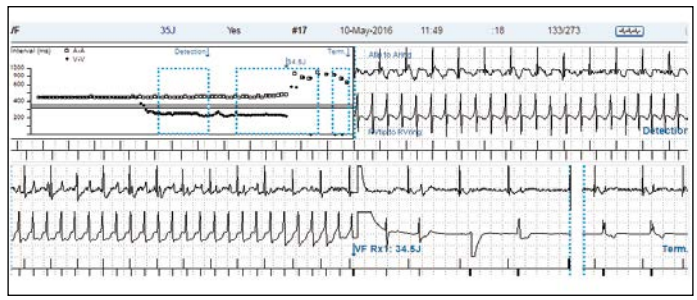
Figure 1. Recording of a monomorphic ventricular tachycardia episode registered via the remote monitoring system from a single-chamber ICD memory



Ventricular tachycardia is higher than 220 bpm. The black dots in the upper left corner show intervals between heart rhythm complexes. The vertical axis represents the cycle duration in milliseconds of ventricular rhythm (the shorter the cycle, the higher the rate). The black line limits the detection zone. The initial rhythm is chaotic, probably due to premature beats. Then, the rhythm turns into sustained ventricular tachycardia. A pack (sequence of stimuli) of anti-tachycardia pacing with subsequent restoration of the ventricular rhythm is visible. The endogram includes a record of two channels: the near field (between the electrode tip and the ring, adhesions without morphology are visible) and the far field (between the ICD body and the shock spiral of the electrode, the morphology of QRS complexes is visible). At the bottom of the picture, antitachycardia pacing restores the normal ventricular rhythm.

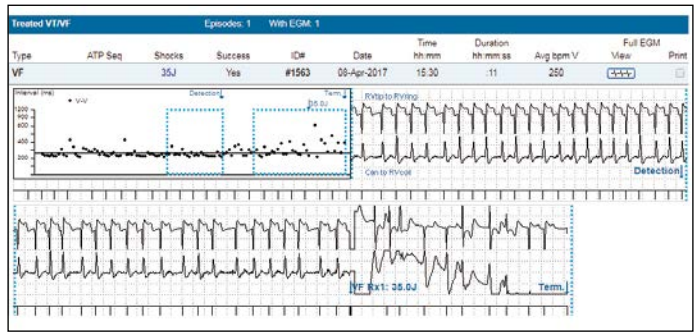
ICD, implantable cardioverter defibrillator.

Figure 2. Recording of a monomorphic ventricular tachycardia episode registered via the remote monitoring system from a dual-chamber ICD memory



Ventricular tachycardia is more than 250 bpm. The upper channel is the atrial rhythm (normal rate). The lower channel is the ventricular rhythm (ventricular tachycardia). The rhythm is restored after an electric shock of 34.5 J.
ICD, implantable cardioverter defibrillator.

Figure 3. Recording of an atrial fibrillation episode registered via the remote monitoring system from a single-chamber ICD memory



The graph and endogram show irregular ventricular rhythm due to an episode of atrial fibrillation erroneously detected by the ICD as ventricular fibrillation, mainly due to a high ventricular rate of about 250 bpm. ICD discharges 35 J.
ICD, implantable cardioverter defibrillator.

ectopia (p=0.029) and the absence of amiodarone therapy (p=0.002).

In the NICM group (n=64), two (2.1%) patients had an episode of monomorphic VTs (cycle duration less than 270 ms, frequency more than 220 per minute), which was corrected using ATP. Both patients had pronounced myocardial fibrosis with arrhythmogenic heterogeneity according to gadolinium-enhanced MRI (Figure 4). In both cases, the cause of NICM was post-myocarditis cardiosclerosis due to the previous myocarditis. Nine (5.5%) patients with NICM reported inadequate activations of ICD due to episodes of tachysystolic AF. During these episodes, the ventricular rate reached the VF detection zone resulting in inadequate shocks despite the use of discriminating algorithms. A total of 101 inadequate shocks were produced.

In both patient groups, the main causes of inadequate pacing were AF episodes with a high ventricular rate within the VF detection area. Inadequate exposures were more commonly reported in male patients (p=0.041) and patients with single-chamber ICDs (p=0.048).

Thus, VAs occurred significantly more frequently in ICM patients with a history of myocardial infarction than in those with NICM. Accordingly, the demand for ICD therapy in patients of Group 1 was significantly higher than in Group 2, 33 (32.6%) versus 2 (3.1%); p<0.006. By the end of the follow-up period, there were 68 (67.3%) patients without adequate ICD activations in the ICM group and 62 (96.9%) in the NICM group (Figure 5). According to gadolinium-enhanced MRI, potentially arrhythmogenic myocardial fibrosis was detected in all patients with VAs and adequate defibrillator activations (Figure 1).

Discussion

In this study, we monitored patients with implanted defibrillators (ICD and CRT-D) for primary SCD prevention under the existing SCD risk criteria (LV≤35%, CHF NYHA FC II-III; FC II-IV in case of for SRT-Ds). Patients were divided into groups of ICM and NICM in accordance with international practice. The study showed a significant need for ICD therapy in Group 1 (ICM patients). There were also fewer inadequate activations in this group. It was not statistically significant, but it is possible that differences can be significant in a more extended follow-up period. In all previous years since the appearance (2005) of

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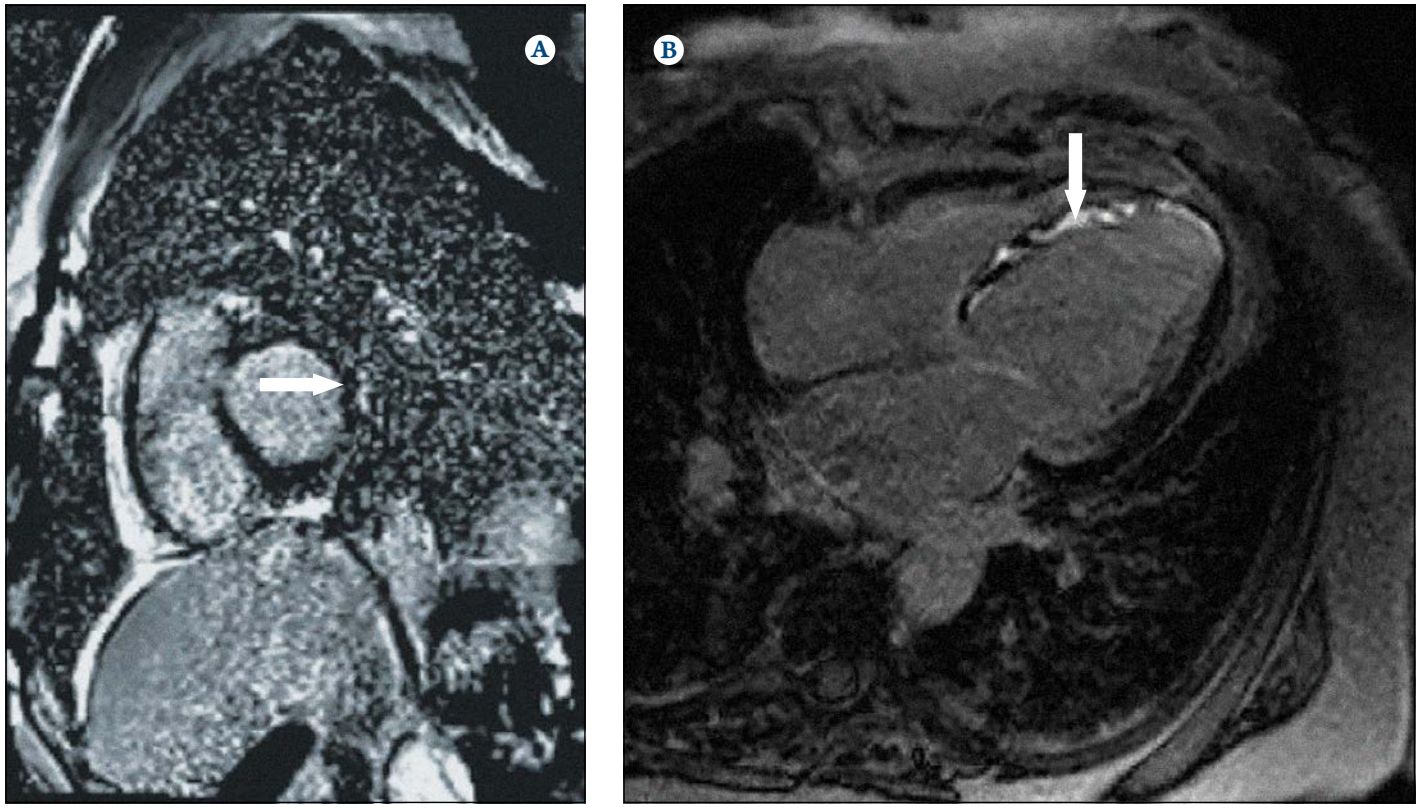
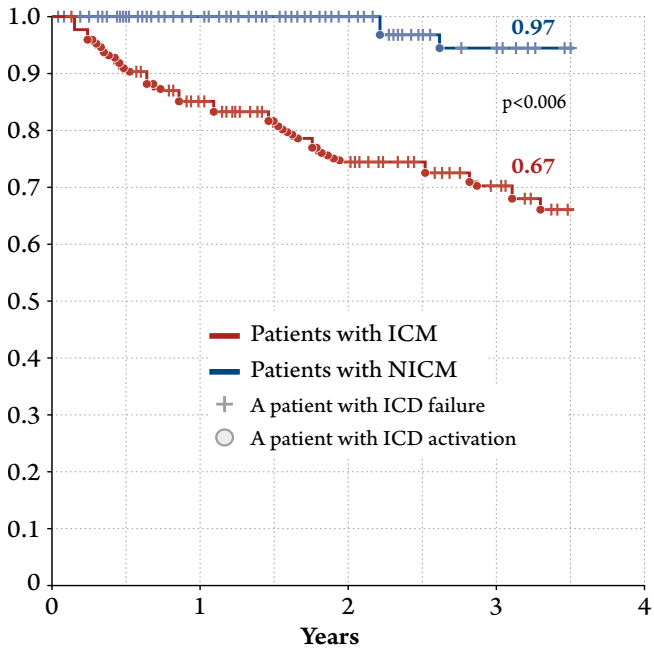
Figure 4. Gadolinium-enhanced cardiac MRI



A – MRI of a patient with nonischemic cardiomyopathy; **B** – MRI of a patient with ischemic cardiomyopathy. The arrows show the areas of fibrosis, i.e., arrhythmogenic substrates characterized by tissue heterogeneity, inclusions, and gray zone. Potential risk zones of ventricular tachyarrhythmias. MRI, magnetic resonance imaging.

Figure 5. Kaplan–Meyer curves indicating a lower number of patients with ICD failure to pace in groups with ischemic cardiomyopathy and nonischemic cardiomyopathy during the follow-up period



The groups differ to a statistically significant degree by an increase in the number of patients with adequate ICD episodes. ICD, implantable cardioverter-defibrillator; ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy.

indications for using ICD in the primary prevention of SCD, the discussion has continued about whether it is reasonable to use them in patients with NICM under the above criteria. As discussed earlier, the NICM group ischaracterized by pronounced heterogeneity in the course of diseases and their arrhythmogenicity, and thus, the risk of SCD. Clearly, the degree of this risk changes over the course of the disease. Our study confirms the low prognostic value of the criteria mentioned in terms of the risk of arrhythmia episodes in patients with NICM, and the results of studies of the efficacy of ICD in such patients: CAT, AMIOVIRT, DEFINITE, DANISH [9–13].

It has become clear that grouping NICM patients contradicts the principles of the etiopathogenetic treatment approach. This approach in international practice may be due to the modern health care system, in which the surgical objective is to resolve and/or prevent arrhythmia, rather than determine the cause of the disease and establish the nosological diagnosis.There is neither the time nor the resources for this. In a seemingly homogeneous group of patients with idiopathic dilated cardiomyopathy, at least five different genotypes with peculiar clinical manifestations are known. However, as has been recently published [21–26] and shown by our experience, VAs manifested very often during the

disease. In most cases, VAs occur earlier than clinically significant LV dysfunction and CHF. These patients are usually younger than those with ICM. Given all of this, the use of SCD risk criteria based on LVEF and CHF is not effective for in this patient category, since by the time they develop CHF and LVEF decrease to 35%, the risk of dying suddenly of VT/VF will be minimal. The main risk of death will be associated with progression of CHF. It should also be noted that the progression of CHF increases the likelihood of developing AF, which increases the probability of inadequate pacing. Many patients with NICM are thought to die of life-threatening arrhythmias even before the development of clinically significant LV dysfunction, while patients who survive to its development are at relatively low risk of arrhythmic death and higher risk of dying of CHF.

Unfortunately, we were unable to perform gadolinium-enhanced MRI in all our patients, in order to work with statistically significant data. However, NICM patients who had episodes of VAs had myocardial fibrosis of a specific potentially arrhythmogenic structure. Several recent publications [14–18, 27] suggest the use of the presence of potentially arrhythmogenic patterns of myocardial fibrosis shown by gadolinium-enhanced MRI and other myocardial imaging techniques. They can become commonly used as the criteria for primary prevention of SCD in patients with nonischemic arrhythmic heart diseases. The imaging of myocardial fibrosis is a remarkable achievement of our time that allows more accurately determining the risk of fatal arrhythmias and SCD even before their development.

Conclusion

Our study showed that patients with ischemic cardiomyopathy have a higher need for pacing with implantable cardioverter defibrillators than patients with nonischemic cardiomyopathy ($p<0.006$). We observed a low efficacy of traditional selection criteria (left ventricular ejection fraction ≤35%, chronic heart failure of functional class II-III, and chronic heart failure of functional class II-IV for cardiac resynchronization therapy with defibrillators) in the primary prevention of sudden cardiac death with implantable cardioverter-defibrillators in patients with nonischemic cardiomyopathies. The low demand for pacing with implantable cardioverter-defibrillators in patients with nonischemic cardiomyopathies, and more frequent inadequate pacing in this group due to atrial fibrillation, suggests that the criteria for the primary prevention of sudden cardiac death using implantable cardioverter defibrillators (left ventricular ejection fraction ≤35% and clinically significant chronic heart failure) are not similarly effective indications for the use of implantable cardioverter-defibrillators in patients with ischemic and nonischemic cardiomyopathies. Life-threatening ventricular arrhythmias can be thought to manifest in patients with nonischemic cardiomyopathy, before the development of hemodynamically significant left ventricular dysfunction and chronic heart failure. Further research is required in this area.

The author of the article, N.M. Neminushchiy, declares a conflict of interest in connection with conducting educational events for the company Medtronic for compensation.

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