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Review article

European guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death 2022: cardiomyopathy. What's new?

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Abstract

The review provides information on new indications that should be guiden the diagnosis and treatment of ventricular arrhythmias in patients with cardiomyopathy. The analysis of modern definitions and classifications of cardiomyopathy is given. The issues of ventricular arrhythmias in different cardiomyopathy phenotypes, risk stratification of sudden cardiac death and its prevention are considered in detail.

Keywords: ventricular arrhythmias; sudden cardiac death; dilated cardiomyopathy; hypertrophic cardiomyopathy; arrhythmogenic cardiomyopathy; restrictive cardiomyopathy; non-compaction cardiomyopathy.

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Обзорная статья

Европейские рекомендации по лечению пациентов с желудочковыми аритмиями и профилактике внезапной сердечной смерти 2022 года: кардиомиопатии. Что нового?

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Аннотация

В обзоре представлена информация о новых показаниях, которыми следует руководствоваться при диагностике и лечении желудочковых нарушений ритма у пациентов с кардиомиопатиями. Дан анализ современных определений и классификаций кардиомиопатий. Подробно рассмотрены вопросы желудочковых нарушений ритма при разных фенотипах кардиомиопатий, стратификации риска внезапной сердечной смерти и ее профилактики.

Ключевые слова: желудочковые аритмии; внезапная сердечная смерть; дилатационная кардиомиопатия; гипертрофическая кардиомиопатия; аритмогенная кардиомиопатия; рестриктивная кардиомиопатия; некомпактный миокард желудочков.

Как цитировать

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In the new European recommendations for treating patients with ventricular arrhythmias (VAs), a large section is devoted to cardiomyopathies (CMPs). Cardiac arrhythmias are perhaps one of the key clinical symptoms of these relatively rare diseases, and sudden cardiac death (SCD) is the classic complication of most CMPs.

This section focuses on CMPs in the order they are presented in European recommendations.

Dilated cardiomyopathy (DCM)

DCM is characterized by dilatation and systolic dysfunction of the left ventricle or both ventricles and is not associated with ischemic heart disease (IHD) or abnormal hemodynamic loads (such as arterial hypertension and valvular disease) [1, 2]. SCD occurs in 12% of patients with DCM and accounts for 25%–35% of the overall cause of death in DCM [3–5].

According to the literature, DCM affects 1 per 2500–2700 populations [6, 7]. However, the true incidence is most likely higher.

The causes of the appearance of DCM in a patient can be genetic, acquired, or mixed when a genetic predisposition is formed in the presence of external factors, for example, the pre- and postpartum period, alcohol abuse, chemotherapy, and others [8]. Pathogenic mutations are detected in 25–55% of patients with DCM, with a predominant autosomal dominant type of inheritance [2]. More often than others, mutations are found in the titin (*TTN*) (31%) and lamin (*LMNA*) (14.3%) genes [9]. Mutations in genes such as *LMNA, PLN* (encodes the phospholamban protein), *RBM20* (encodes the splicing transcription factor), and *FLNC* (encodes filamin C) are associated with a high risk of VAs and SCD [10–14]. Carriers of desmosomal and *LMNA* mutations have a high incidence of VAs and SCD, which does not depend on the LV ejection fraction (EF) [14]. The identification of pathogenic mutations plays an important role in SCD risk stratification.

The phenotype, particularly in a genetically determined disease, upon disease onset may not correspond to the standard criteria for the disease and may change over time. Thus, in the early disease stages, the patient may only have a decrease in EF without dilatation of the cardiac chambers. In this regard, a new category of DCM has been proposed, that is, hypokinetic nondilated cardiomyopathy (HNDCM) [8]. HNDCM is characterized by LV or biventricular global systolic dysfunction (LVEF <45%) without dilatation, and systolic dysfunction is not associated with abnormal myocardial stress or coronary artery disease [8]. The 2023 European guidelines for the management of patients with CMPs made a special attention to this syndrome. It has been proposed to initially replace the term "hypokinetic nondilated cardiomyopathy" with "nondilated left ventricular cardiomyopathy" (NDLVCMP) and then to identify NDLVCMP as a new independent phenotype of CMPs [15]. The term "NDLVCM" introduced in 2023 appears to be a broader concept than HNDCM. NDLVCMP is characterized by the presence of LV nonischemic scarring or fatty degeneration, regardless of the presence or absence of global or local impairment of wall mobility or isolated global hypokinesia of the LV walls without scarring. This disease is also characterized by ventricular rhythm disturbances.

An integrated approach to diagnosing DCM is essential. The role of echocardiography (EchoCG), particularly in the early disease stages, is undeniable. Magnetic resonance imaging (MRI) of the heart with contrast enhancement enables not only to determine LVEF but also to identify areas of fibrosis and, by their localization, clarify the etiology (ischemic, i.e., subendocardial, transmural fibrosis, corresponding in localization to the blood supply system of a certain coronary artery; nonischemic, i.e., diffuse interstitial intramural, subepicardial fibrosis, or subendocardial, but not corresponding to the blood supply system of a particular coronary artery) [16, 17]. In addition, MRI findings, along with genetic data, can contribute to SCD risk stratification [2]. According to a meta-analysis of 29 studies that pooled MRI findings from 2,948 patients with DCM, late gadolinium enhancement (LGE) is associated with an increased risk of arrhythmic endpoints (VA and SCD), major cardiovascular events, and all-cause death [18]. A recent study of 1020 patients with DCM revealed that both LGE and LVEF were risk markers for all-cause and cardiovascular deaths; however, only LGE was associated with the risk of SCD [19]. The recommendations emphasize the importance of performing cardiac MRI with contrast in patients with DCM/HNDCM.

The registration of electrocardiograms (ECG) is recommended not only for patients but also for their first-degree relatives. The presence of pathology of the sinus and atrioventricular (AV) nodes, most often in combination with bundle branch blocks, when the disease manifests at a young age should raise suspicion of *LMNA*-associated DCM with a poor prognosis [20].

The recommendations for genetic testing are presented in Table 1 [2].

The recommendations for examining patients and their relatives are presented in Table 2 [2].

The 2022 European guidelines provide an algorithm for risk stratification and primary prevention of sudden cardiac death in patients with DCM/HNDCM (Fig. 1) [2].

Given the high risk of SCD, patients with DCM need not only secondary but also primary prevention of SCD. The 2022 European guidelines provide clear guidance on who is eligible for primary and secondary prevention of SCD. Because the main clinical manifestation of DCM/HNDCM is chronic heart failure (CHF), treatment of CHF, in accordance with current recommendations,

is mandatory for at least 3 months before deciding on implantable cardioverter-defibrillator (ICD) implantation for the primary prevention of SCD [2]. The patient's cardiac function and clinical status after 3 months of optimal medical therapy (OMT) must be re-evaluated before the initial prophylactic implantation of an ICD. LVEF with OMT can be significantly improved in DCM caused by myocarditis or TTN mutations [2].

The recommendations for the primary prevention of SCD are presented in Table 3 [2].

The recommendations for the secondary prevention of sudden cardiac death are presented in Table 4 [2].

ICDs reduce the risk of not only arrhythmic death but also death from all causes [2]. Moreover, frequent, painful ICD shocks worsen the quality of life of the patients. The implantation of ICDs with antitachycardia pacing (ATP)

Table 1. Recommendations for genetic testing for dilated/hypokinetic nondilated cardiomyopathy
Таблица 1. Рекомендации по генетическому тестированию при дилатационной / гипокинетической недилатационной кардиомиопатии

Recommendation	Indication class	Evidence level
Genetic testing including at least the *LMNA*, *PLN*, *RBM20*, and *FLNC* genes is recommended for patients with DCM/HNDCM and – disorders of atrioventricular conduction under the age of 50 years; or – with a family history of DCM/HNDCM or cases of sudden cardiac death in a first-degree relative (aged < 50 years)	I	B
Genetic testing including at least the *LMNA*, *PLN*, *RBM20*, and *FLNC* genes should be recommended for risk stratification in patients with overt sporadic DCM/HNDCM diagnosed at a young age or when the patient has signs suggestive of a hereditary etiology of the disease	IIa	C

Note: DCM — dilated cardiomyopathy; HNDCM — hypokinetic nondilated cardiomyopathy; *LMNA* — nuclear lamin gene; *PLN* — phospholamban gene, *RBM20* — gene encoding a splicing transcription factor; *FLNC* — filamin C gene.

Примечание: ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; *LMNA* — ген ядерных ламинов; *PLN* — ген фосфоламбана, *RBM20* — ген, кодирующий транскрипционный фактор сплайсинга; *FLNC* — ген филамина С.

Table 2. Recommendations for the examination of patients suffering from dilated/hypokinetic non-dilated cardiomyopathy and their relatives
Таблица 2. Рекомендации по обследованию пациентов, страдающих дилатационной / гипокинетической недилатационной кардиомиопатией, и их родственников

Recommendation	Indication class	Evidence level
Cardiac MRI with late gadolinium enhancement should be recommended in patients with DCM/HNDCM to evaluate the etiology and risk of ventricular arrhythmia/SCD.	IIa	B
In patients with DCM/HNDCM, electrophysiological testing should be recommended if there is a history of syncope, and the cause remains unexplained after noninvasive evaluation.	IIa	B
First-degree relatives of patients with DCM/HNDCM are advised to undergo an ECG and echocardiogram if – the diagnosis was established in an index patient aged < 50 years or clinical signs indicating a hereditary cause of the disease are present; or – the presence of DCM/HNDCM or premature SCD in the family history	I	C
An ECG and echocardiogram may be recommended for first-degree relatives of patients with apparently sporadic DCM/HNDCM	IIb	C

Note: SCD — sudden cardiac death; DCM — dilated cardiomyopathy; HNDCM — hypokinetic nondilated cardiomyopathy; MRI — magnetic resonance imaging.

Примечание: ВСС — внезапная сердечная смерть; ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; МРТ — магнитно-резонансная томография.

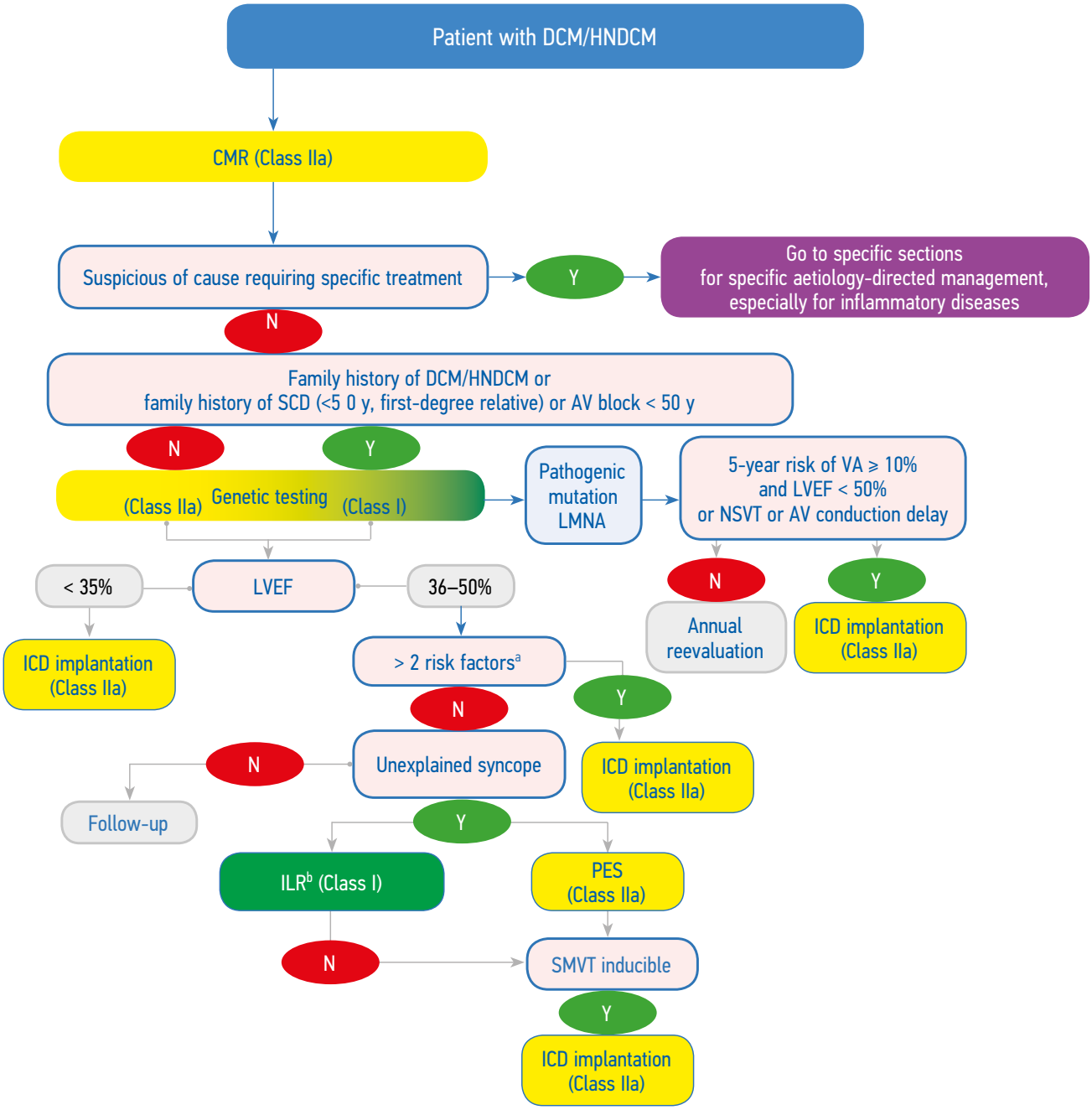

Fig. 1. Algorithm for risk stratification and primary prevention of sudden cardiac death in patients with dilated cardiomyopathy/hypokinetic non-dilated cardiomyopathy [2]

AV — atrioventricular; CMR — cardiac magnetic resonance; DCM — dilated cardiomyopathy; HNDCM — hypokinetic non-dilated cardiomyopathy; ICD — implantable cardioverter defibrillator; ILR — implantable loop recorder; LMNA — nuclear lamin gene; LVEF — left ventricular ejection fraction; N — no; NSVT — non-sustained ventricular tachycardia; PES, programmed electrical stimulation; SCD — sudden cardiac death; SMVT — sustained monomorphic ventricular tachycardia; VA — ventricular arrhythmias; Y — yes

[a] Risk factors: unexplained syncope, pathogenic variants in PLN, FLNC, or RBM20, LGE on CMR, inducible SMVT at PES. [b] The 2018 ESC Guidelines for the diagnosis and management of syncope

Рис. 1. Алгоритм стратификации риска и первичной профилактики внезапной сердечной смерти у пациентов с дилатационной кардиомиопатией / гипокинетической недилатационной кардиомиопатией [2].

АВ — атриовентрикулярная; ВСС — внезапная сердечная смерть; ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; ЖА — желудочковые аритмии; ИПР — имплантируемые петлевые регистраторы; ИКД — имплантируемый кардиовертер-дефибриллятор; ЛЖ — левый желудочек; МРТ — магнитно-резонансная томография; НУЖТ — неустойчива желудочковая тахикардия; УМЖТ — устойчивая мономорфная желудочковая тахикардия; ФВ — фракция выброса; ЭФИ — электрофизиологическое исследование; *LMNA* — ген ядерных ламинов

[a] Обмороки, фиброз при МРТ сердца, индуцируемые устойчивые мономорфные желудочковые тахикардии при эндоЭФИ, патогенные мутации в *LMNA*, *PLN*, *FLNC* и генах *RBM20*. [b] Согласно рекомендациям ESC 2018 года по диагностике и лечению обмороков

Таблица 3. Рекомендации по первичной профилактике внезапной сердечной смерти у пациентов, страдающих дилатационной / гипокинетической недилатационной кардиомиопатией
Table 3. Recommendations for the primary prevention of sudden cardiac death in patients suffering from dilated/hypokinetic nondilated cardiomyopathy

Recommendation	Indication class	Evidence level
ICD implantation should be recommended in patients with DCM/HNDCM, symptomatic heart failure (NYHA classes II–III), and LVEF ⩽35% after ⩾3 months of OMT	IIa	A
ICD implantation should be recommended for patients with DCM/HNDCM who have a pathogenic *LMNA* mutation if the estimated 5-year risk of life-threatening ventricular arrhythmias is ⩾10% and in the following cases: – unstable ventricular tachycardia; or – LVEF <50%; or – disorders of AV conduction	IIa	B
ICD implantation should be recommended for patients with DCM/HNDCM, LVEF <50%, and presence of ⩾2 risk factors: – syncope – late gadolinium enhancement in cardiac MRI – inducible sustained monomorphic ventricular tachycardia with endoEPS (electrophysiological study) – pathogenic *LMNA*, *PLN*, *FLNC*, and *RBM20* mutations	IIa	C

Note: AV — atrioventricular; CMR — cardiac magnetic resonance; DCM — dilated cardiomyopathy; HNDCM — hypokinetic non-dilated cardiomyopathy; EF — ejection fraction; ICD — implantable cardioverter defibrillator; LV, left ventricul; NYHA, New York Heart Association; LMNA — nuclear lamin gene; PES — programmed electrical stimulation; PLN — phospholamban gene; RBM20 — gene encoding a splicing transcription factor; FLNC — filamin C gene. [a] Based on the risk calculator LMNA-risk VTA calculator Risk Prediction Score for Life-Threatening Ventricular Tachyarrhythmias in Laminopathies (https://lmna-risk-vta.fr/).

Примечание: АВ — атриовентрикулярное; ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; ИКД — имплантируемый кардиовертер-дефибриллятор; ЛЖ — левый желудочек; МРТ — магнитно-резонансная томография; ОМТ — оптимальная медикаментозная терапия; ФВ — фракция выброса; ЭФИ — электрофизиологическое исследование; NYHA — New York Heart Association; *LMNA* — ген ядерных ламинов; *PLN* — ген фосфоламбана, *RBM20* — ген, кодирующий транскрипционный фактор сплайсинга; *FLNC* — ген филамина С. [a] Риск рассчитывается с помощью калькулятора LMNA-risk VTA calculator Risk Prediction Score for Life-Threatening Ventricular Tachyarrhythmias in Laminopathies (https://lmna-risk-vta.fr/)

Таблица 4. Рекомендации по вторичной профилактике внезапной сердечной смерти у пациентов, страдающих дилатационной / гипокинетической недилатационной кардиомиопатией
Table 4. Recommendations for secondary prevention of sudden cardiac death in patients suffering from dilated/hypokinetic nondilated cardiomyopathy

Recommendation	Indication class	Evidence level
ICD implantation is recommended for patients with DCM/HNDCM who have experienced sudden circulation arrest due to ventricular tachycardia/ventricular fibrillation or hemodynamically intolerable sustained monomorphic ventricular tachycardia	I	B
Catheter ablation in specialized centers should be recommended for patients with DCM/HNDCM and recurrent, symptomatic, sustained monomorphic ventricular tachycardia, frequent ICD shocks for sustained monomorphic ventricular tachycardia, and if antiarrhythmic drugs are ineffective, contraindicated, or intolerant	IIa	C
The addition of amiodarone orally or substitution of a beta blocker with sotalol should be recommended in patients with DCM/HNDCM and ICD who experience recurrent, symptomatic ventricular arrhythmias despite optimal device programing and beta-blocker treatment	IIa	B
ICD implantation is recommended in patients with DCM/HNDCM and hemodynamically tolerated sustained monomorphic ventricular tachycardia	IIa	C

Note: DCM — dilated cardiomyopathy; HNDCM — hypokinetic nondilated cardiomyopathy; ICD — implantable cardioverter defibrillator.

Примечание: ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; ИКД — имплантируемый кардиовертер-дефибриллятор.

functionality and optimization of ICD programing can reduce the number of ICD shocks delivered in response to ventricular tachycardia (VT); however, additional drug therapy is almost always required to reduce symptomatic episodes of VA [2]. The recommendations for antiarrhythmic drug selection are based on the results of the OPTIC trial, where 412 patients with ICD within 21 days after VT/ventricular fibrillation (VF) were randomized to three antiarrhythmic treatment groups: amiodarone plus beta-blocker, sotalol alone, or beta-blocker alone. The frequency of ICD shocks after 1 year was 10.3% in the amiodarone and beta-blocker group, 24.3% in the sotalol group, and 38.5% in the beta-blocker group [21]. Only limited data are available on the efficiency and safety of sodium channel blockers in reducing the number of ICD activations in DCM [2]. They may be useful for reducing VAs only in a few patients without severe heart failure and low LVEF. The recommendations emphasize the need to align the level of efficiency and drug-related side effects when choosing antiarrhythmic therapy.

In DCM, monomorphic VTs predominate (Fig. 2), which are based on the reentry mechanism.

To reduce the number of monomorphic VTs, catheter ablation can be used in addition to drug therapy. However, the rate of recurrent VTs after catheterization procedures in patients with DCM is higher than that in patients with IHD, the VT-free survival 1 year after a catheter procedure in DCM is 40.5%, whereas in IHD, it is 57% [22]. Some patients require repeated procedures, after which the probability of the absence of recurrence of arrhythmia increases. When the arrhythmic focus is subepicardial, epicardial ablation is required in

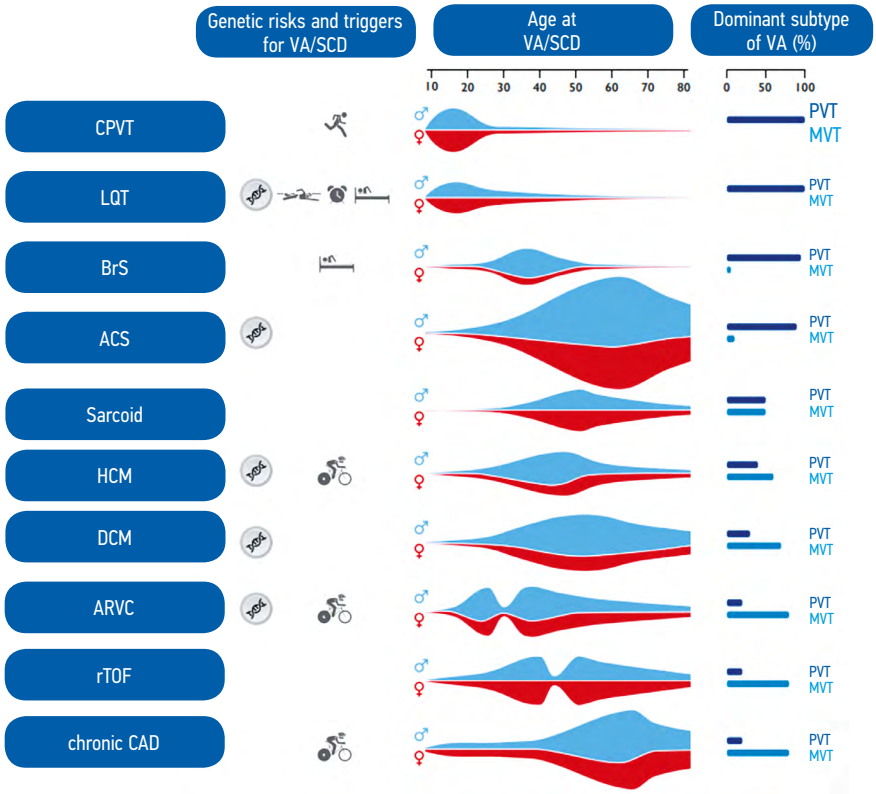

Fig. 2. Genetic risk for VA/SCD, typical triggers for VA/SCD, age at presentation with VA/SCD, sex predominance, and typical VA in different diseases associated with VA/SCD

— presence of genetic risks; — trigger — physical and emotional stress; — trigger — swimming, diving; — syncope during sleep; —trigger — a sharp sound; — trigger – physical stress

ACS — acute coronary syndrome; ARVC — arrhythmogenic right ventricular cardiomyopathy; BrS — Brugada syndrome; CAD — coronary artery disease; CPVT — catecholaminergic polymorphic ventricular tachycardia; DCM — dilated cardiomyopathy; HCM — hypertrophic cardiomyopathy; LQT — long QT syndrome; MVT — monomorphic ventricular tachycardia; PVT — polymorphic ventricular tachycardia; rTOF — repaired tetralogy of Fallot; SCD — sudden cardiac death; VA — ventricular arrhythmia

Рис. 2. Генетический риск желудочковой аритмии / внезапной сердечной смерти, типичные триггеры желудочковой аритмии / внезапной сердечной смерти, возраст при появлении желудочковой аритмии / внезапной сердечной смерти, преобладание пола и вариант типичной желудочковой аритмии при различных заболеваниях, связанных с желудочковой аритмией / внезапной сердечной смертью [2]

— наличие генетических рисков; — триггер — физическая и эмоциональная нагрузки; — триггер — плавание, ныряние; — синкопальные состояния во сне; — триггер — резкий звук; — триггер — физическая нагрузка

ВСС — внезапная сердечная смерть; ЖА — желудочковые аритмии; МЖТ — мономорфная желудочковая тахикардия; ПЖТ — полиморфная желудочковая тахикардия

Table 5. General recommendations for patients with mutations in genes encoding lamin
Таблица 5. Общие рекомендации для пациентов с мутацией генов, кодирующих синтез ламинов

Recommendation	Indication class	Evidence level
Patients with DCM/HNDCM and the *LMNA* mutation are not recommended to participate in high-intensity training, including competitive sports	III	C

Note: DCM — dilated cardiomyopathy; HNDCM — hypokinetic nondilated cardiomyopathy; *LMNA* — nuclear lamin gene.
Примечание: ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; *LMNA* — ген ядерных ламинов.

27%–30% of the procedures [22, 23]. The outcome is particularly poor in patients with pathogenic *LMNA* mutations. Because of the deep transmural, anteroseptal location of the substrate, transcoronary ethanol ablation, bipolar ablation, and surgical ablation may be required [24–26].

The general recommendations for patients with *LMNA* mutations are presented in Table 5.

Arrhythmogenic right ventricular cardiomyopathy (ACM)

ACM is characterized by the replacement of the myocardium with fibrous and fatty tissue [27]. Currently, it is more correct to talk about ACM of both ventricles [28]. The prevalence of ACM in the population ranges from 1:1000 to 1:5000 [29]. Most often, ACM is caused by pathogenic mutations in desmosomal genes (*Dsc2,* desmocollin-2; *Dsg2,* desmoglein-2; *Dsp,* desmoplakin; *Pkg,* plakoglobin; *Pkp2*, plakophilin-2) and less often in nondesmosomal genes. Similar to DCM, in ACM, some mutations are associated with a high risk of developing VA at a young age [30, 31]. The diagnosis of ACM is complex and requires searching and assessing certain criteria. Although the recommendations contain a reference in Guidelines 2022 to the publication of Corrado et al. (2020) [28], the criteria for diagnosing ACM are not the 2020 Padua criteria but the previous 2010 criteria [32]. As an advantage, the Padua criteria provide an algorithm for diagnosing not only the right ventricular but also the LV process. Figure 3 presents the ECG of our 16-year-old patient with arrhythmogenic cardiomyopathy. ECG was performed during sinus rhythm. At the beginning of the recording, the moment of the cessation of unstable polymorphic VT (three complexes) was recorded. In addition, frequent polymorphic single and paired premature ventricular complexes (PVC) of the bigeminy type were noted. The major Padua criterion for the diagnosis of right ventricular ACM was identified, namely, inverted *T* waves in right precordial

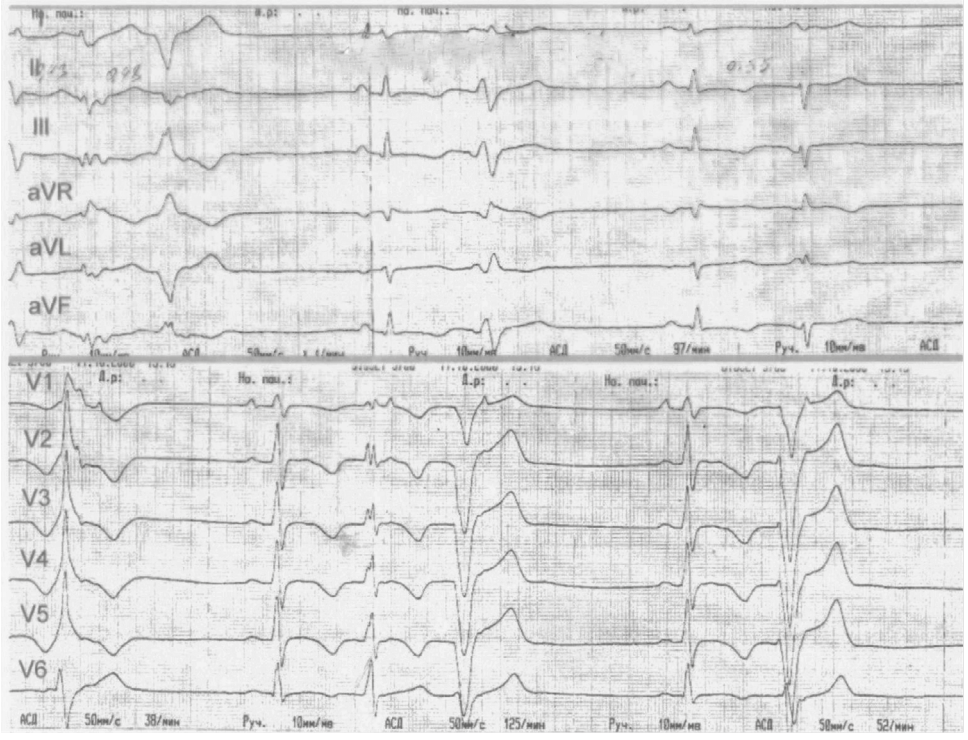

Fig. 3. Electrocardiogram of a patient suffering from biventricular arrhythmogenic cardiomyopathy (explanation in the text)
Рис. 3. Электрокардиограмма пациента, страдающего бивентрикулярной аритмогенной кардиомиопатией (объяснение в тексте)

leads V1, V2, and V3 in a patient with complete pubertal development, and the absence of complete right bundle branch block. In addition, minor Padua criteria were noted for the diagnosis of LV ACM, which are inverted *T* waves in left precordial leads *V4–V6* in the absence of complete left bundle branch (LBB) block and low-amplitude *QRS* (< 0.5 mV peak-to-peak) in the limb leads to the absence of obesity, emphysema, and pericardial effusion.

Figure 4 presents the ECG of one of the patients with right ventricular ACM during classical sustained VT, which is a major criteria for diagnosing right ventricular ACM — VT with QRS morphology similar to LBB block, causing the deviation of the electrical axis to the left upward (not from the right ventricle (RV) outflow tract, the focus is located in the area of the RV free wall).

During the natural disease course in patients with ACM who have not been implanted with ICD, cardiac arrest occurs in 4.6%–6.1% of cases [33–36]. Because of the high incidence of VA and SCD in ACM, risk stratification of adverse arrhythmic events is required. In 16%–19% of cases, the indication for ICD is rapid VT (⩾ 250 beats/min) or VF, which are considered surrogate markers of a potentially life-threatening event [37–40]. Recently, a risk model was developed to predict any persistent VA in ACM based on the analysis of the disease course of 528 patients with an established diagnosis of ACM and no history of VA. During model development, age, sex, arrhythmic syncope, nonsustained VT, number of ventricular PVC, number of leads with *T*-wave inversion, and RV EF (c-index 0.77) were collected [41]. Another model for predicting life-threatening VA was proposed based on the analysis of 864 patients with ACM, which included male sex, age, number of ventricular PVC within 24 h, and number of leads with *T*-wave inversion (c-index 0.74) as predictors [41]. However, validation studies are required before these risk models can be recommended for clinical use [2].

The recommendations for the diagnostics and management of patients with arrhythmogenic cardiomyopathy are presented in Table 6 [2].

The recommendations for the risk stratification and primary prevention of SCD are presented in Table 7 [2].

RV and LV dysfunctions are associated with high arrhythmic risk. The guidelines state that EF thresholds for indications for ICD implantation are difficult to define; however, even asymptomatic patients with severe RV dysfunction (right ventricular area fraction change ⩽ 17% or RV EF ⩽ 35%) should be advised to implant an ICD for the primary prevention of SCD. Similarly, patients with ACM with significant LV disease (LVEF ⩽ 35%) are candidates for ICD implantation according to current ICD guidelines for DCM [2]. An ICD for the primary prevention of SCD should also be recommended in patients with symptomatic ACM (presyncope or palpitations suggestive of VA) and moderate

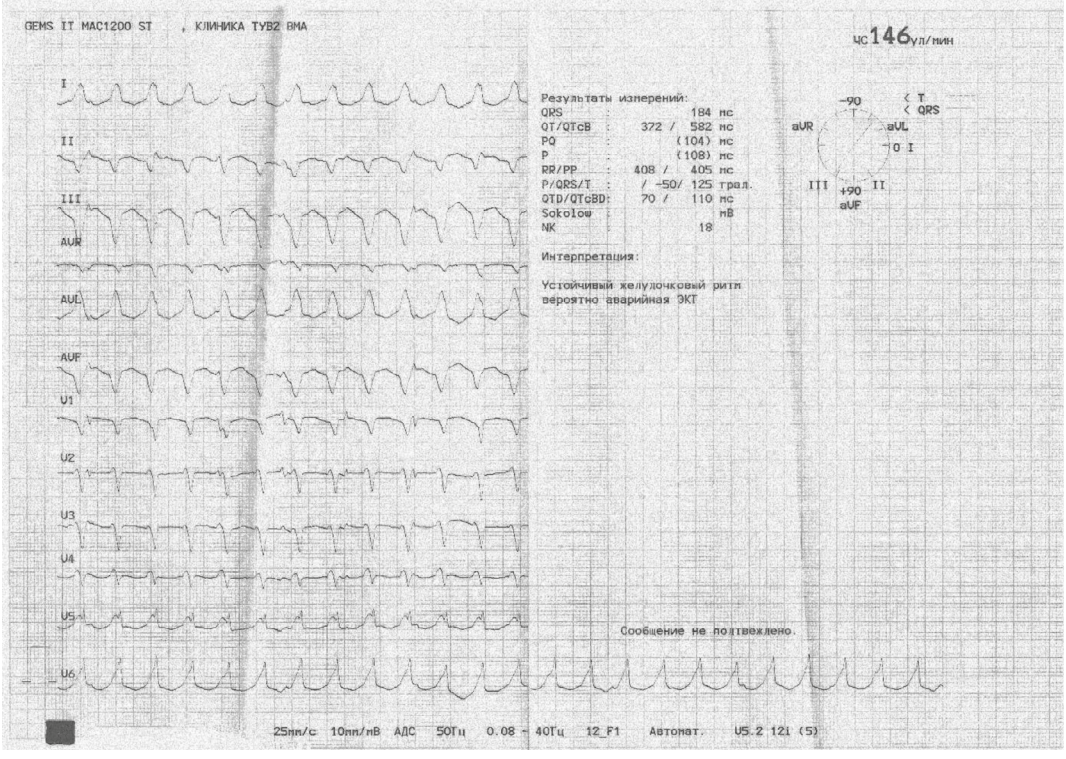

Fig. 4. Electrocardiogram of a patient suffering from right ventricular arrhythmogenic cardiomyopathy, at the time of tipical ventricular tachycardia (explanation in text)

Рис. 4. Электрокардиограмма пациента, страдающего правожелудочковой аритмогенной кардиомиопатией, во время типичной желудочковой тахикардии (объяснение в тексте)

Table 6. Recommendations for the diagnosis and management of patients suffering from arrhythmogenic cardiomyopathy
Таблица 6. Рекомендации по диагностике и ведению пациентов, страдающих аритмогенной кардиомиопатией

Recommendation	Indication class	Evidence level
Cardiac MRI is recommended for patients with suspected ACM	I	B
Genetic counseling and testing are recommended for patients with suspected or established ACM diagnosis	I	B
Patients diagnosed with ACM are advised to avoid high-intensity physical exercise	I	B
Avoidance of high-intensity physical exercise may be recommended for carriers of ACM-associated pathogenic mutations and those without the phenotype	IIb	C
Beta-blocker therapy may be recommended for all patients with an established diagnosis of ACM	IIb	C

Note: ACM — arrhythmogenic cardiomyopathy; MRI — magnetic resonance imaging.

Примечание: АКМП — аритмогенная кардиомиопатия; МРТ — магнитно-резонансная томография.

Table 7. Recommendations for risk stratification and primary prevention of sudden death in arrhythmogenic cardiomyopathy
Таблица 7. Рекомендации по стратификации риска и первичной профилактике внезапной смерт при аритмогенной кардиомиопатии

Recommendation	Indication class	Evidence level
ICD implantation is recommended for patients with a definite diagnosis of ACM and arrhythmogenic syncope	IIa	B
ICD implantation is recommended for patients with an established diagnosis of ACM and a significant decrease in RV systolic function or LV dysfunction	IIa	C
ICD implantation should be recommended in symptomatic patientsa with an established diagnosis of ACM, moderate RV or LV dysfunction or nonsustained ventricular tachycardia, or electrophysiologically induced sustained monomorphic ventricular tachycardia	IIa	C
In patients with ACM and symptoms suggestive of ventricular arrhythmia, electrophysiological testing may be recommended for risk stratification	IIb	C

Note: ACM — arrhythmogenic cardiomyopathy; ICD — implantable cardioverter defibrillator; LV — left ventricle; RV — right ventricle. [a] Presyncope or palpitations indicating ventricular arrhythmias.

Примечание: АКМП — аритмогенная кардиомиопатия; ИКД — имплантируемый кардиовертер-дефибриллятор; ЛЖ — левый желудочек; ПЖ — правый желудочек. [a] Предобморочные состояния или учащенное сердцебиение, указывающее на желудочковые аритмии.

Table 8. Recommendations for secondary prevention of sudden death and treatment of ventricular arrhythmias in arrhythmogenic cardiomyopathy
Таблица 8. Рекомендации по вторичной профилактике внезапной смерти и лечению желудочковых аритмий

Recommendation	Indication class	Evidence level
ICD implantation is recommended in patients with ACM and ventricular tachycardia or ventricular fibrillation accompanied by hemodynamic instability	I	C
In patients with ACM and nonsustained or sustained ventricular tachycardia, beta-blocker therapy is recommended	I	C
In patients with ACM and recurrent symptomatic sustained monomorphic ventricular tachycardia or ICD shocks for sustained monomorphic ventricular tachycardia despite beta-blocker therapy, catheter ablation should be recommended in specialized centers	IIa	C
Patients with ACM and indications for an ICD should be recommended to install a device with the ability to program anti-tachycardia pacing to relieve sustained monomorphic ventricular tachycardia	IIa	B
ICD implantation is recommended for patients with ACM with hemodynamically tolerated sustained monomorphic ventricular tachycardia	IIa	C
Patients with ACM and recurrent symptomatic ventricular tachycardia despite beta-blocker therapy should be treated with other antiarrhythmic agents	IIa	C

Note: ACM — arrhythmogenic cardiomyopathy; ICD — implantable cardioverter defibrillator.

Примечание: АКМП — аритмогенная кардиомиопатия; ИКД — имплантируемый кардиовертер-дефибриллятор.

Table 9. Recommendations for the management of relatives of a patient with arrhythmogenic cardiomyopathy
Таблица 9. Рекомендация по ведению родственников больного аритмогенной кардиомиопатией

Рекомендация	Класс показаний	Уровень доказательности
First-degree relatives of patients with ACM an electrocardiogram and echocardiogram are recommended	I	C

Note: ACM — arrhythmogenic cardiomyopathy.
Примечание: АКМП — аритмогенная кардиомиопатия.

RV dysfunction (EF of 40%–35%) and/or moderate LV dysfunction (EF of 45%–35%) [2].

The recommendations for the secondary prevention of sudden cardiac death and treatment of ventricular arrhythmias are presented in Table 8 [2].

Beta-blockers are recommended as first-line therapy in both symptomatic and asymptomatic cases, although this is not supported by clinical trial data [2]. Sotalol is notably effective in preventing the reproducibility of VT during electrophysiological tests [43]; however, it does not suppress clinically significant arrhythmias in real life [43, 44]. Treatment with amiodarone or class 1 drugs is associated with a trend toward lower recurrent VT rates compared with sotalol [45]. The addition of flecainide to beta-blockers or sotalol was useful in a small group of patients [46]. Note that first-class drugs are contraindicated in patients with reduced EF. Catheter ablation may be an alternative to drug therapy. When choosing a therapeutic strategy, potential risks, drug side effects, and patient preferences must be considered [47].

The recommendations for managing relatives of patients with ACM are presented in Table 9 [2].

Hypertrophic cardiomyopathy (HCM)

HCM is a disease characterized by an increase in LV wall thickness in the absence of pathological conditions associated with myocardial load, such as arterial hypertension or valvular diseases [1, 48].

Mutations in genes encoding the synthesis of sarcomeric proteins (myosin binding protein C [MYBPC3], myosin heavy chains [MYH7], cardiac troponin T [TNNT2], cardiac troponin I [TNNI3], and α-tropomyosin [TPM1], essential and regulatory light chains myosin [MYL3 and MYL2], and actin [ACTS]) are detected in 30%–60% of patients, most often in those with HCM diagnosed at a young age or with a family history of HCM [49, 50]. The recommendations emphasize the need for genetic testing of probands and screening of first-degree relatives.

In adults, the estimated prevalence of HCM is 1 per 500 populations [51]. Among children, the rate is much lower.

According to previous studies, annual mortality in HCM ranges from 0.5% to 2% [2, 48, 52]. Most HCM-associated deaths in patients aged up to 60 years occur suddenly, whereas older patients more often die from stroke, heart failure, obstruction, or supraventricular arrhythmias. The annual incidence of SCD or related ICD activation is approximately 0.8%; however, it largely depends on the age and risk profile of a patient [53–56]. SCD can also be triggered by exercise and participation in competitive sports [57].

Planning a management strategy for a patient with HCM begins with stratifying the risk of sudden cardiac death. A 5-year risk stratification scale for SCD has been developed based on seven factors, namely, age, LV wall thickness, left atrial (LA) dimension, LV outflow tract (LVOT) obstruction, nonsustained VT, unexplained syncope, and family history of SCD (HCM Risk-SCD: https://doc2do.com/hcm/webHCM.html) [2]. The calculator is not intended for evaluating professional athletes or persons with metabolic and infiltrative diseases, after myectomy or ethanol septal ablation. To stratify the risk of SCD in children (1–16 years), a special pediatric HCM risk-kids model was recently developed, including unexplained syncope, maximum LV wall thickness, LA diameter, LVOT gradient, and nonsustained VT (https://hcmriskkids.org) [2, 58].

The 2022 guidelines emphasize additional factors that are not reflected in the SCD risk model that should be considered in patients at intermediate or low estimated risk. Important additional factors include LV systolic dysfunction, aneurysm in the LV apex, extensive areas of late signal enhancement on contrast-enhanced cardiac MRI corresponding to fibrosis, and presence of single or multiple sarcomeric mutations [50, 59–64]. The identification of large areas of late MRI signal enhancement (≥15% of the LV mass) has been proposed as a good predictor of SCD. However, fibrosis thresholds are sometimes difficult to use because the quantification of late signal enhancement depends on the method by which the MR image was acquired and the type and amount of contrast used [2].

Because new risk factors may emerge in a patient's life, periodic risk reassessment is an essential part of long-term patient follow-up.

The advisability of the electrophysiological test to induce VA in HCM, according to the recommendations, is controversial because according to the literature, rhythm disturbances obtained with the electrophysiological test are considered nonspecific, although there are other points of view [65, 66].

The recommendations for risk stratification, prevention of sudden cardiac death, and treatment of ventricular arrhythmias in hypertrophic cardiomyopathy are presented in Table 10 [2].

Table 10. Recommendations for risk stratification, prevention of sudden cardiac death, and treatment of ventricular arrhythmias in hypertrophic cardiomyopathy

Таблица 10. Рекомендации по стратификации риска, профилактике внезапной сердечной смерти и лечению желудочковых аритмий при гипертрофической кардиомиопатии

Recommendation	Indication class	Evidence level
Diagnostic evaluation and general recommendations		
MRI with late gadolinium enhancement is recommended for diagnostic evaluation in a patient with HCM	I	B
Genetic counseling and testing are recommended for patients with HCM.	I	B
High-intensity exercise may be recommended in asymptomatic adult patients with HCM without risk markers for SCD	IIb	C
Risk stratification and primary prevention of SCD		
It is recommended to assess the 5-year risk of SCD at initial diagnosis and thereafter at intervals of 1–3 years or in case of a change in the patient's clinical status	I	C
ICD implantation should be recommended in patients aged 16 years or more with an estimated 5-year risk of SCD ⩾ 6%	IIa	B
ICD implantation should be recommended for HCM in patients aged 16 years or more with an intermediate 5-year risk of SCD (4%–6%) if they have the following: a) extensive late gadolinium enhancement on cardiac MRI (usually ⩾ 15% of the LV mass) or b) LVEF < 50% or c) abnormal blood pressure response during an exercise test or d) aneurysms of the LV apex or e) sarcomeric pathogenic mutation	IIa	B
ICD implantation may be recommended for HCM in patients aged 16 years or more with an estimated 5-year risk of SCD of 4%–6% without additional risk factors	IIb	B
ICD implantation may be recommended for HCM in patients aged 16 years or more with a low estimated 5-year risk of sudden cardiac death (< 4%) and a) significant late gadolinium enhancement on MRI (⩾ 15% of the LV mass); or b) LVEF < 50%; or c) LV apical aneurysm	IIb	B
In children younger than 16 years with HCM and an estimated 5-year risk of sudden death ⩾ 6% (based on the HCM rish-kids assessment), ICD implantation should be recommended	IIa	B
Secondary prevention of SCD and treatment of VA		
ICD implantation is recommended for patients with HCM with hemodynamically intolerable VT or VF	I	B
In patients with HCM and hemodynamically tolerated sustained VT, the implantation of an ICD should be recommended	IIa	C
In patients with HCM and recurrent, symptomatic VA, or recurrent ICD activations, treatment with antiarrhythmic drugs should be recommended	IIa	C
Catheter ablation in specialized centers may be recommended for some HCM patients with recurrent, symptomatic, sustained monomorphic VT or frequent ICD activation caused by sustained monomorphic VT when antiarrhythmic drugs are ineffective, intolerant, or contraindicated.	IIb	C
Management of relatives of a patient with HCM		
First-degree relatives of patients with HCM are recommended to undergo ECG and echocardiographic examination	I	C

Note: SCD — sudden cardiac death; HCM — hypertrophic cardiomyopathy; VA — ventricular arrhythmias; VT — ventricular tachycardia; ICD — implantable cardioverter defirillator; LV — left ventricle; MRI — magnetic resonance imaging; EF — ejection fraction; VF — ventricular fibrillation.

Примечание: ВСС — внезапная сердечная смерть; ГКМП — гипертрофическая кардиомиопатия; ЖА — желудочковые аритмии; ЖТ — желудочковая тахикардия; ИКД — имплантируемый кардиовертер-дефириллятор; ЛЖ — левый желудочек; МРТ — магнитно-резонансная томография; ФВ — фракция выброса; ФЖ — фибрилляция желудочков.

Patients who experience cardiac arrest due to VT/VF or hemodynamically intolerable VT are at high risk of life-threatening VA and require an ICD for secondary prevention of SCD [2, 48, 67–69]. Following ICD implantation for primary or secondary prevention of SCD, the most common documented subtype of VA is sustained monomorphic VT, in which ATP is successful in 69%–76.5% of all episodes. In this regard, during implantation, preference should be given to ICDs with ATP functionality [70–72]. No randomized clinical or cohort studies have confirmed the important role of medications in preventing SCD in HCM [2, 48, 73]. Amiodarone can reduce the number of VA; however, data on its effectiveness in preventing SCD are inconsistent [73, 74]. Disopyramide and beta-blockers are effective in controlling symptoms and LVOT obstruction; however, no evidence shows that they reduce the risk of SCD [2, 48]. Similarly, surgical myectomy or ethanol ablation is not recommended to reduce the SCD risk in patients with LVOT obstruction [2, 48]. Despite the lack of clear data on the efficiency of antiarrhythmic drugs, beta-blockers, amiodarone, sotalol, and sodium channel blockers are prescribed to patients with HCM and symptomatic VAs [2]. Catheter ablation may also be used in carefully selected patients with HCM and sustained monomorphic VT for whom antiarrhythmic drugs are ineffective, contraindicated, or intolerable. The recommendations emphasize that the results after ablation for HCM are worse than those for other diseases of nonischemic etiology [75–77].

Left ventricular non-compaction (LVNC)

Since 1995, along with HCM, DCM, RCM, and ACM, a group of "unclassified" cardiomyopathies has been identified, including LVNC or noncompact cardiomyopathy (NCM) and Takotsubo syndrome. In the 2023 European Society of Cardiology guidelines on cardiomyopathies, the concept of "unclassified" cardiomyopathies was abolished and replaced by "syndromes associated with cardiomyopathic phenotypes" [15]. Taking into account the change in the approach to the classification of cardiomyopathies, the 2023 recommendations propose using the term "left ventricle hypertrabecularity" instead of LVNC.

In this article, we use the term LVNC because this is the term used in the 2022 European guidelines for the treatment of ventricular arrhythmias and the prevention of SCD. LVNC includes a heterogeneous group of phenotypically different diseases characterized by specific changes in the LV myocardium and sometimes RV myocardium [2, 78–80]. A structural pathology common to all phenotypes is abnormal trabeculae in the LV/RV or both ventricles, most often in the apical region [78–80]. Figure 5 presents an echocardiogram of one of our patients.

LVNC may include LV dilatation, LV hypertrophy, systolic dysfunction, diastolic dysfunction, or both, and it may be associated with various congenital heart defects [78–80].

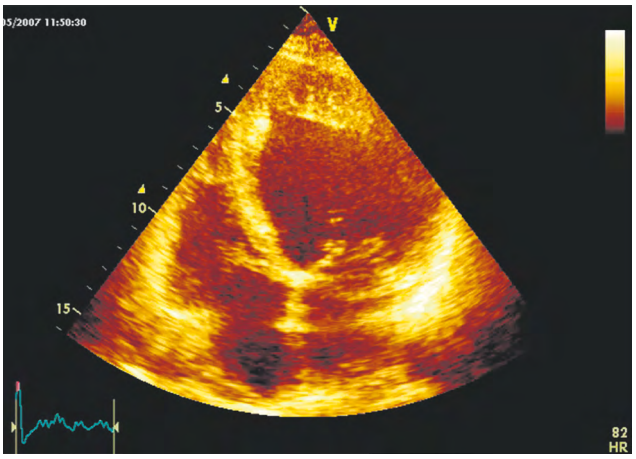

Fig. 5. Echocardiogram of a patient with left ventricular hypertrabeculation. Apical 4-chamber view. Noteworthy is the dilatation of the left ventricle and pronounced trabecularity in the area at the apex

Рис. 5. Эхокардиограмма пациента, имеющего гипертрабекулярность миокарда левого желудочка. Верхушечная 4-камерная позиция. Обращают на себя внимание дилатация левого желудочка и выраженная трабекулярность в области верхушки

Clinically, the disease manifests as CHF, life-threatening VAs, complete AV block, and bundle branch blocks. Up to nine variants of the disease, hemodynamically and clinically occurring differently [79, 80]:

1) Isolated or benign form: LVNC with structural changes, absent hemodynamic disturbances, and clinical signs (registered in athletes and pregnant women);

2) Arrhythmogenic form: the main clinical manifestation is cardiac arrhythmia, primarily VA;

3) Dilated form: in addition to characteristic morphological changes, there is dilation and decreased contractility of the LV;

4) Hypertrophic form, with no pronounced dilatation, but with myocardial hypertrophy, primarily thickening of the myocardium, which is not involved in the noncompaction process;

5) "Mixed" form of non-compaction;

6) Restrictive form, in which severe diastolic dysfunction predominates;

7) Biventricular form of the non-compaction;

8) Excessive trabecularity of the RV with a normal structure of the LV;

9) Combination of non-compaction with congenital heart defects.

The association of the disease with genetic abnormalities is detected in 30%–50% of patients. Several genes encoding sarcomeric or cytoskeletal proteins have been identified; however, the identified genetic abnormalities occur not only in NCM but also in other cardiomyopathies, including Barth syndrome [79, 80].

Diagnostics of non-compaction are difficult; various proposed diagnostic criteria have not yet been validated, and

Table 11. Recommendations for cardioverter defibrillator implantation in left ventricular non-compaction
Таблица 11. Рекомендации по имплантации кардиовертера-дефибриллятора при некомпактном миокарде левого желудочка

Recommendation	Indication class	Evidence level
In patients with noncompact cardiomyopathy diagnosed according to MRI or echocardiographic findings, ICD implantation should be recommended for primary prevention of SCD for the same indications as for DCM/HNDCM.	IIa	C

Note: SCD — sudden cardiac death; DCM — dilated cardiomyopathy; HNDCM — hypokinetic nondilated cardiomyopathy; ICD — implantable cardioverter defibrillator, MRI — magnetic resonance imaging.

Примечание: ВСС — внезапная сердечная смерть; ДКМП — дилатационная кардиомиопатия; ГНДКМП — гипокинетическая недилатационная кардиомиопатия; ИКД — имплантируемый кардиовертер-дефибриллятор: МРТ — магнитно-резонансная томография.

Table 12. Recommendations for implantation of a cardioverter-defibrillator in patients with cardiac amyloidosis
Таблица 12. Рекомендации по имплантации кардиовертера-дефибриллятора пациентам, страдающим амилоидозом сердца

Recommendation	Indication class	Evidence level
ICD should be recommended in patients with light-chain amyloidosis or transthyretin-associated amyloid CMP and hemodynamically intolerable VT	IIa	C

Note: VT — ventricular tachycardia; ICD — implantable cardioverter defibrillator; CMP — cardiomyopathy.

Примечание: ЖТ — желудочковая тахикардия; ИКД — имплантируемый кардиовертер-дефибриллятор; КМП — кардиомиопатия.

none of them are given as diagnostic criteria in the recommendations. Cardiovascular mortality in patients with NCM is similar to that in patients with DCM [81]. The detection of focal fibrosis on contrast-enhanced MRI is associated with serious cardiovascular events, including arrhythmic events [82]. However, the risk stratification criteria for SCD in NCM have not yet been developed. Thus, in the future, combining MRI criteria with genetic testing data may overcome the current uncertainty regarding risk stratification for adverse events in NCM [2, 83]. In the meantime, when deciding on ICD implantation for diagnosed NCM, the criteria recommended for DCM/HNDCM should be used. The recommendations for ICD implantation for LVNC are presented in Table 11 [2].

Restrictive cardiomyopathy (RCM)

The RCM phenotype is rare and can result from various causes, including genetically determined and acquired interstitial fibrosis, infiltrative disorders (e.g., amyloidosis), and storage diseases (e.g., Anderson–Fabry disease). Their identification is crucial for the choice of therapy because of certain diseases in which in the clinical course restrictive hemodynamics comes to the fore, and there is a specific disease-modifying therapy. Heart failure is a major symptom of any type of RCM. Depending on the phenotype, atrial or ventricular arrhythmias may predominate, including fatal ones. For example, in Fabry disease, the most reported cardiovascular deaths were classified as SCD [84]. Because RCM is the rarest among all CMPs, large randomized studies of this pathology are not yet available. The retrospective, observational nature of most minor, single-center studies and the low absolute number of deaths due to cardiovascular disease and SCD in these studies do not currently allow ICDs to be recommended for the primary prevention of SCD in RCM. The guidelines provide advice only ICD for cardiac amyloidosis (Table 12) [2]. Currently, an ICD should be recommended for patients with amyloid CMP and hemodynamically intolerable VT after careful consideration of the competing risks of nonarrhythmic and noncardiac death.

Recommendations devoted to the urgent issues of determining CMP, diagnostics, risk stratification of SCD, and treatment of VA in CMPs are of great practical interest because fatal ventricular arrhythmias are a common complication of all CMPs.

ADDITIONAL INFORMATION

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REFERENSES

1. Elliott P, Andersson B, Arbustini E, et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. *European Heart Journal.* 2008;29:270–276. DOI: https://doi.org/10.1093/eurheartj/ehm342

2. Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *European Heart Journal.* 2022;00:1–130. DOI: https://doi.org/10.1093/eurheartj/ehac262

3. Køber L, Thune JJ, Nielsen JC, et al. Defibrillator implantation in patients with nonischemic systolic heart failure. *The New England Journal of Medicine.* 2016;375:1221–1230. DOI: https://doi.org/10.1056/NEJMoa1608029

4. Weintraub RG, Semsarian C, Macdonald P. Dilated cardiomyopathy. *Lancet.* 2017;390:400–414. DOI: https://doi.org/10.1016/S0140-6736(16)31713-5

5. Beggs SAS., Jhund PS, Jackson CE, et al. Non-ischaemic cardiomyopathy, sudden death and implantable defibrillators: a review and meta-analysis. *Heart.* 2018;104:144–150. DOI: https://doi.org/10.1136/heartjnl-2016-310850

6. Codd MB, Sugrue DD, Gersh BJ, Melton LJ. Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy. A population-based study in Olmsted County, Minnesota, 1975–1984. *Circulation.* 1989;80:564–572. DOI: https://doi.org/10.1161/01.cir.80.3.564

7. Manolio TA, Baughman KL, Rodeheffer R, et al. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute workshop. *Americal Journal of Cardiology.* 1992;69:1458–1466. DOI: https://doi.org/10.1016/0002-9149(92)90901-a

8. Pinto YM, Elliott PM, Arbustini E, et al. Proposal for a revised definition of dilated cardiomyopathy, hypokinetic non-dilated cardiomyopathy, and its implications for clinical practice: a position statement of the ESC working group on myocardial and pericardial diseases. *European Heart Journal.* 2016;37(23):1850–1858. DOI: https://doi.org/10.1093/eurheartj/ehv727

9. Zhang X-L, Xie J, Lan R-F, et al. Genetic Basis and Genotype–Phenotype Correlations in Han Chinese Patients with Idiopathic Dilated Cardiomyopathy. *Scientific Reports.* 2020;10:2226. DOI: https://doi.org/10.1038/s41598-020-58984-7.

10. Ader F, De Groote P, Réant P, et al. FLNC pathogenic variants in patients with cardiomyopathies: prevalence and genotype-phenotype correlations. *Clinical Genetics.* 2019;96:317–329. DOI: https://doi.org/10.1111/cge.13594

11. Kayvanpour E, Sedaghat-Hamedani F, Amr A, et al. Genotype-phenotype associations in dilated cardiomyopathy: meta-analysis on more than 8000 individuals. *Clinical Research in Cardiology.* 2017;106:127–139. DOI: https://doi.org/10.1007/s00392-016-1033-6

12. Ortiz-Genga MF, Cuenca S, Dal Ferro M, et al. Truncating FLNC mutations are associated with high-risk dilated and arrhythmogenic cardiomyopathies. *Journal of the American College of Cardiology.* 2016;68:2440–2451. DOI: https://doi.org/10.1016/j.jacc.2016.09.927

13. van den Hoogenhof MMG, Beqqali A, Amin AS, et al. RBM20 mutations induce an arrhythmogenic dilated cardiomyopathy related to disturbed calcium handling. *Circulation.* 2018;138:1330–1342. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.031947

14. Gigli M, Merlo M, Graw SL, et al. Genetic risk of arrhythmic phenotypes in patients with dilated cardiomyopathy. *Journal of the American College of Cardiology.* 2019;74:1480–1490. DOI: https://doi.org/10.1016/j.jacc.2019.06.072

15. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. *European Heart Journal.* 2023;44(37):1–124. DOI: https://doi.org/10.1093/eurheartj/ehad194

16. Iles LM, Ellims AH, Llewellyn H, et al. Histological validation of cardiac magnetic resonance analysis of regional and diffuse interstitial myocardial fibrosis. *European Heart Journal–Cardiovascular Imaging.* 2015;16:14–22. DOI: https://doi.org/10.1093/ehjci/jeu182

17. Olivas-Chacon CI, Mullins C, Stewart K, et al. Magnetic Resonance Imaging of Non-ischemic Cardiomyopathies: A Pictorial Essay. *Pictorial Essay.* 2015;5:37. DOI: https://doi.org/10.4103/2156-7514.159564

18. Di Marco A, Anguera I, Schmitt M, et al. Late gadolinium enhancement and the risk for ventricular arrhythmias or sudden death in dilated cardiomyopathy: systematic review and meta-analysis. *JACC: Heart Fail.* 2017;5:28–38. DOI: https://doi.org/10.1016/j.jchf.2016.09.017

19. Klem I, Klein M, Khan M, et al. Relationship of LVEF and myocardial scar to long-term mortality risk and mode of death in patients with nonischemic cardiomyopathy. *Circulation.* 2021:143:1343–1358. DOI: https://doi.org/10.1161/CIRCULATIONAHA.120.048477

20. Goidescu CM. Dilated cardiomyopathy produced by lamin A/C gene mutations. *Cluju Med.* 2013;86(4):309–312.

21. Connolly SJ, Dorian P, Roberts RS, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. *JAMA.* 2006;295:165–171. DOI: https://doi.org/10.1001/jama.295.2.165

22. Dinov B, Fiedler L, Schönbauer R, et al. Outcomes in catheter ablation of ventricular tachycardia in dilated nonischemic cardiomyopathy compared with ischemic cardiomyopathy: results from the Prospective Heart Centre of Leipzig VT (HELP-VT) Study. *Circulation.* 2014;129:728–736. DOI: https://doi.org/10.1161/CIRCULATIONAHA.113.003063

23. Muser D, Santangeli P, Castro SA, et al. Long-term outcome after catheter ablation of ventricular tachycardia in patients with nonischemic dilated cardiomyopathy. *Circulation Arrhythmia and Electrophysiology.* 2016;9:004328. DOI: https://doi.org/10.1161/CIRCEP.116.004328

24. Ebert M, Richter S, Dinov B, et al. Evaluation and management of ventricular tachycardia in patients with dilated cardiomyopathy. *Heart Rhythm.* 2019;16:624–631. DOI: https://doi.org/10.1016/j.hrthm.2018.10.028

25. Kumar S, Androulakis AFA, Sellal J-M, et al. Multicenter experience with catheter ablation for ventricular tachycardia in lamin A/C cardiomyopathy. *Circulation Arrhythmia and Electrophysiology.* 2016;9:004357. DOI: https://doi.org/ 10.1161/CIRCEP.116.004357

26. Oloriz T, Silberbauer J, Maccabelli G, et al. Catheter ablation of ventricular arrhythmia in nonischemic cardiomyopathy: anteroseptal versus inferolateral scar sub-types. *Circulation Arrhythmia and Electrophysiology.* 2014;7:414–423. DOI: https://doi.org/10.1161/CIRCEP.114.001568

27. Corrado D, Link MS, Calkins H. Arrhythmogenic right ventricular cardiomyopathy. *The New England Journal of Medicine*. 2017;376:1489–1490. DOI: https://doi.org/10.1056/NEJMra1509267

28. Corrado D, Marra MP, Zorzi A, et al. Diagnosis of arrhythmogenic cardiomyopathy: The Padua criteria. *International Journal of Cardiology*. 2020;319:106–114. DOI: https://doi.org/10.1016/j.ijcard.2020.06.005.

29. Mazzanti A, Ng K, Faragli A, et al. Arrhythmogenic right ventricular cardiomyopathy: clinical course and predictors of arrhythmic risk. *Journal of the American College of Cardiology*. 2016;68:2540–2550. DOI: https://doi.org/10.1016/j.jacc.2016.09.951

30. Bhonsale A, Groeneweg JA, James CA, et al. Impact of genotype on clinical course in arrhythmogenic right ventricular dysplasia/cardiomyopathy-associated mutation carriers. *European Heart Journal*. 2015;36:847–855. DOI: https://doi.org/10.1093/eurheartj/ehu509

31. Rigato I, Bauce B, Rampazzo A, et al. Compound and digenic heterozygosity predicts life time arrhythmic outcome and sudden cardiac death in desmosomal gene-related arrhythmogenic right ventricular cardiomyopathy. *Circulation Cardiovascular Genetics*. 2013;6:533–542. DOI: https://doi.org/10.1161/CIRCGENETICS.113.000288

32. Novikova TN, Novikov VI, Sayganov SA, Shcherbakova VA. 2022 Esc guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: what is new? *Cardiac Arrhythmias*. 2022;2(3):7–30. DOI.org/10.17816/cardar110961

33. Hulot J-S, Jouven X, Empana J-P, et al. Natural history and risk stratification of arrhythmogenic right ventricular dysplasia/cardiomyopathy. *Circulation*. 2004;110:1879–1884. DOI: https://doi.org/10.1161/01.CIR.0000143375.93288.82

34. Wang W, Cadrin-Tourigny J, Bhonsale A, et al. Arrhythmic outcome of arrhythmogenic right ventricular cardiomyopathy patients without implantable defibrillators. *Journal of Cardiovascular Electrophysiology*. 2018;29:1396–1402. DOI: https://doi.org/10.1111/jce.13668

35. Pinamonti ., Dragos AM, Pyxaras SA, et al. Prognostic predictors in arrhythmogenic right ventricular cardiomyopathy: results froma 10-year registry. *European Heart Journal*. 2011;32:1105–1113. DOI: https://doi.org/10.1093/eurheartj/ehr040

36. Brun F, Groeneweg JA, Gear K, et al. Risk stratification in arrhythmic right ventricular cardiomyopathy without implantable cardioverter-defibrillators. *JACC Clinical Electrophysiology*. 2016;2:558–564. DOI: https://doi.org/10.1016/j.jacep.2016.03.015

37. Bhonsale A, James CA, Tichnell C, et al. Incidence and predictors of implantable cardioverter-defibrillator therapy in patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy undergoing implantable cardioverter-defibrillator implantation for primary prevention. *Journal of the American College of Cardiology*. 2011;58:1485–1496. DOI: https://doi.org/10.1016/j.jacc.2011.06.043

38. Corrado D, Calkins H, Link MS, et al. Prophylactic implantable defibrillator in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia and no prior ventricular fibrillation or sustained ventricular tachycardia. *Circulation*. 2010;122:1144–1152. DOI: https://doi.org/10.1161/CIRCULATIONAHA.109.913871

39. Platonov PG, Haugaa KH, Bundgaard H, et al. Primary prevention of sudden cardiac death with implantable cardioverter-defibrillator therapy in patients with arrhythmogenic right ventricular cardiomyopathy. *American Journal of Cardiology*. 2019;123:1156–1162. DOI: https://doi.org/10.1016/j.amjcard.2018.12.049

40. Link MS, Laidlaw D, Polonsky B, et al. Ventricular arrhythmias in the North American multidisciplinary study of ARVC: predictors, characteristics, and treatment. *Journal of the American College of Cardiology*. 2014;64:119–125. DOI: https://doi.org/10.1016/j.jacc.2014.04.035

41. Cadrin-Tourigny J, Bosman LP, Nozza A, et al. A new prediction model for ventricular arrhythmias in arrhythmogenic right ventricular cardiomyopathy. *European Heart Journal*. 2019;40:1850–1858. DOI: https://doi.org/10.1093/eurheartj/ehz103

42. Cadrin-Tourigny J, Bosman LP, Wang W, et al. Sudden cardiac death prediction in arrhythmogenic right ventricular cardiomyopathy: a multinational collaboration. *Circulation Arrhythmia and Electrophysiology*. 2021;14:008509. DOI: https://doi.org/10.1161/CIRCEP.120.008509

43. Wichter T, Borggrefe M, Haverkamp W, et al. Efficacy of antiarrhythmic drugs in patients with arrhythmogenic right ventricular disease. Results in patients with inducible and noninducible ventricular tachycardia. *Circulation*. 1992;86:29–37. DOI: https://doi.org/10.1161/01.cir.86.1.29

44. Marcus GM, Glidden DV, Polonsky B, et al. Efficacy of antiarrhythmic drugs in arrhythmogenic right ventricular cardiomyopathy: a report from the North American ARVC Registry. *Journal of the American College of Cardiology*. 2009;54:609–615. DOI: https://doi.org/10.1016/j.jacc.2009.04.052

45. Mahida S, Venlet J, Saguner AM, et al. Ablation compared with drug therapy for recurrent ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy: results from a multicenter study. *Heart Rhythm*. 2019;16:536–543. DOI: https://doi.org/10.1016/j.hrthm.2018.10.016

46. Ermakov S, Gerstenfeld EP, Svetlichnaya Y, Scheinman MM. Use of flecainide in combination antiarrhythmic therapy in patients with arrhythmogenic right ventricular cardiomyopathy. *Heart Rhythm*. 2017;14:564–569. DOI: https://doi.org/10.1016/j.hrthm.2016.12.010

47. Santangeli P, Zado ES, Supple GE, et al. Long-term outcome with catheter ablation of ventricular tachycardia in patients with arrhythmogenic right ventricular cardiomyopathy. *Circulation Arrhythmia and Electrophysiology*. 2015;8:1413–1421. DOI: https://doi.org/10.1161/CIRCEP.115.003562

48. Ommen SR, Mital S, Burke MA, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology/ American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2020;142:533–557. DOI: https://doi.org/10.1161/CIR.0000000000000938

49. Heliö T, Elliott P, Koskenvuo JW, et al. ESC EORP cardiomyopathy registry: real-life practice of genetic counselling and testing in adult cardiomyopathy patients. *ESC Heart Failure*. 2020;7:3013–3021. DOI: https://doi.org/10.1002/ehf2.12925

50. Ho CY, Day SM, Ashley EA, et al. Genotype and life time burden of disease in hypertrophic cardiomyopathy: insights from the Sarcomeric Human Cardiomyopathy Registry (SHaRe). *Circulation*. 2018;138:1387–1398. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.033200

51. Semsarian C, Ingles J, Maron MS, Maron BJ. New perspectives on the prevalence of hypertrophic cardiomyopathy. *Journal of the American College of Cardiology*. 2015;65:1249–1254. DOI: https://doi.org/10.1016/j.jacc.2015.01.019

52. Maron BJ, Rowin EJ, Casey SA, et al. Hypertrophic cardiomyopathy in adulthood as sociated with low cardiovascular

mortality with contemporary management strategies. *Journal of the American College of Cardiology*. 2015;65:1915–1928. DOI: https://doi.org/10.1016/j.jacc.2015.02.061

53. O'Mahony C, Jichi F, Pavlou M, et al. A novel clinical risk prediction model for sudden cardiac death in hypertrophic cardiomyopathy (HCMrisk-SCD). *European Heart Journal*. 2014;35:2010–2020. DOI: https://doi.org/10.1093/eurheartj/eht439

54. O'Mahony C, Jichi F, Ommen SR, et al. International External Validation Study of the 2014 European Society of Cardiology Guidelines on Sudden Cardiac Death Prevention in Hypertrophic Cardiomyopathy (EVIDENCE-HCM). *Circulation*. 2018;137:1015–1023. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.030437

55. Vriesendorp PA, Schinkel AFL, Liebregts M, et al. Validation of the 2014 European Society of Cardiology guidelines risk prediction model for the primary revention of sudden cardiac death in hypertrophic cardiomyopathy. *Circulation Arrhythmia and Electrophysiology*. 2015;8:829–835. DOI: https://doi.org/10.1161/CIRCEP.114.002553

56. Lorenzini M, Anastasiou Z, O'Mahony C, et al. Mortality among referral patients with hypertrophic cardiomyopathy vs the general European population. *JAMA Cardiology*. 2020;5:73–80. DOI: https://doi.org/10.1001/jamacardio.2019.4534

57. Gimeno JR, Tomé-Esteban M, Lofiego C, et al. Exercise-induced ventricular arrhythmias and risk of sudden cardiac death in patients with hypertrophic cardiomyopathy. *European Heart Journal*. 200;30:2599–2605. DOI: https://doi.org/10.1093/eurheartj/ehp327

58. Norrish G, Qu C, Field E, et al. External validation of the HCM Risk-Kids model for predicting sudden cardiac death in childhood hypertrophic cardiomyopathy. *European Journal of Preventive Cardiology*. 2022;29:678–686. DOI: https://doi.org/10.1093/eurjpc/zwab181

59. Chan RH, Maron BJ, Olivotto I, et al. Prognostic value of quantitative contrast-enhanced cardiovascular magnetic resonance for the evaluation of sudden death risk in patients with hypertrophic cardiomyopathy. *Circulation*. 2014;130:484–495. DOI: https://doi.org/10.1161/CIRCULATIONAHA.113.007094

60. He D, Ye M, Zhang L, Jiang B. Prognostic significance of late gadolinium enhancement on cardiac magnetic resonance in patients with hypertrophic cardiomyopathy. *Heart Lung*. 2018;47:122–126. DOI: https://doi.org/10.1016/j.hrtlng.2017.10.008

61. Rowin EJ, Maron BJ, Carrick RT, et al. Outcomes in patients with hypertrophic cardiomyopathy and left ventricular systolic dysfunction. *Journal of the American College of Cardiology*. 2020;75:3033–3043. DOI: https://doi.org/10.1016/j.jacc.2020.04.045

62. Rowin EJ, Maron BJ, Haas TS, et al. Hypertrophic cardiomyopathy with left ventricular apical aneurysm: implications for risk stratification and management. *Journal of the American College of Cardiology*. 2017;69:761–773. DOI: https://doi.org/10.1016/j.jacc.2016.11.063

63. Sadoul N, Prasad K, Elliott PM, et al. Prospective prognostic assessment of blood pressure response during exercise in patients with hypertrophic cardiomyopathy. *Circulation*. 1997;96:2987–2991. DOI: https://doi.org/10.1161/01.cir.96.9.2987

64. Elliott PM, Poloniecki J, Dickie S, et al. Sudden death in hypertrophic cardiomyopathy: identification of high risk patients. *Journal of the American College of Cardiology*. 2000;36:2212–2218. DOI: https://doi.org/10.1016/s0735-1097(00)01003-2

65. Behr ER, Elliott P, McKenna WJ. Role of invasive EP testing in the evaluation and management of hypertrophic cardiomyopathy. *Cardiac Electrophysiology Review*. 2002;6:482–486. DOI: https://doi.org/10.1023/a:1021161114347

66. Gatzoulis KA, Georgopoulos S, Antoniou C-K, et al. Programmed ventricular stimulation predicts arrhythmic events and survival in hypertrophic cardiomyopathy. *International Journal of Cardiology*. 2018;254:175–181. DOI: https://doi.org/10.1016/j.ijcard.2017.10.033

67. Elliott PM, Sharma S, Varnava A, et al. Survival after cardiac arrest or sustained ventricular tachycardia in patients with hypertrophic cardiomyopathy. *Journal of the American College of Cardiology*. 1999;33:1596–1601. DOI: https://doi.org/10.1016/s0735-1097(99)00056-x

68. Cecchi F, Maron BJ, Epstein SE. Long-term outcome of patients with hypertrophic cardiomyopathy successfully resuscitated after cardiac arrest. *Journal of the American College of Cardiology*. 1989; 13:1283–1288. DOI: https://doi.org/10.1016/0735-1097(89)90302-1

69. Maron BJ, Spirito P, Shen W-K, et al. Implantable cardioverter-defibrillators and prevention of sudden cardiac death in hypertrophic cardiomyopathy. *JAMA*. 2007;298:405–412. DOI: https://doi.org/10.1001/jama.298.4.405

70. Link MS, Bockstall K, Weinstock J, et al. Ventricular tachyarrhythmias in patients with hypertrophic cardiomyopathy and defibrillators: triggers, treatment, and implications. *Journal of Cardiovascular Electrophysiology*. 2017;28:531–537. DOI: https://doi.org/10.1111/jce.13194

71. Dallaglio PD, DiMarco A, MorenoWeidmann Z, et al. Antitachycardia pacing for shock prevention in patients with hypertrophic cardiomyopathy and ventricular tachycardia. *Heart Rhythm*. 2020;17:1084–1091. DOI: https://doi.org/10.1016/j.hrthm.2020.02.024

72. Adduci C, Semprini L, Palano F, et al. Safety and efficacy of antitachycardia pacing in patients with hypertrophic cardiomyopathy implanted with an ICD. *Pacing and Clinical Electrophysiology*. 2019;42:610–616. DOI: https://doi.org/10.1111/pace.13665

73. Melacini P, Maron BJ, Bobbo F, et al. Evidence that pharmacological strategies lack efficacy for the prevention of sudden death in hypertrophic cardiomyopathy. *Heart*. 2007;93:708–710. DOI: https://doi.org/10.1136/hrt.2006.099416

74. McKenna WJ, Oakley CM, Krikler DM, Goodwin JF. Improved survival with amiodarone in patients with hypertrophic cardiomyopathy and ventricular tachycardia. *British Heart Journal*. 1985;53:412–416. DOI: https://doi.org/10.1136/hrt.53.4.412

75. Vaseghi M, Hu TY, Tung R, et al. Outcomes of catheter ablation of ventricular tachycardia based on etiology in nonischemic heart disease: an international ventricular tachycardia ablation center collaborative study. *JACC Clinical Electrophysiology*. 2018;4: 1141–1150. DOI: https://doi.org/10.1016/j.jacep.2018.05.007

76. Igarashi M, Nogami A, Kurosaki K, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients with hypertrophic cardiomyopathy and apical aneurysm. *JACC Clinical Electrophysiology*. 2018;4:339–350. DOI: https://doi.org/10.1016/j.jacep.2017.12.020

77. Dukkipati SR, d'Avila A, Soejima K, et al. Long-term outcomes of combined epicardial and endocardial ablation of monomorphic ventricular tachycardia related to hypertrophic cardiomyopathy. *Circulation Arrhythmia and Electrophysiology*. 2011;4:185–194. DOI: https://doi.org/10.1161/CIRCEP.110.957290

78. Novikov VI, Novikova TN. *Cardiomyopathy*. Moscow: MEDpress-inform 2021;112 (In Russ.)

79. Towbin JA, Lorts A, Jefferies JL. Left ventricular non-compaction cardiomyopathy. *Lancet*. 2015;386(9995):813-25. DOI: https://doi.org/10.1016/S0140-6736(14)61282-4.

80. Gerecke BJ, Engberding R. Noncompaction Cardiomyopathy – History and Current Knowledge for Clinical Practice. *Journal of Clinical Medicine*. 2021;10(11):2457. DOI: https://doi.org/10.3390/jcm10112457.

81. Aung N, Doimo S, Ricci F, et al. Prognostic significance of left ventricular noncompaction: systematic review and meta-analysis of observational studies. *Circulation Cardiovascular Imaging*. 2020;13:009712. DOI: https://doi.org/10.1161/CIRCIMAGING.119.009712

82. Grigoratos C, Barison A, Ivanov A, et al. Meta-analysis of the prognostic role of late gadolinium enhancement and global systolic impairment in left ventricular noncompaction. *JACC Cardiovascular Imaging*. 2019;12:2141–2151. DOI: https://doi.org/10.1016/j.jcmg.2018.12.029

83. Richard P, Ader F, Roux M, et al. Targeted panel sequencing in adult patients with left ventricular non-compaction reveals a large genetic heterogeneity. *Clinical Genetics*. 2019;95:356–367. DOI: https://doi.org/10.1111/cge.13484

84. Baig S, Edward NC, Kotecha D, et al. Ventricular arrhythmia and sudden cardiac death in Fabry disease: a systematic review of risk factors in clinical practice. *Europace*. 2018;20:153–161. DOI: https://doi.org/10.1093/europace/eux261

СПИСОК ЛИТЕРАТУРЫ

1. Elliott P., Andersson B., Arbustini E., et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases // Eur Heart J. 2008. Vol. 29. P. 270–276. DOI: https://doi.org/10.1093/eurheartj/ehm342

2. Zeppenfeld K., Tfelt-Hansen J., de Riva M., et al. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death // European Heart J. 2022. Vol. 43, No. 40. P. 3997–4126. DOI: https://doi. org/10.1093/eurheartj/ehac262

3. Køber L., Thune J.J., Nielsen J.C., et al. Defibrillator implantation in patients with nonischemic systolic heart failure // N Engl J. Med. 2016. Vol. 375. P. 1221–1230. DOI: https://doi.org/10.1056/NEJMoa1608029

4. Weintraub R.G., Semsarian C., Macdonald P. Dilated cardiomyopathy // Lancet. 2017. Vol. 390. P. 400–414. DOI: https://doi.org/10.1016/S0140-6736(16)31713-5

5. Beggs S.A.S., Jhund P.S., Jackson C.E., et al. Non-ischaemic cardiomyopathy, sudden death and implantable defibrillators: a review and meta-analysis // Heart. 2018. Vol. 104. P. 144–150. DOI: https://doi.org/10.1136/heartjnl-2016-310850

6. Codd M.B., Sugrue D.D., Gersh B.J., Melton L.J. Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy. A population-based study in Olmsted County, Minnesota, 1975–1984 // Circulation. 1989. Vol. 80. P. 564–572. DOI: https://doi.org/10.1161/01.cir.80.3.564

7. Manolio T.A., Baughman K.L., Rodeheffer R., et al. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute workshop // Am J Cardiol. 1992. Vol. 69. P. 1458–1466. DOI: https://doi.org/10.1016/0002-9149(92)90901-a

8. Pinto Y.M., Elliott P.M., Arbustini E., et al. Proposal for a revised definition of dilated cardiomyopathy, hypokinetic non-dilated cardiomyopathy, and its implications for clinical practice: a position statement of the ESC working group on myocardial and pericardial diseases // Eur Heart J. 2016. Vol. 37(23). P. 1850–1858. DOI: https://doi.org/10.1093/eurheartj/ehv727.

9. Zhang X.-L., Xie J., Lan R.-F., et al. Genetic Basis and Genotype–Phenotype Correlations in Han Chinese Patients with Idiopathic Dilated Cardiomyopathy // Scientific Reports. 2020. Vol. 10. P. 2226. DOI: https://doi.org/10.1038/s41598-020-58984-7.

10. Ader F., De Groote P., Réant P., et al. FLNC pathogenic variants in patients with cardiomyopathies: prevalence and genotype-phenotype correlations // Clin Genet. 2019. Vol. 96. P. 317–329. DOI: https://doi/10.1111/cge.13594

11. Kayvanpour E., Sedaghat-Hamedani F., Amr A., et al. Genotype-phenotype associations in dilated cardiomyopathy: meta-analysis on more than 8000 individuals // Clin Res Cardiol. 2017. Vol. 106. P. 127–139. DOI: https://doi.org/10.1007/s00392-016-1033-6

12. Ortiz-Genga M.F., Cuenca S., Dal Ferro M., et al. Truncating FLNC mutations are associated with high-risk dilated and arrhythmogenic cardiomyopathies // J Am Coll Cardiol. 2016. Vol. 68. P. 2440–2451. DOI: https://doi.org/10.1016/j.jacc.2016.09.927

13. Earrhythmogenic dilated cardiomyopathy related to disturbed calcium handling // Circulation. 2018. Vol. 138. P. 1330–1342. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.031947

14. Gigli M., Merlo M., Graw S.L., et al. Genetic risk of arrhythmic phenotypes in patients with dilated cardiomyopathy // J Am Coll Cardiol. 2019. Vol. 74. P. 1480–1490. DOI: https://doi.org/10.1016/j.jacc.2019.06.072

15. Arbelo E., Protonotarios A., Gimeno J.R., et al. 2023 ESC Guidelines for the management of cardiomyopathies // European Heart Journal. 2023. Vol. 44, No. 37. P. 1–124. DOI: https://doi.org/10.1093/eurheartj/ehad194

16. Iles L.M., Ellims A.H., Llewellyn H., et al. Histological validation of cardiac magnetic resonance analysis of regional

and diffuse interstitial myocardial fibrosis // European Heart Journal–Cardiovascular Imaging. 2015. Vol. 16. P. 14–22. DOI: https://doi.org/10.1093/ehjci/jeu182

17. Olivas-Chacon C.I., Mullins C., Stewart K., et al. Magnetic Resonance Imaging of Non-ischemic Cardiomyopathies: A Pictorial Essay // Pictorial Essay. 2015. Vol. 5. P. 37. DOI: https://doi.org/10.4103/2156-7514.159564

18. Di Marco A., Anguera I., Schmitt M., et al. Late gadolinium enhancement and the risk for ventricular arrhythmias or sudden death in dilated cardiomyopathy: systematic review and meta-analysis // JACC Heart Fail. 2017. Vol. 5. P. 28–38. DOI: https://doi.org/10.1016/j.jchf.2016.09.017

19. Klem I., Klein M., Khan M., et al. Relationship of LVEF and myocardial scar to long-term mortality risk and mode of death in patients with nonischemic cardiomyopathy // Circulation. 2021. Vol. 143. P. 1343–1358. DOI: https://doi.org/10.1161/CIRCULATIONAHA.120.048477

20. Goidescu C.M. Dilated cardiomyopathy produced by lamin A/C gene mutations // Cluju Med. 2013. Vol. 86(4). P. 309–312.

21. Connolly S.J., Dorian P., Roberts R.S., et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial // JAMA. 2006. Vol. 295. P. 165–171. DOI: https://doi.org/10.1001/jama.295.2.165

22. Dinov B., Fiedler L., Schönbauer R., et al. Outcomes in catheter ablation of ventricular tachycardia in dilated nonischemic cardiomyopathy compared with ischemic cardiomyopathy: results from the Prospective Heart Centre of Leipzig VT (HELP-VT) Study // Circulation. 2014. Vol. 129. P. 728–736. DOI: https://doi.org/10.1161/CIRCULATIONAHA.113.003063

23. Muser D., Santangeli P., Castro S.A., et al. Long-term outcome after catheter ablation of ventricular tachycardia in patients with nonischemic dilated cardiomyopathy // Circ. Arrhythm. Electrophysiol. 2016. Vol. 9. e004328. DOI: https://doi.org/10.1161/CIRCEP.116.004328

24. Ebert M., Richter S., Dinov B., et al. Evaluation and management of ventricular tachycardia in patients with dilated cardiomyopathy // Heart Rhythm. 2019. Vol. 16. P. 624–631. DOI: https://doi.org/10.1016/j.hrthm.2018.10.028

25. Kumar S., Androulakis A.F.A., Sellal J-M., et al. Multicenter experience with catheter ablation for ventricular tachycardia in lamin A/C cardiomyopathy // Circ Arrhythm Electrophysiol. 2016. Vol. 9. e004357. DOI: https://doi.org/10.1161/CIRCEP.116.004357

26. Oloriz T., Silberbauer J., Maccabelli G., et al. Catheter ablation of ventricular arrhythmia in nonischemic cardiomyopathy: anteroseptal versus inferolateral scar subtypes // Circ Arrhythm Electrophysiol. 2014. Vol. 7. P. 414–423. DOI: https://doi.org/10.1161/CIRCEP.114.001568

27. Corrado D., Link M.S., Calkins H. Arrhythmogenic right ventricular cardiomyopathy // N Engl J Med. 2017. Vol. 376. P. 1489–1490. DOI: https://doi.org/10.1056/NEJMra1509267

28. Corrado D., Marra M.P., Zorzi A., et al. Diagnosis of arrhythmogenic cardiomyopathy: The Padua criteria // Int J Cardiol. 2020. Vol. 319. P. 106–114. DOI: https://doi.org/10.1016/j.ijcard.2020.06.005.

29. Mazzanti A., Ng K., Faragli A., et al. Arrhythmogenic right ventricular cardiomyopathy: clinical course and predictors of arrhythmic risk // J Am Coll Cardiol. 2016. Vol. 68. P. 2540–2550. DOI: https://doi.org/10.1016/j.jacc.2016.09.951

30. Bhonsale A., Groeneweg J.A., James C.A., et al. Impact of genotype on clinical course in arrhythmogenic right ventricular dysplasia/cardiomyopathy-associated mutation carriers // Eur Heart J. 2015. Vol. 36. P. 847–855. DOI: https://doi.org/10.1093/eurheartj/ehu509

31. Rigato I., Bauce B., Rampazzo A., et al. Compound and digenic heterozygosity predicts life time arrhythmic outcome and sudden cardiac death in desmosomal gene-related arrhythmogenic right ventricular cardiomyopathy // Circ Cardiovasc Genet. 2013. Vol. 6. P. 533–542. DOI: https://doi.org/10.1161/CIRCGENETICS.113.000288

32. Novikova T.N., Novikov V.I., Sayganov S.A., Shcherbakova V.A. 2022 Esc guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: what is new? // Cardiac Arrhythmias. 2022. Vol. 2 (3). P. 7–30. DOI: https://doi.org/10.17816/cardar110961

33. Hulot J.-S., Jouven X., Empana J.-P., et al. Natural history and risk stratification of arrhythmogenic right ventricular dysplasia/cardiomyopathy // Circulation. 2004. Vol. 110. P. 1879–1884. DOI: https://doi.org/10.1161 /01.CIR.0000143375.93288.82

34. Wang W., Cadrin-Tourigny J., Bhonsale A., et al. Arrhythmic outcome of arrhythmogenic right ventricular cardiomyopathy patients without implantable defibrillators // J Cardiovasc Electrophysiol. 2018. Vol. 29. P. 1396–1402. DOI: https://doi.org/10.1111/jce.13668

35. Pinamonti B., Dragos A.M., Pyxaras S.A., et al. Prognostic predictors in arrhythmogenic right ventricular cardiomyopathy: results from a 10-year registry // Eur Heart J. 2011. Vol. 32. P. 1105–1113. DOI: https://doi.org/10.1093/eurheartj/ehr040

36. Brun F., Groeneweg J.A., Gear K., et al. Risk stratification in arrhythmic right ventricular cardiomyopathy without implantable cardioverter-defibrillators // JACC Clin Electrophysiol. 2016. Vol. 2. P. 558–564. DOI: https://doi.org/10.1016/j.jacep.2016.03.015

37. Bhonsale A., James C.A., Tichnell C., et al. Incidence and predictors of implantable cardioverter-defibrillator therapy in patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy undergoing implantable cardioverter-defibrillator implantation for primary prevention // J Am Coll Cardiol. 2011. Vol. 58. P. 1485–1496. DOI: https://doi.org/10.1016/j.jacc.2011.06.043

38. Corrado D., Calkins H., Link M.S., et al. Prophylactic implantable defibrillator in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia and no prior ventricular fibrillation or sustained ventricular tachycardia // Circulation. 2010. Vol. 122. P. 1144–1152. DOI: https://doi.org/10.1161/CIRCULATIONAHA.109.913871

39. Platonov P.G., Haugaa K.H., Bundgaard H., et al. Primary prevention of sudden cardiac death with implantable cardioverter-defibrillator therapy in patients with arrhythmogenic right ventricular cardiomyopathy // Am J Cardiol. 2019. Vol. 123. P. 1156–1162. DOI: https://doi.org/10.1016/j.amjcard.2018.12.049

40. Link M.S., Laidlaw D., Polonsky B., et al. Ventricular arrhythmias in the North American multidisciplinary study of ARVC: predictors, characteristics, and treatment // J Am Coll Cardiol. 2014. Vol. 64. P. 119–125. DOI: https://doi.org/10.1016/j.jacc.2014.04.035

41. Cadrin-Tourigny J., Bosman L.P., Nozza A., et al. A new prediction model for ventricular arrhythmias in arrhythmogenic right ventricular cardiomyopathy // Eur Heart J. 2019. Vol. 40. P. 1850–1858. DOI: https://doi.org/10.1093/eurheartj/ehz103

42. Cadrin-Tourigny J., Bosman L.P., Wang W., et al. Sudden cardiac death prediction in arrhythmogenic right ventricular cardiomyopathy:

a multinational collaboration // Circ Arrhythm Electrophysiol. 2021. Vol. 14. e008509. DOI: https://doi.org/10.1161/CIRCEP.120.008509

43. Wichter T., Borggrefe M., Haverkamp W., et al. Efficacy of antiarrhythmic drugs in patients with arrhythmogenic right ventricular disease. Results in patients with inducible and noninducible ventricular tachycardia // Circulation. 1992. Vol. 86. P. 29–37. DOI: https://doi.org/10.1161/01.cir.86.1.29

44. Marcus G.M., Glidden D.V., Polonsky B., et al. Efficacy of antiarrhythmic drugs in arrhythmogenic right ventricular cardiomyopathy: a report from the North American ARVC Registry // J Am Coll Cardiol. 2009. Vol. 54. P. 609–615. DOI: https://doi.org/10.1016/j.jacc.2009.04.052

45. Mahida S., Venlet J., Saguner A.M., et al. Ablation compared with drug therapy for recurrent ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy: results from a multicenter study // Heart Rhythm. 2019. Vol. 16. P. 536–543. DOI: https://doi.org/10.1016/j.hrthm.2018.10.016

46. Ermakov S., Gerstenfeld E.P., Svetlichnaya Y., Scheinman M.M. Use of flecainide in combination antiarrhythmic therapy in patients with arrhythmogenic right ventricular cardiomyopathy // Heart Rhythm. 2017. Vol. 14. P. 564–569. DOI: https://doi.org/10.1016/j.hrthm.2016.12.010

47. Santangeli P., Zado E.S., Supple G.E., et al. Long-term outcome with catheter ablation of ventricular tachycardia in patients with arrhythmogenic right ventricular cardiomyopathy // Circ Arrhythm Electrophysiol. 2015. Vol. 8. P. 1413–1421. DOI: https://doi.org/10.1161/CIRCEP.115.003562

48. Ommen S.R., Mital S., Burke M.A., et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology / American Heart Association Joint Committee on Clinical Practice Guidelines // Circulation. 2020. Vol. 142. e533–e557. DOI: https://doi.org/10.1161/CIR.0000000000000938

49. Heliö T., Elliott P., Koskenvuo J.W., et al. ESC EORP cardiomyopathy registry: real-life practice of genetic counselling and testing in adult cardiomyopathy patients // ESC Heart Fail. 2020. Vol. 7. P. 3013–3021. DOI: https://doi.org/10.1002/ehf2.12925

50. Ho C.Y., Day S.M., Ashley E.A., et al. Genotype and life time burden of disease in hypertrophic cardiomyopathy: insights from the Sarcomeric Human Cardiomyopathy Registry (SHaRe) // Circulation. 2018. Vol. 138. P. 1387–1398. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.033200

51. Semsarian C., Ingles J., Maron M.S., Maron B.J. New perspectives on the prevalence of hypertrophic cardiomyopathy // J Am Coll Cardiol. 2015. Vol. 65. P. 1249–1254. DOI: https://doi.org/10.1016/j.jacc.2015.01.019

52. Maron B.J., Rowin E.J., Casey S.A., et al. Hypertrophic cardiomyopathy in adulthood as sociated with low cardiovascular mortality with contemporary management strategies // J Am Coll Cardiol. 2015. Vol. 65. P. 1915–1928. DOI: https://doi.org/10.1016/j.jacc.2015.02.061

53. O'Mahony C., Jichi F., Pavlou M., et al. A novel clinical risk prediction model for sudden cardiac death in hypertrophic cardiomyopathy (HCMrisk-SCD) // Eur Heart J. 2014. Vol. 35. P. 2010–2020. DOI: https://doi.org/10.1093/eurheartj/eht439

54. O'Mahony C., Jichi F., Ommen S.R., et al. International External Validation Study of the 2014 European Society of Cardiology Guidelines on Sudden Cardiac Death Prevention in Hypertrophic Cardiomyopathy (EVIDENCE-HCM) // Circulation. 2018. Vol. 137. P. 1015–1023. DOI: https://doi.org/10.1161/CIRCULATIONAHA.117.030437

55. Vriesendorp P.A., Schinkel A.F.L., Liebregts M., et al. Validation of the 2014 European Society of Cardiology guidelines risk prediction model for the primary revention of sudden cardiac death in hypertrophic cardiomyopathy // Circ Arrhythm Electrophysiol. 2015. Vol. 8. P. 829–835. DOI: https://doi.org/10.1161/CIRCEP.114.002553

56. Lorenzini M., Anastasiou Z., O'Mahony C., et al. Mortality among referral patients with hypertrophic cardiomyopathy vs the general European population // JAMA Cardiol. 2020. Vol. 5. P. 73–80. DOI: https://doi.org/10.1001/jamacardio.2019.4534

57. Gimeno J.R., Tomé-Esteban M., Lofiego C., et al. Exercise-induced ventricular arrhythmias and risk of sudden cardiac death in patients with hypertrophic cardiomyopathy // Eur Heart J. 2009. Vol. 30. P. 2599–2605. DOI: https://doi.org/10.1093/eurheartj/ehp327

58. Norrish G., Qu C., Field E., et al. External validation of the HCM Risk-Kids model for predicting sudden cardiac death in childhood hypertrophic cardiomyopathy // Eur J Prev Cardiol. 2022. Vol. 29. P. 678–686. DOI: https://doi.org/10.1093/eurjpc/zwab181

59. Chan R.H., Maron B.J., Olivotto I., et al. Prognostic value of quantitative contrast-enhanced cardiovascular magnetic resonance for the evaluation of sudden death risk in patients with hypertrophic cardiomyopathy // Circulation. 2014. Vol. 130. P. 484–495. DOI: https://doi.org/10.1161/CIRCULATIONAHA.113.007094

60. He D., Ye M., Zhang L., Jiang B. Prognostic significance of late gadolinium enhancement on cardiac magnetic resonance in patients with hypertrophic cardiomyopathy // Heart Lung. 2018. Vol. 47. P. 122–126. DOI: https://doi.org/10.1016/j.hrtlng.2017.10.008

61. Rowin E.J., Maron B.J., Carrick R.T., et al. Outcomes in patients with hypertrophic cardiomyopathy and left ventricular systolic dysfunction // J Am Coll Cardiol. 2020. Vol. 75. P. 3033–3043. DOI: https://doi.org/10.1016/j.jacc.2020.04.045

62. Rowin E.J., Maron B.J., Haas T.S., et al. Hypertrophic cardiomyopathy with left ventricular apical aneurysm: implications for risk stratification and management // J Am Coll Cardiol. 2017. Vol. 69. P. 761–773. DOI: https://doi.org/10.1016/j.jacc.2016.11.063

63. Sadoul N., Prasad K., Elliott P.M., et al. Prospective prognostic assessment of blood pressure response during exercise in patients with hypertrophic cardiomyopathy // Circulation. 1997. Vol. 96. P. 2987–2991. DOI: https://doi.org/10.1161/01.cir.96.9.2987

64. Elliott P.M., Poloniecki J., Dickie S., et al. Sudden death in hypertrophic cardiomyopathy: identification of high risk patients // J Am Coll Cardiol. 2000. Vol. 36. P. 2212–2218. DOI: https://doi.org/10.1016/s0735-1097(00)01003-2

65. Behr E.R., Elliott P., McKenna W.J. Role of invasive EP testing in the evaluation and management of hypertrophic cardiomyopathy // Card Electrophysiol Rev. 2002. Vol. 6. P. 482–486. DOI: https://doi.org/10.1023/a:1021161114347

66. Gatzoulis K.A., Georgopoulos S., Antoniou C.-K., et al. Programmed ventricular stimulation predicts arrhythmic events and survival in hypertrophic cardiomyopathy // Int J Cardiol. 2018. Vol. 254. P. 175–181. DOI: https://doi.org/10.1016/j.ijcard.2017.10.033

67. Elliott P.M., Sharma S., Varnava A., et al. Survival after cardiac arrest or sustained ventricular tachycardia in patients with

hypertrophic cardiomyopathy // J Am Coll Cardiol. 1999. Vol. 33. P. 1596–1601. DOI: https://doi.org/10.1016/s0735-1097(99)00056-x

68. Cecchi F., Maron B.J., Epstein S.E. Long-term outcome of patients with hypertrophic cardiomyopathy successfully resuscitated after cardiac arrest // J Am Coll Cardiol. 1989. Vol. 13. P. 1283–1288. DOI: https://doi.org/10.1016/0735-1097(89)90302-1

69. Maron B.J., Spirito P., Shen W-K., et al. Implantable cardioverter-defibrillators and prevention of sudden cardiac death in hypertrophic cardiomyopathy // JAMA. 2007. Vol. 298. P. 405–412. DOI: https://doi.org/10.1001/jama.298.4.405

70. Link M.S., Bockstall K., Weinstock J., et al. Ventricular tachyarrhythmias in patients with hypertrophic cardiomyopathy and defibrillators: triggers, treatment, and implications // J Cardiovasc Electrophysiol. 2017. Vol. 28. P. 531–537. DOI: https://doi.org/10.1111/jce.13194

71. Dallaglio P.D., DiMarco A., MorenoWeidmann Z., et al. Antitachycardia pacing for shock prevention in patients with hypertrophic cardiomyopathy and ventricular tachycardia // Heart Rhythm. 2020. Vol. 17. P. 1084–1091. DOI: https://doi.org/10.1016/j.hrthm.2020.02.024

72. Adduci C., Semprini L., Palano F., et al. Safety and efficacy of anti-tachycardia pacing in patients with hypertrophic cardiomyopathy implanted with an ICD // Pacing Clin Electrophysiol. 2019. Vol. 42. P. 610–616. DOI: https://doi.org/10.1111/pace.13665

73. Melacini P., Maron B.J., Bobbo F., et al. Evidence that pharmacological strategies lack efficacy for the prevention of sudden death in hypertrophic cardiomyopathy // Heart. 2007. Vol. 93. P. 708–710. DOI: https://doi.org/10.1136/hrt.2006.099416

74. McKenna W.J., Oakley C.M., Krikler D.M., Goodwin J.F. Improved survival with amiodarone in patients with hypertrophic cardiomyopathy and ventricular tachycardia // Br Heart J. 1985. Vol. 53. P. 412–416. DOI: https://doi.org/10.1136/hrt.53.4.412

75. Vaseghi M., Hu T.Y., Tung R., et al. Outcomes of catheter ablation of ventricular tachycardia based on etiology in nonischemic heart disease: an international ventricular tachycardia ablation center collaborative study // JACC Clin Electrophysiol. 2018. Vol. 4. P. 1141–1150. DOI: https://doi.org/10.1016/j.jacep.2018.05.007

76. Igarashi M., Nogami A., Kurosaki K., et al. Radiofrequency catheter ablation of ventricular tachycardia in patients with hypertrophic cardiomyopathy and apical aneurysm // JACC Clin Electrophysiol. 2018. Vol. 4. P. 339–350. DOI: https://doi.org/10.1016/j.jacep.2017.12.020

77. Dukkipati S.R., d'Avila A., Soejima K., et al. Long-term outcomes of combined epicardial and endocardial ablation of monomorphic ventricular tachycardia related to hypertrophic cardiomyopathy // Circ Arrhythm Electrophysiol. 2011. Vol. 4. P. 185–194. DOI: https://doi.org/10.1161/CIRCEP.110.957290

78. Новиков В.И. Кардиомиопатии / В.И. Новиков, Т.Н. Новикова. Москва: МЕДпресс-информ, 2021. 112 с.ISBN 978-5-00030-850-9

79. Towbin J.A., Lorts A., Jefferies J.L. Left ventricular non-compaction cardiomyopathy // Lancet. 2015. Vol. 386(9995). P. 813-25. DOI: https://doi.org/10.1016/S0140-6736(14)61282-4

80. Gerecke B.J., Engberding R. Noncompaction Cardiomyopathy – History and Current Knowledge for Clinical Practice // J Clin Med. 2021. Vol. 10(11). P. 2457. DOI: https://doi.org/10.3390/jcm10112457

81. Aung N., Doimo S., Ricci F., et al. Prognostic significance of left ventricular noncompaction: systematic review and meta-analysis of observational studies // Circ Cardiovasc Imaging. 2020. Vol. 13. e009712. DOI: https://doi.org/10.1161/CIRCIMAGING.119.009712

82. Grigoratos C., Barison A., Ivanov A., et al. Meta-analysis of the prognostic role of late gadolinium enhancement and global systolic impairment in left ventricular noncompaction // JACC Cardiovasc Imaging. 2019. Vol. 12. P. 2141–2151. DOI: https://doi.org/10.1016/j.jcmg.2018.12.029

83. Richard P., Ader F., Roux M., et al. Targeted panel sequencing in adult patients with left ventricular non-compaction reveals a large genetic heterogeneity // Clin Genet. 2019. Vol. 95. P. 356–367. DOI: https://doi.org/10.1111/cge.13484

84. Baig S., Edward N.C., Kotecha D., et al. Ventricular arrhythmia and sudden cardiac death in Fabry disease: a systematic review of risk factors in clinical practice // Europace 2018. Vol. 20. f153–f161. DOI: https://doi.org/10.1093/europace/eux261

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