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Clinical case of successful treatment of focal ventricular arrhythmia in a patient with arrhythmogenic mitral valve prolapse

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ABSTRACT

The problem of managing of patients with mitral valve prolapse and ventricular arrhythmias — arrhythmogenic mitral valve prolapse — is quite relevant in routine clinical practice, which led to the creation in 2022 of an expert consensuses on the management of such patients. Based on the criteria, it is possible to identify a group of people at high risk of sudden cardiac death and implement measures to prevent death. How to manage patients at moderate risk of sudden cardiac death remains unclear. A clinical case of successful treatment of ventricular arrhythmias in a patient with arrhythmogenic mitral valve prolapse is presented.

Keywords: arrhythmic mitral valve prolapse; ventricular arrhythmias.

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Клинический случай успешного лечения фокусной желудочковой аритмии у пациентки с аритмогенным пролапсом митрального клапана

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АННОТАЦИЯ

Проблема ведения пациентов с пролапсом митрального клапана и желудочковыми нарушениями ритма — аритмогенным пролапсом митрального клапана — достаточно актуальна в клинической практике, что привело к появлению в 2022 году экспертного консенсуса по ведению таких больных. На основании созданных критериев можно выявить группу лиц высокого риска внезапной сердечной смерти при пролапсе митрального клапана и осуществить мероприятия по ее предотвращению. Как вести больных с умеренным риском внезапной сердечной смерти остается не до конца понятным. Предлагается клинический случай успешного лечения фокусной желудочковой аритмии у пациентки с пролапсом митрального клапана.

Ключевые слова: аритмогенный пролапс митрального клапана; желудочковые нарушения ритма.

Как цитировать

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Mitral valve prolapse (MVP) is common in the general population; it is most often detected during routine echocardiography (ECG) and has a benign course [1–3]. Most patients with MVP have no clinical manifestations; however, in some cases, individuals with MVP experience serious complications such as severe mitral regurgitation requiring surgical correction, infective endocarditis, systemic emboli, atrial fibrillation, ventricular arrhythmias (VAs), and even sudden death [4, 5]. Sudden cardiac death (SCD) occurs in 0.2%–0.4% of patients with MVP, which is higher than in the general population [3, 6, 7].

Studies on the causal relationship between MVP and SCD showed an association between myocardial electrical instability and structural changes of the mitral apparatus, such as left ventricular fibrosis in the papillary muscles and inferior basal wall, mitral annular disjunction (MAD), and systolic torsion [3, 6, 7].

In recent years, the incidence arrhythmogenic mitral valve prolapse has been reported, which is defined as MVP associated with frequent or complex VAs, including life-threatening ones (i.e., ventricular tachycardia (VT) and ventricular fibrillation (VF) in the absence of any other arrhythmic substrate [with or without MAD]) [8]. An expert consensus on the management of these patients has been published [6]. According to data from various studies, most cases of SCD occur in young healthy women with MAD [3, 7, 9]. A clinical profile of a patient with arrhythmogenic MVP was developed based on case studies presented in the literature. It includes a young or middle-aged woman with lesions in both mitral valve flaps, conduction disturbances in the His bundle branch system, repolarization disorders (*ST* segment displacement and *T* plaque inversion), and polymorphic ventricular extrasystoles with a morphology resembling a right bundle branch block [3, 7, 10, 11].

This case study presents the treatment of focal VA in a middle-aged patient with MVP.

A 54-year-old woman with complaints of heart palpitations and a freezing sensation was admitted to the cardiology clinic of the Mechnikov North-Western State Medical University on October 10, 2023. No conditions in the other organ systems were reported.

The patient's medical history indicates that she first experienced heart palpitations at the age of 30. However, at that time, an examination for rhythm disturbances was not conducted, and no arrhythmias were observed on electrocardiogram (ECG). At the same age, she began to have elevated blood pressure up to 160/90 mmHg, which was subsequently treated with hypotensive medication (ACE inhibitor + Ca-antagonist), resulting in a favorable outcome. Moreover, her total cholesterol levels gradually increased up to 6.6 mmol/L (with triglyceride levels at 1.2 mmol/L, HDL-C at 1.84 mmol/L, LDL-C at 4.21 mmol/L, and an atherogenicity coefficient of 2.6) over an extended period. However, no hypolipidemic therapy was prescribed.

The initial 24-hour ECG monitoring was conducted on March 17, 2020, in the absence of pharmacological intervention. Sinus rhythm with heart rate (HR) ranging 58–140 beats per minute (bpm), with a mean HR of 86 bpm, was noted. Submaximal HR was achieved. The number of type 1 single ventricular extrasystoles was 332 (15 per hour), whereas the number of type 2 single ventricular extrasystoles was 91 (4 per hour). Additionally, seven paired ventricular monomorphic extrasystoles were found. The number of single supraventricular extrasystoles was 114 (5 per hour). No ischemic changes were observed. The patient was prescribed 5 mg of bisoprolol by a cardiologist at her place of residence.

Four months later, a control 24-hour ECG monitoring was performed in conjunction with therapy. The patient exhibited a sinus rhythm with HR ranging 58–138 bpm (average HR: 78 bpm). Additionally, she demonstrated submaximal HR and type 1 single ventricular extrasystoles at a rate of 122 per hour (5 per minute), type 2 single ventricular extrasystoles at a rate of 31 per hour (1 per minute), and single supraventricular extrasystoles at a rate of 79 per hour (3 per minute). ECG revealed no ischemic changes. Considering the favorable clinical response to β-blockers, the patient was instructed to continue therapy.

In 2021 (after experiencing severe stress and the effects of the novel coronavirus), the patient reported increase in the frequency of attacks, which occurred several times a week. These attacks manifested as a sensation of heart palpitations during periods of physical exertion and at rest. The patient described this sensation as "as if everything is tumbling inside." Moreover, during these attacks, the patient experienced dyspnea. Furthermore, two episodes of presyncope were observed, occurring during complete well-being and at rest, accompanied by a sensation of heart palpitations.

A 24-hour ECG was conducted on November 9, 2022; the results are presented in Figure 1. The sinus rhythm exhibited HR ranging 55–136 bpm (average HR: 76 bpm). The frequency of type 1 single ventricular extrasystoles was 19,361 (805 per hour), whereas the frequency of type 2 single ventricular extrasystoles was 1,067 (44 per hour). Additionally, the frequency of paired ventricular monomorphic extrasystoles was 1,267 (53 per hour). Paired ventricular polymorphic extrasystoles were observed at a rate of 364 per hour (15 per hour), whereas nonsustained monomorphic VT was noted at a rate of 40 per day (2 per hour) only during daytime. Similarly, nonsustained polymorphic VT was observed at a rate of 27 per hour (1 per hour) only during daytime.

The patient was initially prescribed sotalol at 120 mg per day during the outpatient phase. However, subsequent attempts to increase the dosage were associated with the development of marked bradycardia, indicating the need to maintain the previous dosage.

In conjunction with sotalol therapy, on June 27, 2023, 24-hour ECG monitoring was performed. The patient

exhibited a sinus rhythm with HR ranging 59–119 bpm (average HR: 79 bpm). Additionally, she displayed type 1 single ventricular extrasystoles, with a total of 16,553 observed over the monitoring period, representing an average of 696 per hour. The frequency of type 2 single ventricular extrasystoles was 67 instances (3 per hour); paired ventricular monomorphic extrasystoles was 982 (41 per hour); paired ventricular polymorphic extrasystoles was 135 (6 per hour) during the day, with no occurrences at night; and nonsustained VT was 34 (1 per hour) during the day, with no occurrences at night (Figure 2).

Considering the persistence of ventricular rhythm disturbances (VRD) of high degree, the patient was admitted to the Cardiology Department of the Mechnikov North-Western State Medical University for examination and determination of further treatment.

The patient's anamnesis showed that her grandmother suddenly died at the age of 42, and her father was

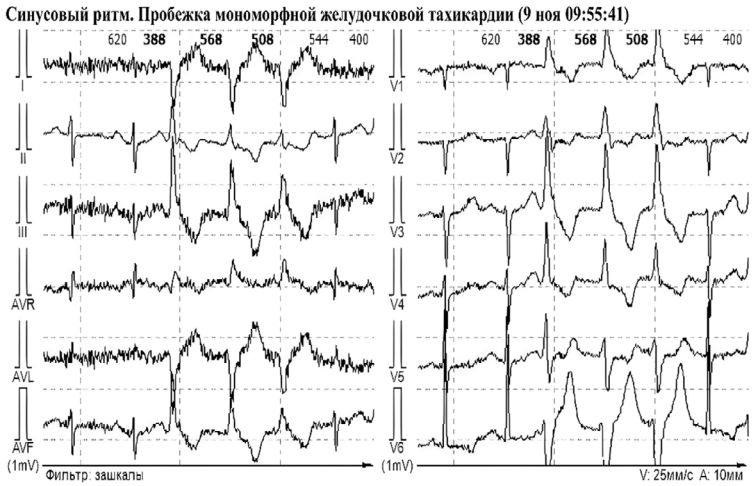

Fig. 1. Episodes of nonsustained VT according to 24-hour ECG monitoring on 11.09.2022

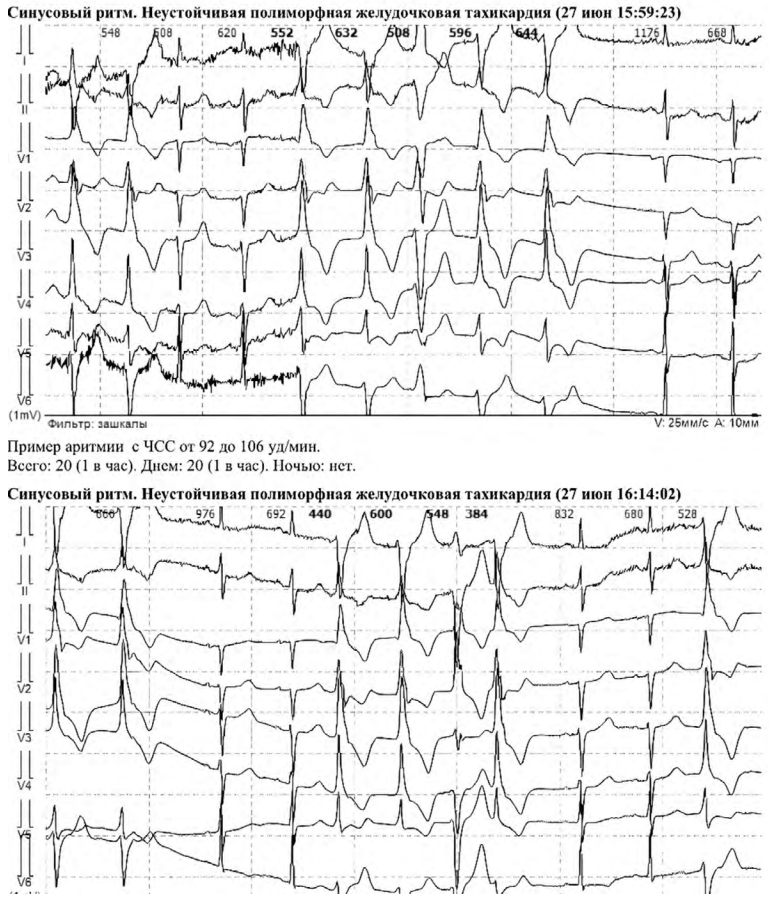

Fig. 2. Episodes of nonsustained polymorphic ventricular tachycardia according to 24-hour ECG monitoring on June 27, 2023

diagnosed with MVP and VRD, for which he was taking drug therapy (the patient found it difficult to answer). We invited the patient's father to the clinic for examination; however, he did not show up.

Since her youth, the patient has been involved in sports (athletics); she has been examined in sports clinics, and no pathology has been detected. No menstrual disorders were detected, and one pregnancy ended with medical abortion at the age of 17 (for social reasons). The patient smokes up to five cigarettes a day for 30 years.

Initial observation upon admission demonstrated that the patient's condition was satisfactory. The patient displayed clear consciousness. She weighed 56 kg, and her height was measured at 165 cm. The patient's pulse rate was 65 bpm, exhibiting an arrhythmic pattern (extrasystole). The characteristics of the pulse were satisfactory. Additionally, the boundaries of relative cardiac bluntness were not dilated. The heart tones were muffled, and no pathological murmurs were audible. The arterial pressure was 125/90 mmHg, and the chest was of the normal shape. The respiratory rate was 16 per minute, and at auscultation, breathing was rigid and conducted in all sections. No adverse respiratory noises were noted.

A series of clinical and laboratory investigations were conducted, including a comprehensive blood analysis and biochemical assessment, which did not reveal any pathological abnormalities. Examination of thyroid status showed no abnormalities.

ECG revealed a sinus rhythm, with a HR of 64 bpm. A blockade of the anterior-upper branch of the left His bundle was observed. A gradual increase in rV1–>V3 was found. Furthermore, an abnormality in the repolarization process was determined, manifesting as a biphasic, weakly positive T wave in leads V4–V6 (Figure 3).

EchoCG data, which was collected for the first time over the entire observation period, indicated that the left ventricle (LV) was not enlarged, the myocardium was not thickened, the interventricular septum was 8 mm, and the LV posterior wall was 9 mm. Additionally, no local contractility disorders were identified, and global contractility was maintained, with an LV ejection fraction of 61.2%. Myxomatous mitral valve

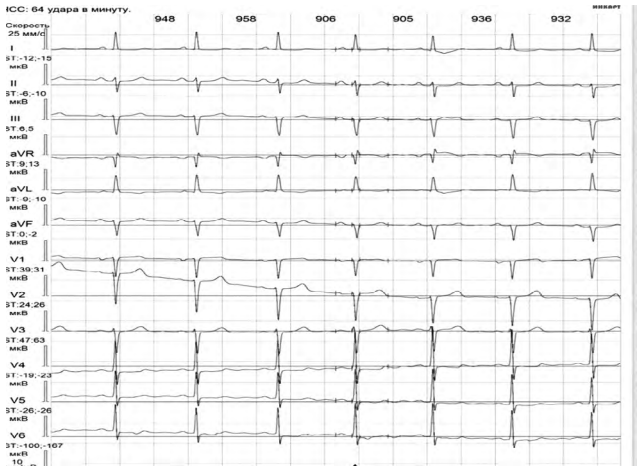

Fig. 3. Electrocardiogram on October 10, 2023

degeneration was observed, along with prolapse of both mitral valve leaflets in the second stage, with a measurement of 8 mm. Stage 1 regurgitation was observed, with a VC of 4 mm (Figure 4).

Upon analysis of the ECG and 24-hour ECG monitoring results of the patient, the localization of premature ventricular contraction in relation to the MV apparatus was not determined. This included the anterior and posterior papillary muscles and anterior and posterior sections of the mitral annulus. The ventricular complexes did not meet the existing criteria for these localizations [6]. However, the morphology of the complexes indicated that they originated from the LV.

Considering the presence of risk factors for ischemic heart disease (e.g., dyslipidemia, arterial hypertension, hereditary predisposition, and smoking), ischemic genesis of rhythm disturbances was excluded through a stress test (stress-echoCG) (with sotalol withdrawal). The results of the stress test demonstrated that the patient achieved a submaximal HR at a workload of 75 watts (equivalent to 8.40 METs). The initial examination yielded no evidence of local contractility disorders. No local contractility disorders were observed at the peak of the load. During the test, rhythm disturbances, including single and paired polymorphic extrasystoles (bigeminy), and episodes of nonsustained VT, were identified. However, the frequency of these disturbances

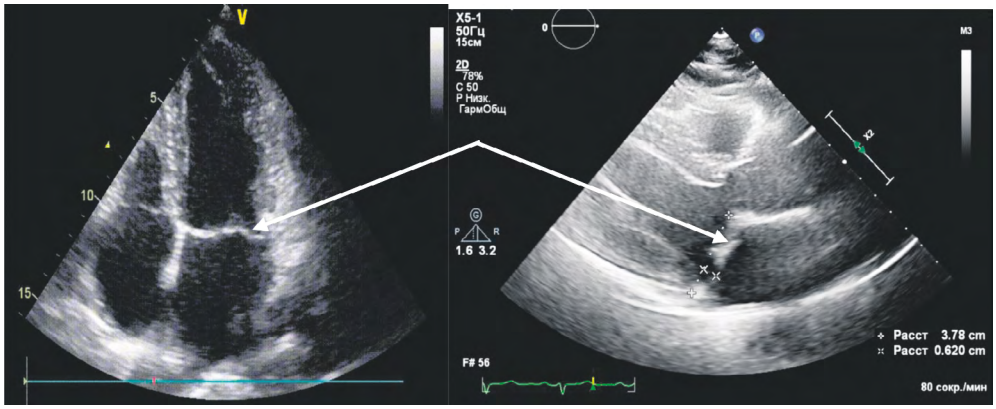

Fig. 4. Echocardiogram on 10/11/2023. Arrows show mitral valve prolapse

did not significantly increase with the increase in exercise level and HR. The pretest ECG revealed single polymorphic extrasystoles, which exhibited no significant change in frequency during exercise. In the recovery period, the degree of severity of single and paired polymorphic extrasystoles did not differ from that observed in the preload test data.

Subsequently, the patient underwent diagnostic coronary angiography, which showed that the coronary arteries were unchanged. To exclude myocarditis and identify the morphologic substrate of VRDs, myocardial magnetic resonance imaging (MRI) with contrast (gadolinium) was conducted. Cardiac MRI was performed on a tomograph with a 3T magnetic field induction, in accordance with the standard protocol, with targeted assessment of the mitral valve.

MRI data indicated that the contractile function was found to be LV ejection fraction of 61% (59%–77%), with a stroke volume of 84 mL (57–113 mL). The end-diastolic volume was recorded at 138 mL (86–166 mL), and the end-diastolic volume index was 85 mL/m^2 (56–90 mL/m^2). Additionally, the end-systolic volume was 54 mL (22–59 mL), and the end-systolic volume index was 33 ml/m^2 (14–33 ml/m^2). The myocardial mass was 139 g (72–144 g), with a mass index of 87 g (48–78 g) (normal values for age and sex are provided in parentheses). Analysis of the images obtained in Cine mode determined posterior mitral valve leaflet prolapse, whereas no indications of MAD were identified. In a series of delayed accumulation of contrast agent in the volume of 20 mL, no evidence of accumulation in the myocardium was identified. Furthermore, no data were obtained regarding inflammatory and fibrotic changes.

Owing to the ineffectiveness of antiarrhythmic therapy, radiofrequency catheter ablation (RFA) of the area of the most frequent arrhythmia was performed, as well as an extended protocol of endocardial electrophysiological study (eEPS), considering the patient's risk factors for SCD. The patient was referred to the Department of Surgical Treatment of Complex Cardiac Dysrhythmias.

The results of the eEPS indicated that, at the level of programmed stimulation, AV conduction was decremental without gaps or ECHO responses. Ultra-frequent stimulation did not induce atrial fibrillation, atrial flutter, or atrial tachycardia. In ultra-frequent stimulation from the LV apex and LV output tract, up to three extrastimuli were applied without inducing the LV. An electroanatomical map was constructed, presenting the earliest activation in the anterior septal region, closer to the LV apex, in response to LV extrasystole. In this zone, RF current with a 40 W power was applied for at least 2 minutes, resulting in the disappearance of VE.

In the postoperative period, the patient exhibited a notable enhancement in her overall well-being, accompanied by improvement of cardiac palpitations. She was discharged for outpatient treatment, with the following recommendations: atorvastatin, 40 mg per day; perindopril, 4 mg per day; amlodipine, 5 mg per day; and bisoprolol, 5 mg per day.

In February 2024, a 24-hour ECG monitoring was conducted on an outpatient basis. The patient exhibited a sinus rhythm with HR ranging 57–139 bpm (mean HR: 76 bpm). Moreover, the patient displayed type 1 single ventricular extrasystoles at a rate of 118 per hour (5 per hour), type 2 single ventricular extrasystoles at a rate of 28 per hour (1 per hour), and single supraventricular extrasystoles at a rate of 79 per hour (3 per hour). No ischemic changes were identified on ECG. The ECG monitoring data indicated that the intervention had a favorable antiarrhythmic effect.

DISCUSSION

The clinical manifestations of MVP are often determined by the severity of mitral regurgitation (MR) [4, 6], with a severe degree of which, left atrial and LV remodeling develops. In cases wherein the MR volume is insignificant and the left heart chambers are of normal size, the prognosis for MVP is

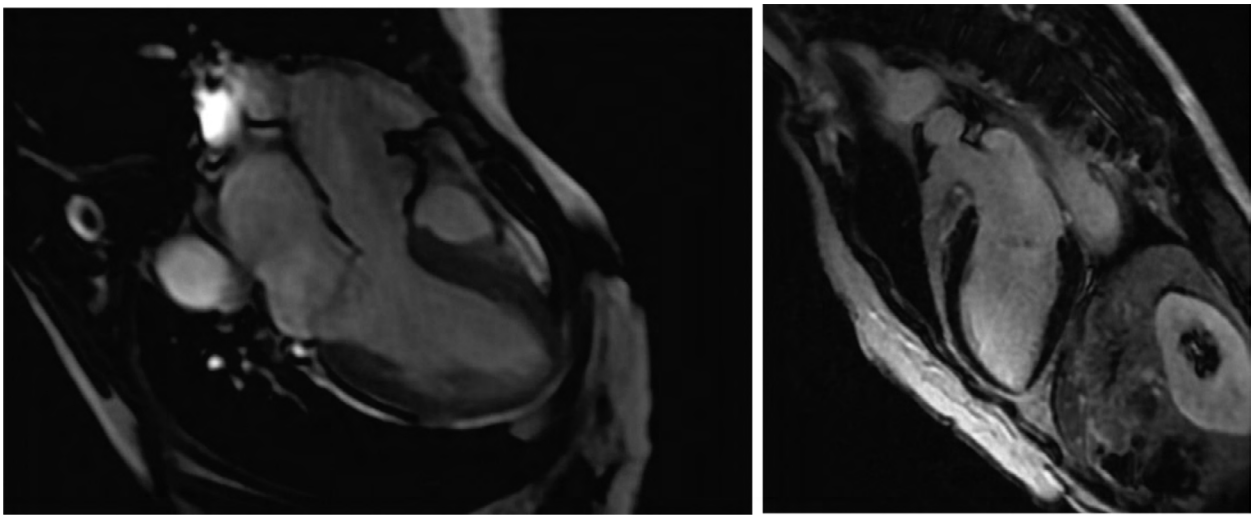

Fig. 5. Myocardial magnetic resonance imaging. Phase of delayed contrast enhancement

considered favorable [12]. Conversely, several studies have demonstrated that individuals with MVP may experience life-threatening VRDs and SCD events, irrespective of the degree of MR or LV dysfunction [9, 13, 14].

In a study by Essayagh B et al. on a large cohort of patients (n = 595) with isolated MVP, VPDs were rarely identified on 24-hour ECG monitoring. However, unstable VT, which occurred in 9% of patients and manifested as ≥180 bpm, was identified as a predictor of SCD.

In recent years, various studies have demonstrated a significant correlation between MAD and MVP. These findings substantiate the hypothesis that a higher prevalence of MAD is evident in patients with MVP than in those with MVP and no arrhythmia [11, 15]. Conversely, evidence shows that MAD is associated with complex arrhythmic events in the absence of MVP, indicating that MAD may be considered a marker for malignant VRDs [16].

Considering the worsening of clinical symptoms and the appearance of more severe VRDs on ECG monitoring after a new coronavirus infection in 2021, our patient was assumed to have viral myocarditis. Clinical cases of increasing clinical symptoms in patients with MVP during COVID-19 have been described; however, all of them were associated with cardiac insufficiency in such patients due to acute myocarditis and increased MR without subsequent increase in VRDs [17].

In 28%–37% [18, 19] of patients with MVP, MRI shows areas of fibrosis, often localized in the annulus and papillary muscles, as well as in the inferior basal wall of the LV [20]. In our patient, cardiac MRI did not reveal severe MR and LV dysfunction, as well as MAD, signs of current or transferred myocarditis, and foci of fibrosis. This is common in idiopathic VAs. Furthermore, the occurrence of VRDs in MVP may be associated with the anatomical substrate (foci of papillary muscle fibrosis, involvement of Purkinje fibers, etc.), that is, the reentry mechanism, and with the tension of subvalvular structures with the realization of the postdepolarization mechanism [6]. Indirect indications of this condition may be the repolarization disturbances, which was observed in our patient, manifesting as biphasic, weakly positive *T* waves in leads V4–V6. Furthermore, the presence of myxomatous mitral valve abnormalities does not exclude the possibility of underlying structural pathology in other regions of the myocardium, including at the cellular level, which may not be detected through MRI.

With the absence of established protocols for patients with MVP, the standard protocol for endocardial EPS is employed in accordance with the consensus for AMVP [6]. In an independent systematic review on this topic [21], in patients with MVP who survived an episode of SCD, VT was induced in 5% of cases, supraventricular tachycardia in 23%, and FV in 18%. In 55% of cases, VRDs were not induced. Based on these findings, the authors concluded that the diagnostic value of eEPS using the standard protocol in this situation is limited.

In the present case, EPS demonstrated early activation in the anterior septal region, in proximity to the apex, concurrent with LV extrasystole. VT was not induced. Subsequent RF interaction in this area resulted in the elimination of a prevalent type of monomorphic VE, contributing to a reduction in the number of other types. The mapping data indicated the absence of focal activity from the papillary muscle structures.

Thus, despite the unproven association of the early activation zone according to eEPS data with the mitral valve area in our patient, the patient's pathology may be considered as arrhythmogenic MVP, because according to the expert consensus [6], the category of persons with AMVP includes patients with MVP (with or without MAD) with frequent (>5% of the total number of complexes) and/or polymorphic, paired VE, supraventricular tachycardia, VT, LV, and VF in the absence of other proven arrhythmogenic substrate. Furthermore, the patient exhibited characteristics of the phenotype of arrhythmogenic MVP, which was possibly of hereditary origin. She was a middle-aged woman with an asthenic physique, presenting with prolapse of two mitral valve leaflets, biphasic repolarization disorder on ECG, and polymorphic ventricular extrasystoles with right bundle branch block morphology. Additionally, ECG showed weakly positive T waves in leads V4–V6. As previously stated, the patient's father has MVP and is undergoing treatment for arrhythmias, and the patient's grandmother (on her father's side) suddenly died at the age of 42.

According to the 2022 expert consensus on the management of patients with arrhythmogenic MVP from the European Heart Rhythm Association (EHRA), high-risk patients are defined as patients with sustained VT originating from a non-right or non-LV outflow tract and those with spontaneous or unstable VT exceeding 180 bmp, syncopal states, ECG changes, SCD in close relatives, severe MR, MAD, and contrast accumulation on MRI. Our patient corresponded to the moderate risk group: polymorphic VE, unstable VT >180 bpm, frequent and paired VE, repolarization abnormalities on ECG, and history of presyncope. Therefore, arrhythmologists were requested to perform a complete eEPS.

Patients with arrhythmogenic MVP are usually prescribed the same antiarrhythmic drugs as other patients with VRDs [8, 10]. However, there are currently no studies confirming their efficacy in this pathology. According to the EHRA expert consensus [6], four treatment options are currently considered to prevent SCD in patients with arrhythmogenic MVP, namely, medical therapy, catheter ablation, ICD implantation, and mitral valve surgery. Treatment for arrhythmogenic MVP are aimed at improving symptom tolerance and survival.

Catheter ablation is an effective treatment for malignant arrhythmias in patients with MVP [10, 11, 22, 23]. F.F. Syed et al. demonstrated that RFA is feasible in patients with MVP with symptomatic, drug-resistant VAs [18].

Currently, data supporting the efficacy of cardioverter defibrillator (CD) implantation in patients at high risk for SCD in MVP are limited. Some experts recommend the use of eEPS to distinguish the risk of SCD in these patients and recommend CD implantation for primary prevention of SCD induction of sustained VT [6, 7, 15]. CD implantation in patients with arrhythmogenic MVP who have experienced cardiac arrest is performed according to the principle of secondary prevention of SCD [6, 7, 10, 11].

In our patient, antiarrhythmic drugs were ineffective, CD implantation or MR correction was not indicated, and the VRDs were symptomatic despite medical therapy. Therefore, considering the frequency and nature of the VRDs and eEPS data, an invasive intervention, namely, catheter ablation of the arrhythmogenic focus, was performed, which proved to be effective. However, the patient should be monitored by a cardiologist because MVP persists and the presence of another occult arrhythmogenic substrate cannot be excluded, as it is known that SCD develops years after the detection of VRDs in patients with AMVP [6].

CONCLUSIONS

Currently, arrhythmogenic MVP has been increasingly described. Clinical, electrocardiographic, and electrophysiological data reveal an association between MVP and SCD. Patients with mitral annular disruption are at highest risk for SCD. Moreover, malignant VAs are found in patients with MVP without MAD.

However, the mechanisms of VRDs in patients with MVP require further investigation using various more accurate methods of invasive and noninvasive mapping and the study of the cellular mechanisms of rhythm disturbances. Further search for risk markers and development of optimal evidence-based treatment strategies in these patients are warranted. General practitioners should be aware that “harmless” MVP can be fatal; thus, patients with MVP who complain of arrhythmias should undergo 24-hour ECG monitoring.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors confirm that their authorship complies with the international ICMJE criteria (all authors have made a significant contribution to the development of the concept, research, and preparation of the article, as well as read and approved the final version before its publication). Personal contribution of the authors: N.S. Tretyakova — examination of the patient, primary data obtaining, analyzing the data obtained, writing the text; S.A. Boldueva — experimental design, writing the main part of the text; making final edits; I.A. Leonova — experimental design, writing the text; literature review; O.S. Shvetsova — examination of the patient, primary data obtaining, analyzing the data obtained; L.S. Evdokimova — MRI investigation, literature review.

Competing interests. The authors declare that they have no competing interests.

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Informed consent for publication. Written consent was obtained from the patient for publication of relevant medical information and all accompanying images within the manuscript.

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ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией. Вклад каждого автора: Н.С. Третьякова — обследование пациента, получение первичных данных, анализ полученных данных, написание текста; С.А. Болдуева — концепция и дизайн исследования, написание текста; внесение окончательной правки; И.А. Леонова — концепция и дизайн исследования, анализ литературы, написание текста; О.С. Швецова — обследование пациентки, получение первичных данных, анализ полученных данных; Л.С. Евдокимова — выполнение МРТ-исследования, обзор литературы.

Конфликт интересов. Авторы заявляют об отсутствии потенциального конфликта интересов, требующего раскрытия в данной статье.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования. Все исследования, представленные в данной статье, выполнялись в рамках рутинной клинической практики по полису обязательного медицинского страхования

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REFERENCES

1. Avierinos JF, Gersh BJ, Melton LJ, 3rd, et al. Natural history of asymptomatic mitral valve prolapse in the community. *Circulation*. 2002;106(11):1355–1361. doi: 10.1161/01.cir.0000028933.34260.09

2. Freed LA, Benjamin EJ, Levy D, et al. Mitral valve prolapse in the general population: The benign nature of echocardiographic features in the Framingham Heart Study. *J Am Coll Cardiol*. 2002;40(7): 1298–1304. doi: 10.1016/s0735-1097(02)02161-7

3. Shirobokikh OE, Bylova NA. Mitral valve prolapse and sudden cardiac death: who is in the risk group? *The Russian Archives of Internal Medicine*. 2016;6(3):25–29. EDN: VZTUIB doi: 10.20514/2226-6704-2016-6-3-25-29

4. Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American college of cardiology/American heart association joint committee on clinical practice guidelines. *Circulation*. 2021;143(5):35–71. doi: 10.1161/CIR.0000000000000932

5. Myslitskaya GV, Novikov VI, Uzilevskaya PA. Heart rhythm disorders in mitral valve prolapse syndrome. *Cardiology*. 1986;(8): 49–53. (In Russ.)

6. Sabbag A, Essayagh B, Barrera JDR, et al. EHRA expert consensus statement on arrhythmic mitral valve prolapse and mitral annular disjunction complex in collaboration with the ESC Council on valvular heart disease and the European Association of Cardiovascular Imaging endorsed cby the Heart Rhythm Society, by the Asia Pacific Heart Rhythm Society, and by the Latin American Heart Rhythm Society. *Europace*. 2022;24(12):1981–2003. doi: 10.1093/europace/euac125

7. Kuzhel DA, Matyushin GV, Savchenko EA. Arrhythmic mitral valve prolapse: new menaces of the known disease. *Rational Pharmacotherapy in Cardiology*.2023;19(1):77–82. EDN: NGNGWE doi: 10.20996/1819-6446-2023-01-05

8. Kubala M, Essayagh B, Michelena HI, et al. Arrhythmic mitral valve prolapse in 2023: Evidence-based update. *Front Cardiovasc Med*. 2023;10:1130174. doi: 10.3389/fcvm.2023.1130174

9. Essayagh B, Sabbag A, Antoine C, et al. Presentation and outcome of arrhythmic mitral valve prolapse. *J Am Coll Cardiol*. 2020;76(6):637–649. doi: 10.1016/j.jacc.2020.06.029

10. Basso C, Iliceto S, Thiene G, Perazzolo Marra M. Mitral valve prolapse, ventricular arrhythmias, and sudden death. *Circulation*. 2019;140(11):952–964. doi: 10.1161/CIRCULATIONAHA.118.034075

11. Korovesis TG, Koutrolou-Sotiropoulou P, Katritsis DG. Arrhythmogenic mitral valve prolapse. *Arrhythm Electrophysiol Rev*. 2022;11:e16. doi: 10.15420/aer.2021.28

12. Turker Y, Ozaydin M, Acar G, et al. Predictors of ventricular arrhythmias in patients with mitral valve prolapse. *Int J Cardiovasc Imaging*. 2010;26(2):139–145. doi: 10.1007/s10554-009-9514-6

13. Muthukumar L, Rahman F, Jan MF, et al. The Pickelhaube sign: novel echocardiographic risk marker for malignant mitral valve prolapse syndrome. *JACC Cardiovasc Imaging*. 2017;10(9): 1078–1080. doi: 10.1016/j.jcmg.2016.09.016

14. Han HC, Ha FJ, Teh AW, et al. Mitral valve prolapse and sudden cardiac death: a systematic review. *J Am Heart Assoc*. 2018;7(23):e010584. doi: 10.1161/JAHA.118.010584

15. 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. *Eur Heart J.* 2022;43(40):3997–4126. doi: 10.1093/eurheartj/ehac262

16. Dejgaard LA, Skjølsvik ET, Lie ØH, et al. The mitral annulus disjunction arrhythmic syndrome. *J Am Coll Cardiol*. 2018;72(14):1600–1609. doi: 10.1016/j.jacc.2018.07.070

17. Alsagaff MY, Shonafi KA, Handari SD, et al. An unexpected overlap syndrome of mitral valve prolapse with COVID-19 related myocarditis: case report from two patients. *Ann Med Surg (Lond).* 2023;85(4):1276–1281. doi: 10.1097/MS9.0000000000000522

18. Kitkungvan D, Nabi F, Kim RJ, et al. Myocardial fibrosis in patients with primary mitral regurgitation with and without prolapse. *J Am Coll Cardiol*. 2018;72(8):823–834. doi: 10.1016/j.jacc.2018.06.048

19. Constant Dit Beaufils AL, Huttin O, Jobbe-Duval A, et al. Replacement myocardial fibrosis in patients with mitral valve prolapse: relation to mitral regurgitation, ventricular remodeling, and arrhythmia. *Circulation*. 2021;143(18):1763–1774. doi: 10.1161/CIRCULATIONAHA.120.050214

20. Marra MP, Basso C, De Lazzari M, et al. Morphofunctional abnormalities of mitral annulus and arrhythmic mitral valve prolapse. *Circ Cardiovasc Imaging*. 2016;9(8):e005030. doi: 10.1161/CIRCIMAGING.116.005030

21. Han HC, Ha FJ, Teh AW, et al. Mitral valve prolapse and sudden cardiac death: a systematic review. *J Am Heart Assoc*. 2018;7(23):e010584. doi: 10.1161/JAHA.118.010584

22. Syed FF, Ackerman MJ, McLeod CJ, et al. Sites of successful ventricular fibrillation ablation in bileaflet mitral valve prolapse syndrome. *Circ Arrhythm Electrophysiol.* 2016;9(5):e004005. doi: 10.1161/CIRCEP.116.004005

23. Hong T, Yang M, Zhong L, et al. Ventricular premature contraction associated with mitral valve prolapse. *Int J Cardiol.* 2016;221: 1144–1149. doi: 10.1016/j.ijcard.2016.06.252

СПИСОК ЛИТЕРАТУРЫ

1. Avierinos J.F., Gersh B.J., Melton L.J. 3rd, et al. Natural history of asymptomatic mitral valve prolapse in the community // Circulation. 2002. Vol. 106, N. 11. P. 1355–1361. doi: 10.1161/01.cir.0000028933.34260.09

2. Freed L.A., Benjamin E.J., Levy D., et al. Mitral valve prolapse in the general population: The benign nature of echocardiographic features in the Framingham Heart Study // J Am Coll Cardiol. 2002. Vol 40, N 7. P. 1298–1304. doi: 10.1016/s0735-1097(02)02161-7

3. Широбоких О.Е., Былова Н.А. Пролапс митрального клапана и внезапная сердечная смерть: кто в группе риска? // Архивъ внутренней медицины. 2016. Т. 6, № 3. С. 25–29. EDN: VZTUIB doi: 10.20514/2226-6704-2016-6-3-25-29

4. Otto C.M., Nishimura R.A., Bonow R.O., et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American college of cardiology/American heart association joint committee on clinical practice guidelines // Circulation. 2021. Vol. 143, N 5. P. 35–71. doi: 10.1161/CIR.0000000000000932

5. Мыслицкая Г.В., Новиков В.И., Узилевская Р.А. Нарушения сердечного ритма при синдроме пролапса митрального клапана // Кардиология. 1986. № 8. С. 49–53.

6. Sabbag A., Essayagh B., Barrera J.D.R., et al. EHRA expert consensus statement on arrhythmic mitral valve prolapse and mitral annular disjunction complex in collaboration with the ESC Council on valvular heart disease and the European Association of Cardiovascular Imaging endorsed cby the Heart Rhythm Society, by the Asia Pacific Heart Rhythm Society, and by the Latin American Heart Rhythm Society // Europace. 2022. Vol. 24, N 12. P. 1981–2003. doi: 10.1093/europace/euac125
7. Кужель Д.А., Матюшин Г.В., Савченко Е.А. Аритмогенный пролапс митрального клапана: новые угрозы известного заболевания // Рациональная Фармакотерапия в Кардиологии. 2023. Т. 19, № 1. С. 77–82. EDN: NGNGWE doi: 10.20996/1819-6446-2023-01-05
8. Kubala M., Essayagh B., Michelena H.I., et al. Arrhythmic mitral valve prolapse in 2023: Evidence-based update // Front Cardiovasc Med. 2023. Vol. 10. P. 1130174. doi: 10.3389/fcvm.2023.1130174
9. Essayagh B., Sabbag A., Antoine C., et al. Presentation and outcome of arrhythmic mitral valve prolapse // J Am Coll Cardiol. 2020. Vol. 76, N 6. P. 637–649. doi: 10.1016/j.jacc.2020.06.029
10. Basso C., Iliceto S., Thiene G., Perazzolo Marra M. Mitral valve prolapse, ventricular arrhythmias, and sudden death // Circulation. 2019. Vol. 140, N 11. P. 952–964. doi: 10.1161/CIRCULATIONAHA.118.034075
11. Korovesis T.G., Koutrolou-Sotiropoulou P., Katritsis D.G. Arrhythmogenic mitral valve prolapse // Arrhythm Electrophysiol Rev. 2022. Vol. 11. P. e16. doi: 10.15420/aer.2021.28
12. Turker Y., Ozaydin M., Acar G., et al. Predictors of ventricular arrhythmias in patients with mitral valve prolapse // Int J Cardiovasc Imaging. 2010. Vol. 26, N 2. P. 139–145. doi: 10.1007/s10554-009-9514-6
13. Muthukumar L., Rahman F., Jan M.F., et al. The Pickelhaube sign: novel echocardiographic risk marker for malignant mitral valve prolapse syndrome // JACC Cardiovasc Imaging. 2017. Vol. 10, N 9. P. 1078–1080. doi: 10.1016/j.jcmg.2016.09.016
14. Han H.C., Ha F.J., Teh A.W., et al. Mitral valve prolapse and sudden cardiac death: a systematic review // J Am Heart Assoc. 2018. Vol. 7, N 23. P. e010584. doi: 10.1161/JAHA.118.010584
15. 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 // Eur Heart J. 2022. Vol. 43, N 40. P. 3997–4126. doi: 10.1093/eurheartj/ehac262
16. Dejgaard L.A., Skjølsvik E.T., Lie Ø.H., et al. The mitral annulus disjunction arrhythmic syndrome // J Am Coll Cardiol. 2018. Vol. 72, N 14. P. 1600–1609. doi: 10.1016/j.jacc.2018.07.070
17. Alsagaff M.Y., Shonafi K.A., Handari S.D., et al. An unexpected overlap syndrome of mitral valve prolapse with COVID-19 related myocarditis: case report from two patients // Ann Med Surg (Lond). 2023. Vol. 85, N 4. P. 1276–1281. doi: 10.1097/MS9.0000000000000522
18. Kitkungvan D., Nabi F., Kim R.J., et al. Myocardial fibrosis in patients with primary mitral regurgitation with and without prolapse // J Am Coll Cardiol. 2018. Vol. 72, N 8. P. 823–834. doi: 10.1016/j.jacc.2018.06.048
19. Constant Dit Beaufils A.L., Huttin O., Jobbe-Duval A., et al. Replacement myocardial fibrosis in patients with mitral valve prolapse: relation to mitral regurgitation, ventricular remodeling, and arrhythmia // Circulation. 2021. Vol. 143, N 18. P. 1763–1774. doi: 10.1161/CIRCULATIONAHA.120.050214
20. Marra M.P., Basso C., De Lazzari M., et al. Morphofunctional abnormalities of mitral annulus and arrhythmic mitral valve prolapse // Circ Cardiovasc Imaging. 2016. Vol. 9, N 8. P. e005030. doi: 10.1161/CIRCIMAGING.116.005030
21. Han H.C., Ha F.J., Teh A.W., et al. Mitral valve prolapse and sudden cardiac death: a systematic review // J Am Heart Assoc. 2018. Vol 7, N 23. P. e010584. doi: 10.1161/JAHA.118.010584
22. Syed F.F., Ackerman M.J., McLeod C.J., et al. Sites of successful ventricular fibrillation ablation in bileaflet mitral valve prolapse syndrome // Circ Arrhythm Electrophysiol. 2016. Vol. 9, N 5. P. e004005. doi: 10.1161/CIRCEP.116.004005
23. Hong T., Yang M., Zhong L., et al. Ventricular premature contraction associated with mitral valve prolapse // Int J Cardiol. 2016. Vol. 221. P. 1144–1149. doi: 10.1016/j.ijcard.2016.06.252

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