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Arrhythmic phenotypes of cardiac laminopathies: a case series

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ABSTRACT

This article conveys clinical cases of patients with cardiac laminopathy caused by mutations in the *LMNA* gene, the early manifestations of which were supraventricular, ventricular tachyarrhythmias and conduction disorders in the absence of myocardial structural changes. Moreover, it is shown the evolution of rhythm and conduction abnormalities during the follow-up period, as well as the tendency of mutation carriers in the *LMNA* gene to develop life-threatening ventricular tachyarrhythmias and conduction disorders with a high risk of sudden cardiac death. Furthermore, herein are provided key recommendations of European and American experts regarding the concept of distinguishing laminopathies for mandatory molecular genetic testing, given that *LMNA* mutations are associated with a poor prognosis. The data obtained confirm the importance of conducting a molecular genetic study using high-throughput sequencing of genes associated with hereditary rhythm disorders, including the *LMNA* gene, in the presence of clinical manifestations such as syncope, conduction disorders (atrioventricular block, sinus node dysfunction), supraventricular and ventricular tachyarrhythmias in combination with a family history, notably in the absence of structural heart diseases. Timely molecular genetic testing may facilitate the appropriate treatment including a cardioverter-defibrillator implantation.

Keywords: laminopathy; LMNA gene; sudden cardiac death; supraventricular and ventricular tachyarrhythmias; conduction disorders.

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Аритмические проявления кардиоламинопатии (клинические наблюдения)

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АННОТАЦИЯ

Представлены клинические наблюдения двух пациентов с кардиоламинопатией, обусловленной мутациями в гене *LMNA*, ранними проявлениями которой были наджелудочковые, желудочковые нарушения ритма и проводимости при отсутствии структурных изменений в сердце. Показана эволюция нарушений ритма и проводимости за период наблюдения, склонность носителей мутаций в гене *LMNA* к развитию злокачественных желудочковых тахиаритмий и нарушений проводимости с высоким риском внезапной сердечной смерти. Также приведены основные положения европейских и американских экспертов относительно концепции выделения ламиновых фенотипов для обязательного молекулярно-генетического тестирования, так как носители мутаций *LMNA* ассоциированы с плохим прогнозом. Полученные данные подтверждают важность проведения молекулярно-генетического исследования методом высокопроизводительного секвенирования генов, ассоциированных с наследственными нарушениями ритма, включая ген *LMNA*, при наличии таких клинических признаков, как синкопальные состояния, нарушения проводимости (атриовентрикулярные блокады, дисфункция синусового узла), суправентрикулярные и желудочковые тахиаритмии в сочетании с семейным анамнезом даже при отсутствии структурных нарушений миокарда, для верификации диагноза кардиоламинопатии и определения стратегии лечения. Своевременное проведение молекулярно-генетического тестирования позволяет определить оптимальную тактику лечения и необходимость профилактической имплантации кардиовертера-дефибриллятора.

Ключевые слова: ламинопатия; ген LMNA; внезапная сердечная смерть; наджелудочковые и желудочковые тахиаритмии; нарушения проводимости.

Как цитировать

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ЭКО•ВЕКТОР

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INTRODUCTION

Laminopathies constitute a group of hereditary diseases caused by mutations in the *LMNA* gene, which encodes two nuclear membrane proteins, lamin A and lamin C. These disorders exhibit marked phenotypic heterogeneity, encompassing cardiac diseases, neuromuscular disorders, and metabolic abnormalities [1]. To date, 498 *LMNA* mutations have been described, associated with >15 phenotypes [2].

The spectrum of cardiac involvement ranges from supraventricular tachycardia and/or conduction disorders to dilated cardiomyopathy (DCM) and ventricular tachyarrhythmias [3]. The clinical course of cardiac laminopathies is characterized by a high incidence of arrhythmic events, including sudden cardiac death (SCD), sustained ventricular tachycardia (VT), severe bradycardia, and high-grade atrioventricular (AV) block, even in the presence of mild left ventricular (LV) dysfunction [4]. Notably, electrical dysfunction often precedes structural heart abnormalities by several years to more than a decade, according to published data [2, 5].

In its typical form, electrical disease manifests with mild arrhythmias before or during the third decade of life. It has been reported that after the age of 30 92% of patients with *LMNA* mutations develop arrhythmias, including first-degree AV block, frequent premature ventricular contractions (PVCs), or nonsustained paroxysmal VT [6]. With age, second- or third-degree AV block typically develops and may lead to SCD [7]. Overall, 44% of patients older than 30 years eventually require pacemaker implantation due to bradyarrhythmias [6].

Supraventricular tachyarrhythmias, including atrial fibrillation (AF), atrial flutter (AFL), and focal atrial tachycardia, occur as manifestations of atrial disease. In particular, AF has been shown to progress from paroxysmal to persistent or permanent forms (45%) and is associated with a high incidence of thromboembolic complications (10% within 7 years) [2].

Ventricular arrhythmias, including cardiac arrest, VT, and ventricular fibrillation (VF), are typical manifestations of laminopathies [5, 8]. However, life-threatening arrhythmias often appear as the first clinical sign, as they are usually preceded by non-life-threatening arrhythmias or mild structural cardiac diseases[9]. The incidence SCD in cardiac laminopathies is significantly higher than that of end-stage chronic heart failure (HF), as SCD occurs at least four times more frequently than lethal outcome from HF and in 50% of cases occurs before the onset of symptomatic structural cardiac abnormalities [5]. Consequently, the only reliable method for SCD prevention is the implantation of a cardioverter-defibrillator (ICD) [6].

Although the clinical manifestations of *LMNA* mutations have been extensively described, primarily in patients with DCM phenotype, early arrhythmic manifestations, arrhythmic event progression, and the natural course of the disease remain clinically relevant.

The study aims to assess early arrhythmic manifestations in patients with cardiac laminopathies in the absence of structural cardiac abnormalities based on clinical case observations.

CASE DESCRIPTION 1

A 23-year-old woman (Patient K.) was admitted to the cardiology department of the Republican Scientific and Practical Center of Cardiology in January 2024 with complaints of frequent palpitations accompanied by dizziness, occasional irregular heartbeats, and dyspnea during high-intensity physical exertion or while climbing more than three flights of stairs. Anamnesis morbi: the patient reported transient loss of consciousness with perioral cyanosis until the age of six, for which she was followed up by neurologists. Her syncopal episodes resolved after the age of six. Neurological examination revealed no abnormalities. There was no family history of syncope or SCD. The patient experienced arrhythmias since 2018 (Table 1). That same year, patient underwent a transesophageal electrophysiological study, which induced irregular tachycardia with a heart rate of 130–180 bpm, an ectopic *P* wave with varying coupling intervals, namely multifocal atrial tachycardia. In 2019, cardiac magnetic resonance imaging (MRI) revealed a left ventricular ejection fraction (LVEF) of 53%, with chamber dimensions within age-related norms. Following COVID-19 in 2020 and 2021, her condition deteriorated, with increased episodes of palpitations and irregular heartbeats. Her treatment included propafenone in combination with the β-blocker metoprolol. Before hospitalization, she had been taking ethacizine.

Upon hospital admission, the patient was in fair condition. Auscultation revealed short bursts of tachycardia with a heart rate of approximately 120 bpm. Blood pressure (BP) was 90/60 mm Hg. The initial electrocardiogram (ECG) showed rhythm and conduction disorders, including premature supraventricular contractions (PSVCs), first-degree AV block, left anterior fascicular block, and intraventricular conduction delay (Fig. 1). Transthoracic echocardiography (TTE) performed on January 12, 2024, showed LVEF of 56% and the presence of left ventricular false tendon. Considering the clinical presentation and instrumental findings, the patient underwent an endocardial electrophysiological study (EPS) and radiofrequency ablation (RFA) of the arrhythmogenic substrate, which was located in the anterior wall of the left atrium (LA) near the mitral annulus and the anterior portion of the interatrial septum at the transition to the anterior LA wall. A repeat cardiac MRI showed a slight decrease in LVEF to 51%.

Based on the patient's medical history, clinical presentation, findings from the standard 12-lead ECG and 24-hour ECG monitoring, as well as the absence of structural abnormalities on TTE and cardiac MRI, the final clinical diagnosis was multifocal atrial tachycardia with first-degree

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Table 1. Evolution of rhythm and conduction disorders during 24-hour electrocardiographic monitoring

Examination date	Identified parameters						
	Supraventricular arrhythmias	Ventricular arrhythmias	SA block	AV block, interatrial block	*RR* max, ms	Pharmacotherapy at the time of examination	Interventions
October 19, 2018	PSVCs—718 (0.3%) SVT—61 (the longest tachycardia—961 beats with HR 120/min with transient AV block)	PVCs—254 (<1%)	–	Transient first-degree AV block at night	1720	–	EPS (2018)
January 12, 2022	PSVCs—2193 (4%) SVT—258 (the longest tachycardia—86 beats with HR 121/min)	PVCs—27 (<1%)	–	Persistent first-degree AV block throughout monitoring	1960	–	–
September 21, 2023	PSVCs—2060 (3.73%) SVT—308 (the longest tachycardia—61 beats with HR 125/min)	PVCs—148 (0.15%) VT—2 (the longest tachycardia—11 beats)	–	Persistent first-degree AV block throughout monitoring	2096	–	–
January 17, 2024	PSVCs—45 (0.1%) SVT—1 (7 beats with HR 105/min)	PVCs—172 (0.2%)	–	First-degree AV block, left anterior fascicular block	1700	Ethacizine	EPS, RFA of atrial ectopy (January 15, 2024)
March 23, 2024	PSVCs—1380 (2%) SVT—9 (the longest achycardia—7 beats with HR 105/min)	PVCs—258 (<1%) VT—1 (3 beats with HR 161/min)	Wandering atrial pacemaker within the sinoatrial node—atria	First-degree AV block, intraventricular conduction delay	1692	Metoprolol, Amiodarone	–
August 19, 2024	PSVCs—3.719 (4%) SVT—20 (the longest tachycardia—21 beats with HR 105/min)	PVCs—833 (1%) VT—4 (the longest tachycardia—6 beats with HR 126/min)	Wandering atrial pacemaker within the sinoatrial node—atria; sinus bradycardia; slow idioventricular escape rhythm	First-degree AV block, intraventricular conduction delay	1976	Metoprolol	–
September 16, 2024 (48-hour ECG monitoring)	PSVCs—1.118 (0.6%) SVT—18 (the longest tachycardia—172 beats with HR 113/min)	PVCs—1.121 (0.6%) VT—2 (the longest tachycardia—7 beats with HR 60/min)	–	First-degree AV block, left bundle branch block	2200	Metoprolol	EPS (September 17, 2024): HV Interval 86 ms. Dual-chamber pacemaker implantation (September 17, 2024)

Note: AV, atrioventricular; EPS, electrophysiological study; HR, heart rate; HV, His-ventricle interval; PSVCs, premature supraventricular complexes; PVCs, premature ventricular complexes; RFA, radiofrequency ablation; SA, sinoatrial; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

AV block. The patient was discharged for outpatient follow-up with a recommendation to take bisoprolol 1.25 mg in the morning under HR and BP monitoring, with a follow-up 24-hour ECG monitoring one month after RFA.

One month after RFA, the patient reported recurrent episodes of palpitations and frequent irregular heartbeats. In March 2024, 24-hour ECG monitoring recorded supraventricular arrhythmias (2%), as well as wandering atrial pacemaker, first-degree AV block, and intraventricular conduction delay throughout the monitoring period, during ongoing therapy with metoprolol and amiodarone. In June 2024, 24-hour ECG monitoring continued to show supraventricular (<1%) and ventricular (<1%) arrhythmias, along with episodes of wandering atrial pacemaker within the sinoatrial node and atria, sinus bradycardia, and second-degree sinoatrial (SA) block type I, with a longest recorded *R-R* interval of 1916 ms, and first-degree AV block. In August 2024, repeat 24-hour ECG monitoring performed under ongoing metoprolol therapy revealed episodes of idioventricular rhythm (Fig. 2) and nonsustained paroxysms of ventricular tachycardia (Fig. 3). Genetic testing identified a c.241T>C (rs1553261977) nucleotide sequence variant, resulting in a tyrosine-to-histidine substitution at codon 81 of the *LMNA* protein sequence (p.Tyr81His). This variant has been previously reported in three patients from two unrelated families with diseases associated with *LMNA* gene mutations [2]. Additionally, it was observed in a patient with cardiomyopathy and skeletal muscle weakness, whose parents were not carriers of the p.Tyr81His variant, suggesting a *de novo* mutation. The variant is absent from population databases, and the tyrosine residue is highly conserved. Based on the genotyping results, the diagnosis was revised to cardiac laminopathy with arrhythmia and conduction abnormalities. Direct Sanger sequencing of the patient's clinically unaffected mother did not reveal the p.Tyr81His variant in the *LMNA* gene.

Considering the clinical presentation, laboratory and instrumental examination results, the patient was readmitted to the cardiology department in September 2024 for an EPS and further management planning. TTE revealed LVEF of 56%, hypokinesis of the basal anteroseptal and basal septal segments of the LV, and no chamber dilation. Two-dimensional strain

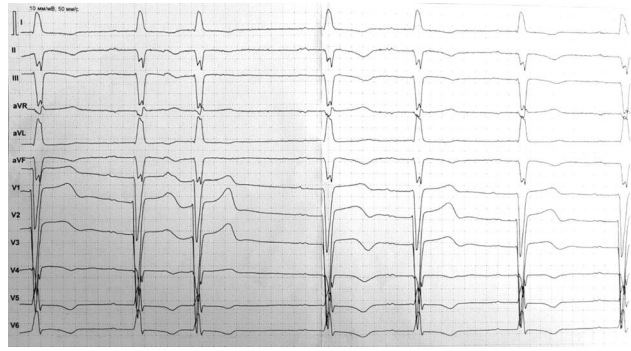

Fig. 1. Standard 12-lead electrocardiogram of patient K. at baseline. Sinus rhythm with first-degree atrioventricular block (*PR* interval, 240 ms) and left anterior fascicular block (*QRS* duration, 110 ms).

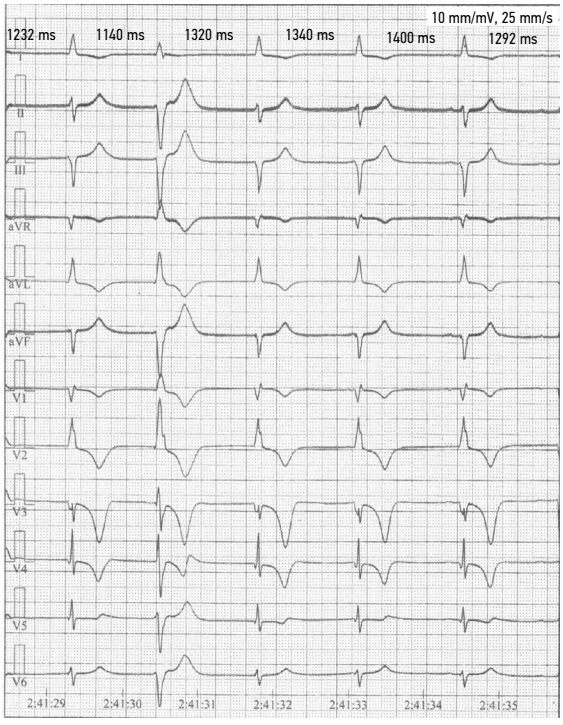

Fig. 2. A fragment of 24-hour electrocardiographic monitoring of patient K. An episode of left ventricular fascicular escape rhythm with atrioventricular dissociation

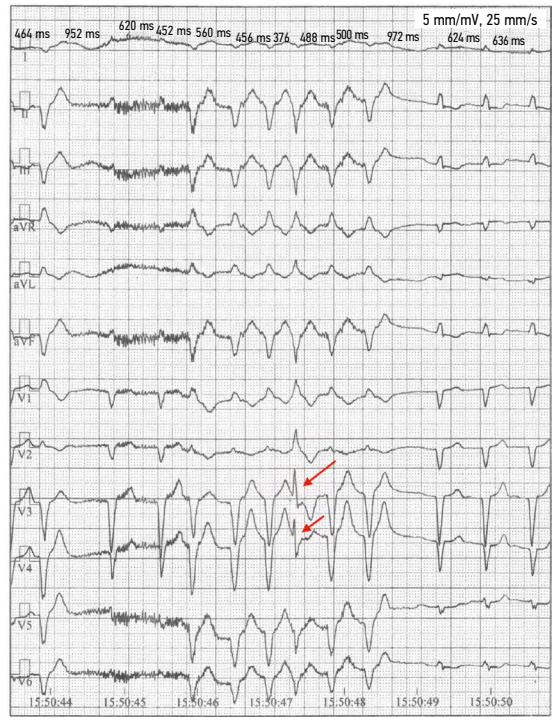

Fig. 3. A fragment of 24-hour electrocardiographic monitoring of patient K. A paroxysm of nonsustained ventricular tachycardia (fusion complexes marked with red arrows) with transformation into supraventricular tachycardia

echocardiography (2D-strain echocardiography) showed no reduction in global longitudinal strain (GLS = –20.0%) (Fig. 4). Nonetheless, further EPS demonstrated a prolonged *HV* interval of 86 ms (Fig. 5).

According to the 2021 European Society of Cardiology guidelines on cardiac pacing and cardiac resynchronization therapy, patients with unexplained syncope and bifascicular block are indicated for pacemaker implantation if the baseline *HV* interval exceeds 70 ms during incremental atrial pacing or pharmacological provocation (Class I recommendation, Level of Evidence B) [10]. Based on the EPS findings, the decision was made to implant a dual-chamber pacemaker.

After pacemaker implantation, the patient was discharged for outpatient follow-up in fair condition with a recommendation to continue metoprolol succinate 50 mg in the morning.

CASE DESCRIPTION 2

A 41-year-old woman (patient P.) was admitted to the cardiac surgery department with complaints of weakness, malaise, and anxiety. According to the patient, in 2013, at the age of 32, she experienced dizziness, and an examination revealed conduction disorders—second-degree AV block and

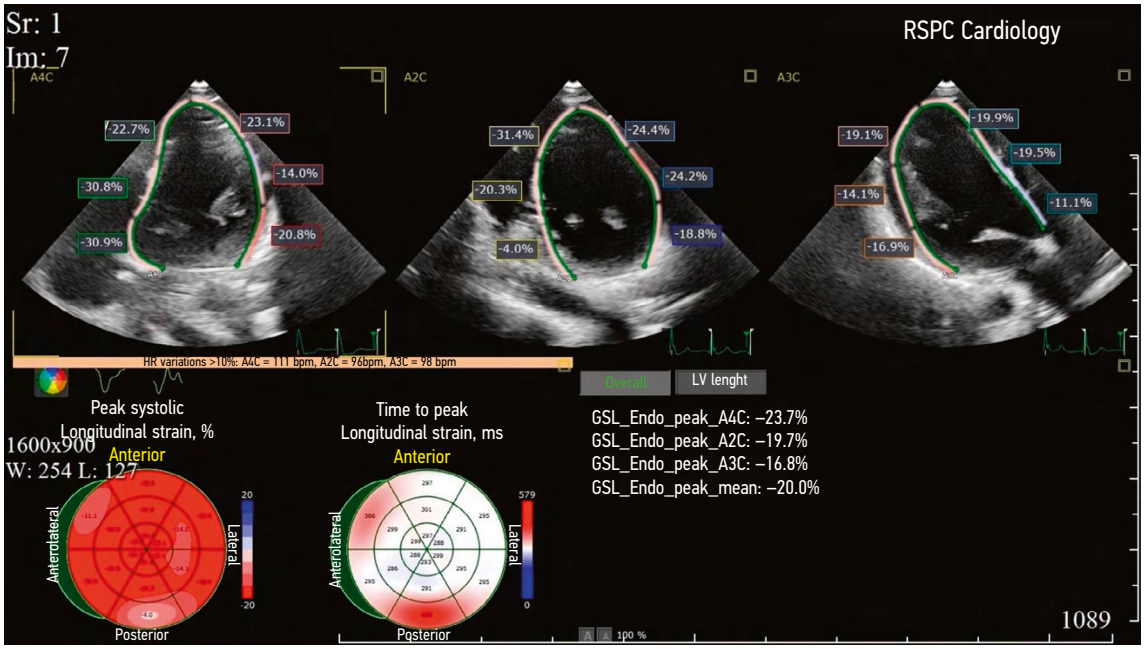

Fig. 4. Two-dimensional strain echocardiography of patient K. Left ventricular global longitudinal strain (–20.0%).

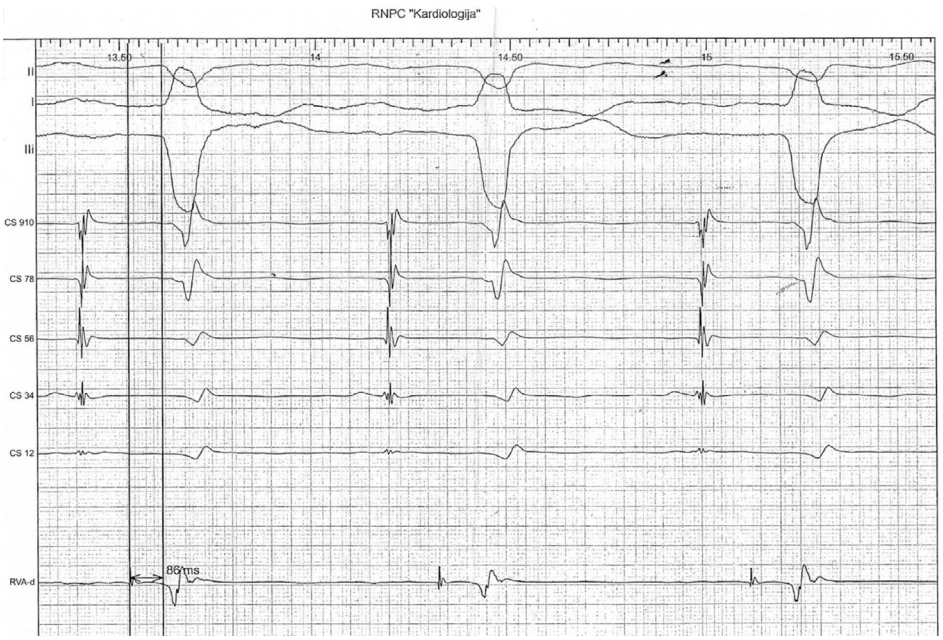

Fig. 5. Endocardial electrophysiological study of patient K. *HV* interval prolongation (86 ms).

second-degree SA block. That same year, a pacemaker was implanted. In 2016, pacemaker lead revision was performed due to lead displacement. In November 2021, following COVID-19, the patient experienced an episode of VT/VF, which was terminated by electrical cardioversion. TTE showed no valvular abnormalities, pacemaker leads were detected in the right heart chambers, and LVEF was 69%, indicating preserved left ventricular systolic function. ECG revealed sinus rhythm with a HR of 72 bpm, inverted *T* waves in leads *V1–V4*, and flattened *T* waves in leads *V5–V6* (Fig. 6). Coronary angiography demonstrated intact coronary arteries. An EPS was performed: during programmed stimulation of the right ventricular apex with three extrastimuli without adrenaline (St1-St2-St3-500-240-150-240), an induced sustained VF episode occurred (Fig. 7), which was terminated by 150 J electrical cardioversion. Based on the EPS findings, the decision was made to replace the pacemaker with an ICD.

A genetic study identified a novel nucleotide sequence variant, c.1409A > C, in exon 11 of the *LMNA* gene, resulting in a lysine-to-threonine substitution at position 470 of the protein sequence (p.Lys470Thr). This amino acid position is highly conserved among vertebrate species. *In silico* analysis predicts that this alteration causes deleterious changes to the protein. Additionally, likely pathogenic missense mutations

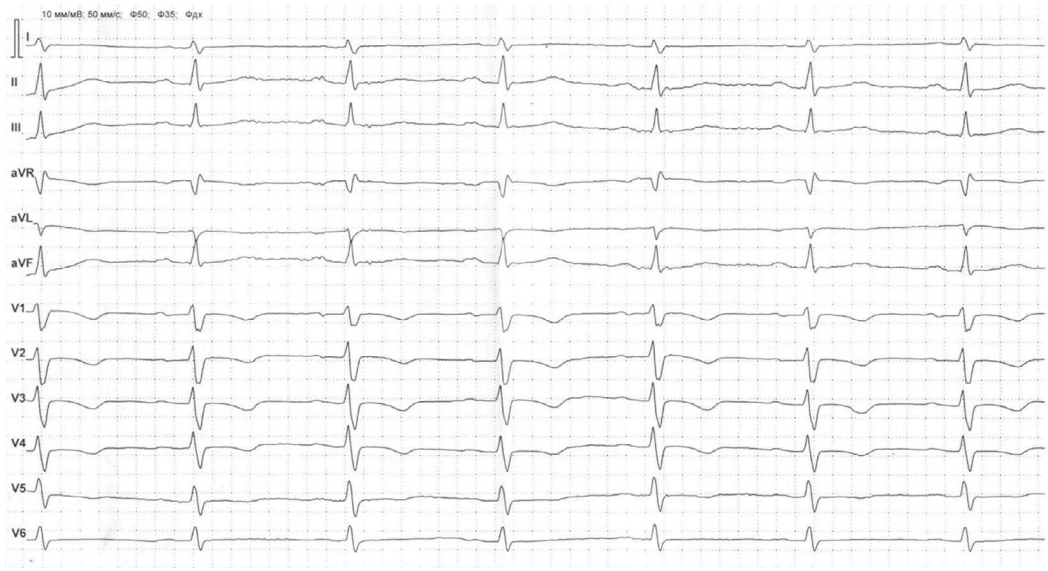

Fig. 6. Twelve-lead electrocardiogram of patient's P. showing *T*-wave inversion in leads *V1–V4.*

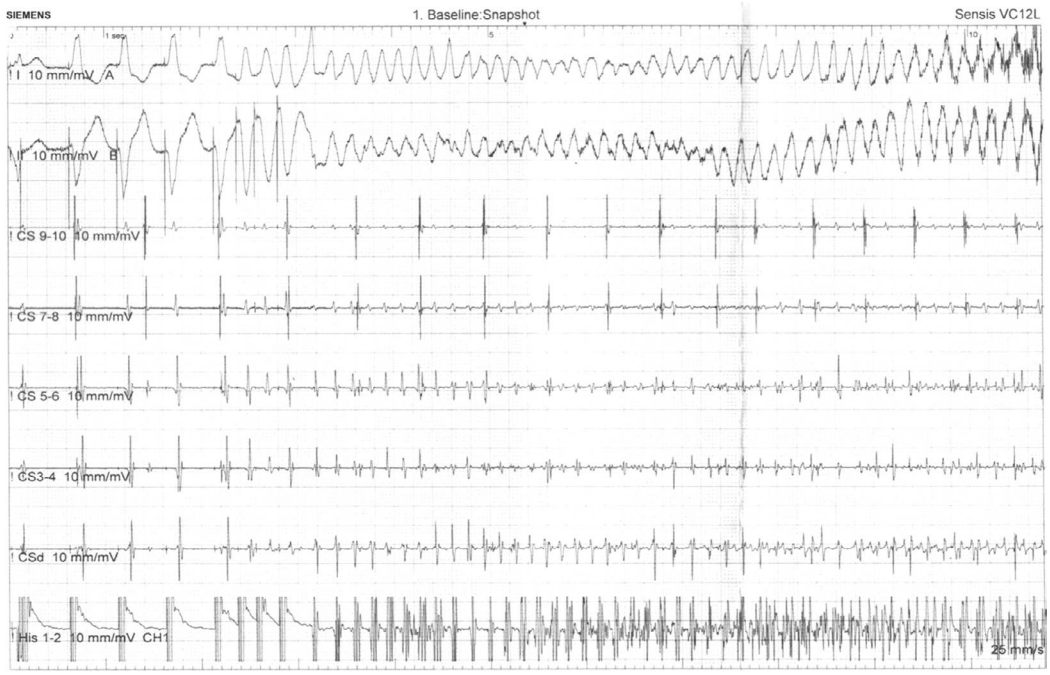

Fig. 7. Endocardial electrophysiological study of patient P. demonstrating sustained ventricular fibrillation.

have been reported in adjacent positions (p.Arg471His, p.Arg471Gly, p.Trp467Arg, etc.), further supporting the functional significance of this region. Based on the preponderance of currently available evidence, this variant is classified as likely pathogenic.

DISCUSSION

In these clinical cases, we have provided a detailed description of the spectrum and evolution of arrhythmic events in carriers of *LMNA* gene mutations who had no structural abnormalities detected by cardiac imaging. Notably, atrial arrhythmias and conduction disorders are frequently observed in laminopathy and may contribute to the progression of atriopathy in affected patients. Therefore, the mechanism underlying *LMNA* mutation–associated atriopathy requires further investigation. Herein, we confirmed the predisposition of *LMNA* mutation carriers to malignant ventricular tachyarrhythmias [11]. In Patient P., sustained VT with transformation into VF developed after an 8-year follow-up period, leading to the implantation of an ICD.

A study by Kumar et al. [2] analyzed the clinical features observed in lamin mutation carriers. The prevalence of clinical manifestations significantly increased from the initial assessment to the median follow-up (7 years): AV block from 46% to 57%, atrial arrhythmias from 39% to 63%, ventricular arrhythmias from 16% to 34%, and LV systolic dysfunction from 44% to 57%. ICDs were implanted in 59% of patients with LV systolic dysfunction or AV block. End-stage HF developed in 19% of patients, and the mortality rate was 13%. Among patients without systolic dysfunction at baseline, LV systolic dysfunction subsequently developed in 24% of cases, and end-stage HF occurred in 7%.

In 2022, the EHRA/HRS/APRS/LAHRS guidelines on the genetic diagnosis of cardiomyopathies and channelopathies were updated [12], incorporating a dedicated section on the diagnostic evaluation of laminopathies. European and American experts agree on the concept of identifying lamin phenotypes as an indication for mandatory molecular genetic testing, given that *LMNA* mutations are associated with poor prognosis.

In the updated 2022 guidelines on the management of patients with ventricular arrhythmias and SCD prevention, as well as the 2023 guidelines on cardiomyopathy management, SCD risk stratification was refined in the subgroup of patients with *LMNA* mutations [13, 14]. The association of *LMNA* mutations with early atrial and ventricular arrhythmias, conduction abnormalities, and a high risk of SCD was demonstrated. A multicenter study involving 269 *LMNA* mutation carriers identified independent risk factors for life-threatening arrhythmias, including nonsustained VT, LVEF ⩽ 45%, male sex, and non-missense mutations [9]. Another study involving 589 *LMNA* mutation carriers identified AV block as an additional risk factor. Subsequently, a *risk calculator* was developed to estimate the probability of life-threatening ventricular arrhythmias[1] [4]. For primary SCD prevention, ICD placement is recommended for patients with a predicted five-year risk of ⩾ 10% and a cardiac phenotype (e.g., nonsustained VT, LVEF < 50%, or AV conduction delay) to prevent unnecessary ICD implantation in mutation carriers without cardiac involvement.

Thus, the findings underscore the importance of molecular genetic testing using high-throughput sequencing of genes associated with inherited arrhythmias, including *LMNA*, in patients presenting with syncope, conduction abnormalities (AV block, sinus node dysfunction), and supraventricular or ventricular tachyarrhythmias, particularly with a family history even in the absence of structural myocardial abnormalities. This approach is crucial for confirming cardiac laminopathy and guiding optimal treatment strategies.

CONCLUSION

Supraventricular and ventricular tachyarrhythmias, along with conduction abnormalities, may represent early manifestations of cardiac laminopathy in *LMNA* mutation carriers, preceding structural myocardial alterations, systolic dysfunction, and the development of a dilated phenotype. Comprehensive medical vigilance and timely genetic testing are essential for confirming the diagnosis and optimizing treatment strategies.

ADDITIONAL INFORMATION

Authors' contribution. S.M. Komissarova, concept and design of the article, writing draft, patient follow-up; N.M. Rineiska, data curation, diagnostic studies, writing draft, review and editing, literature review; N.N. Chakova, conducting and interpreting the results of genetic analysis, writing draft; A.Yu. Dubovik, writing draft, patients' curation; S.S. Niyazova, conducting and interpreting the results of the genetic analysis; T.V. Sevruk, conducting and interpreting the results of echocardiography. 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).

Consent for publication. The authors have received written informed voluntary consent from the patient's legal representatives to publish personal data.

Competing interests. The authors declare that they have no conflict of interest.

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[1] *LMNA-risk VTA calculator: Risk Prediction Score for Life-Threatening Ventricular Tachyarrhythmias in Laminopathies* [Internet]. Available from: https://lmna-risk-vta.fr/. Accessed March 25, 2025.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. С.М. Комиссарова — концепция и дизайн статьи, написание текста, динамическое наблюдение за пациентами; Н.М. Ринейская — анализ полученных данных, диагностические исследования, написание текста, обзор литературы; Н.Н. Чакова — проведение и интерпретация результатов генетического анализа пациентов, написание текста; А.Ю. Дубовик — написание текста, курация пациентов; С.С. Ниязова — проведение и интерпретация результатов генетического анализа пациентов; Т.В. Севрук — проведение и интерпретация эхокардиографии. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

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Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при написании статьи.

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