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An arrhythmic variant of the manifestation of paraneoplastic Loeffler endomyocarditis. Clinical case

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ABSTRACT

A clinical case of chronic undulating course of parneoplastic Loeffler endomyocarditis, the leading manifestations of which were ventricular arrhythmias, is presented. The paper demonstrates the complexity of early diagnosis of a rare pathology in a polymorbid patient and attempts to identify the "keys" to the correct diagnostic and therapeutic tactics for managing such patients.

Keywords: hypereosinophilic syndrome; hypereosinophilia; reciprocal ventricular tachycardia; Loeffler endocarditis; eosinophilic myocarditis.

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Аритмический вариант манифестации паранеопластического эндомиокардита Леффлера. Клинический случай

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АННОТАЦИЯ

Представлен клинический случай хронического волнообразного течения парнеопластического эндомиокардита Леффлера, ведущими проявлениями которого стали желудочковые нарушения ритма. В работе демонстрируется сложность ранней диагностики редкой патологии у полиморбидного пациента и предпринимается попытка определить «ключи» к верной диагностической и лечебной тактике ведения подобных пациентов.

Ключевые слова: гиперэозинофильный синдром; гиперэозинофилия; реципрокная желудочковая тахикардия; эндокардит Леффлера; эозинофильный миокардит.

Как цитировать

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INTRODUCTION

Loeffler endomyocarditis (LEM) is a cardiac manifestation of hypereosinophilic syndrome (HES), wherein hypereosinophilia (eosinophil count >1500/μL) [1] and target-organ damage are obligatory components, caused by the degranulation of large numbers of eosinophils with the release of significant amounts of cytokines (i.e., interleukin (IL)-3, IL-5, and granulocyte-macrophage colony-stimulating factor) into organs and tissues [2–5].

The presence of characteristic echocardiographic signs allows the accurate diagnosis of LEM at late stages[6,7]; however, the course of the disease at these stages is typically irreversible. Early diagnosis is challenging owing to the absence of overt and unambiguous symptoms. Nevertheless, early diagnosis and prompt intervention can prevent irreversible structural changes. We present a case of confirmed LEM wherein the initial clinical manifestation was recurrent ventricular rhythm disturbances.

CLINICAL CASE

A 60-year-old man was first admitted to St. Petersburg City Pokrovskaya Hospital in January 2023 with a ventricular tachycardia (VT) paroxysm. The ambulance team attempted to medically control the VT; however, this was ineffective, as were three subsequent defibrillator discharges, after which the patient was admitted to hospital. In the hospital, VT persisted after intravenous administration of 300 mg of amiodarone and was treated by electrical cardioversion (ECV). The electrocardiography (ECG) record of the first VT was lost; however, its description was preserved in the documents: the frequency of the VT was 176 beats per minute (bpm), QRS complex had the form of complete block of the right bundle branch (RBBB) and left anterior fascicular branch (LAFB) (left VT), and QRS width was 150 ms.

The patient's medical history showed that the patient had suffered a non-ST elevation myocardial infarction in 2008 and underwent an anterior interventricular artery (AIVA) stenting the same year and a coronary artery bypass graft (CABG) in 2012. No ventricular rhythm disturbances were recorded until January 2023. At the first hospitalization in January 2023, VT occurrence was attributed to coronary heart disease (CHD) and post-infarction cardiosclerosis. The patient refused further evaluation and treatment and was discharged at his own request on hospitalization day 2.

Additionally, the patient had a history of peripheral carcinoma of the right lung, for which a right lung lobectomy was performed in 2014. In 2021, the patient underwent radiation therapy for carcinoma of the left lung. In December 2022, metastases to the pleura, lymph nodes, and mediastinum were detected. Since 2016, the patient has been under observation for chronic lymphocytic leukemia.

At the time of discharge, an echocardiographic study (EchoCG) was conducted, and no peculiarities were identified. The laboratory data available at that time are presented in Tables 1 and 2.

Table 1. Laboratory test results from January to November 2023

Indicators	Data				Reference values
	January 10, 2023	January 11, 2023	October 17, 2023	November 05, 2023	
Hemoglobin, g/ L	91	115	103	95	130–160
Hematocrit, %	27	33.9	38.4	29.8	40–48
Erythrocytes, 10^{12}/L	3.13	4	3.56	3.32	4–5.6
Leukocytes,10^{9}/L	65.1	84.2	71.7	173.12	4–9
Lymphocytes,10^{9}/L	51.7	65.1	40	–	1.2–3
Segmented neutrophils, %	20	4	29	38.3	47–72
Eosinophils, %	5	3	29	15	0.5–5
Platelets, 10^{9}/L	161	205	160	73	180–320
ESR, mm/h	16	59	55	40	1–10

Table 2. Dynamics of high-sensitivity troponin level in blood samples

	January 10, 2023 01 hour and 51 minutes	January 10, 2023 11 hours and 13 minutes	October 17, 2023	October 18, 2023	October 18, 2023	October 31, 2023	November 11, 2023
Troponin (I) (N 0-34 ng/L)	5.7	26.8	983.1	1259.3	1510.3	1501.9	438.2

In April 2023, the patient underwent chemotherapy for carcinoma metastases using vinorelbine at 60 mg/m^2 on days 1, 8, and 21. Thereafter, the patient had no symptoms until September 2023.

In October 2023, the patient was readmitted to Saint Petersburg City Pokrovskaya Hospital because of an atypical pain in the precordial region and frequent VT paroxysms, occurring 3–4 times per day. On October 17, 2023, ECG exhibited alterations identical to those observed in the January 2023 (Fig. 1).

Considering the elevated high-sensitivity troponin levels upon admission and tendency for these levels to remain elevated, a series of diagnostic procedures were conducted on hospitalization day 1, including coronary angiography, coronary shuntography, and ventriculography.

Coronary angiography findings:

- Right type of coronary blood supply.
- Left coronary artery without stenosis.
- AIVA: condition after stenting from the orifice (2012), chronic occlusion in the stent. Periphery: filled from the CABG and right coronary artery (RCA) collaterals.
- Diagonal artery: filled by a functioning CABG and retrogradely by collaterals of the circumflex artery (CA).
- CA: main branch without stenosis.
- Marginal artery: eccentric stenosis in the proximal third not exceeding 60%.
- RCA: moderately changed in proximal and middle third; stenosis not more than 60%.

Coronary shuntography

The CABG to AIVA function was satisfactory, with no anastomosis defects. In the case of chronic occlusion of AIVA immediately after anastomosis, the shunt functions on the septal and diagonal branches that originate from more proximal segments of the artery. Additionally, the apical segment of AIVA is filled retrogradely from the RCA pool.

Ventriculography showed no focal contractility abnormalities in the left ventricle (LV) and ejection fraction > 55%.

Subsequently, high troponin levels persisted throughout the hospitalization (Table 2).

Since hospitalization day 1, recurrent episodes of VT accompanied by a decline in blood pressure were observed. The ventricular nature of the arrhythmia was unquestionable. VT was diagnosed using the criteria by Vereckei A. et al. [8]: a QRS complex type R in the aVR lead and a Vi/Vt ratio ⩽1 (Vi, rate of voltage change during the first 40 ms of the QRS complex; Vt, rate of voltage change during the last 40 ms of the QRS complex).

The initial episodes of VT were brief and self-limiting, resolving spontaneously. However, they eventually became longer in duration and necessitated the administration of ECV.

Two distinct types of VT were identified during the course of the patient's hospital stay. The morphology of the complexes in both cases was practically identical, and the shape of the QRS complexes fully coincided with the description of VT from January 2023. Specifically, the shape was that of

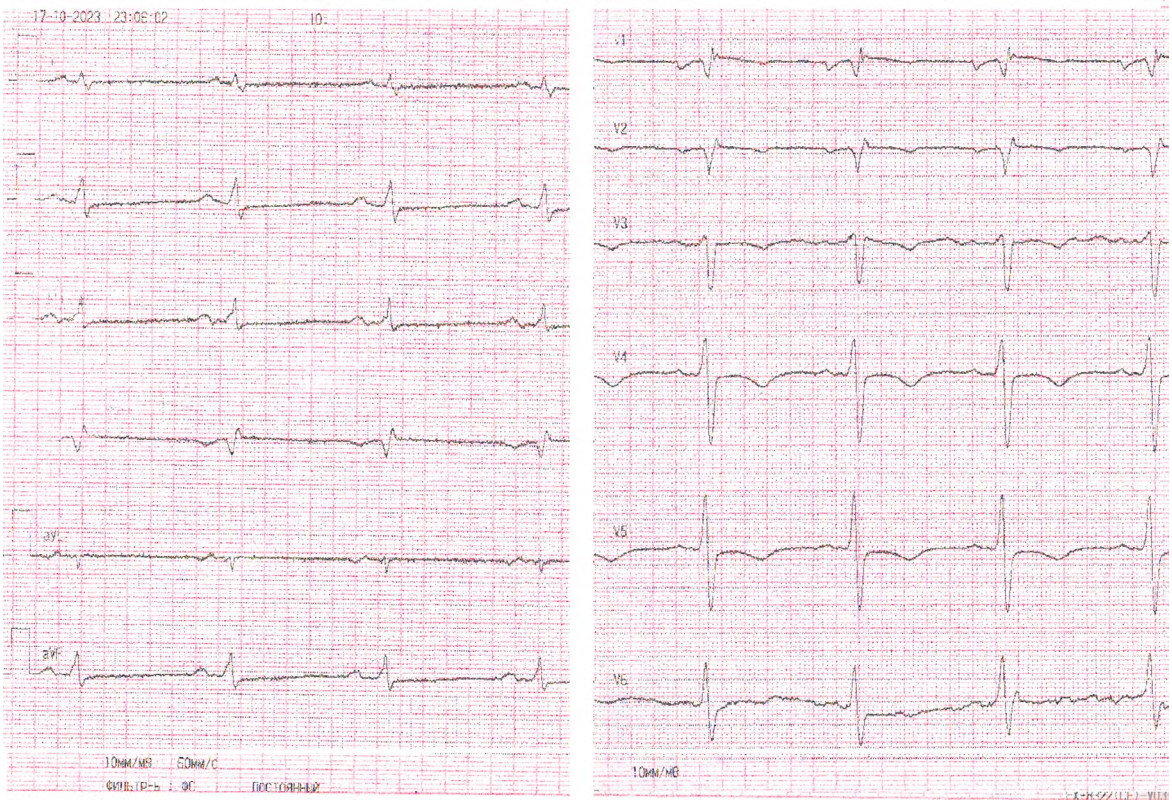


Fig. 1. ECG on October 17, 2023. Sinus rhythm with a rate of 88 per minute. Cardiac rotation of the right ventricle anteriorly and the apex posteriorly. Left atrium enlargement. Disseminated diffuse myocardial changes

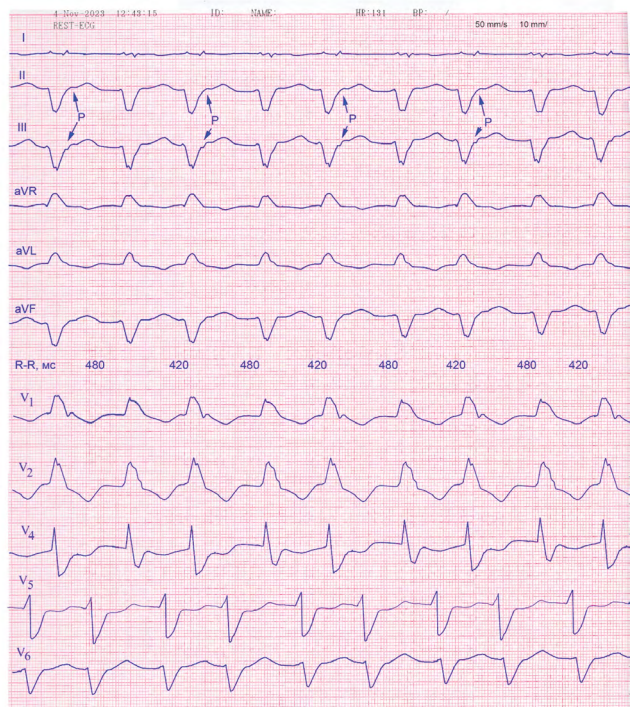


Fig. 2. ECG on November 4, 2023. Monomorphic reciprocal left ventricular tachycardia with a rate of 131 per minute. *QRS* complex is 150 ms and has the shape of a complete RBBB and LAFB, *R*-shape in a*VR* lead, and Vi/Vt ratio < 1 in V5 lead. The regular fluctuations of the *R-R* intervals can be explained by conduction through reentry loops of different sizes

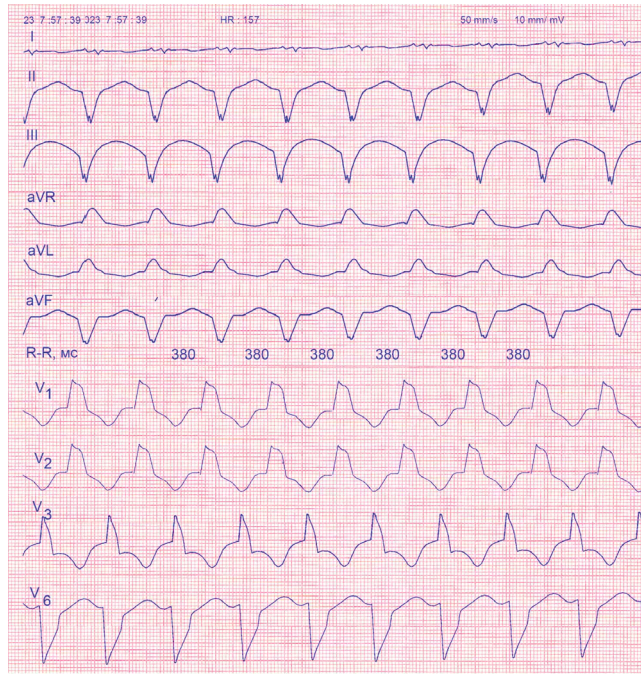


Fig. 3. ECG no. 2, November 4, 2023. Monomorphic reciprocal left ventricular tachycardia with a rate of 157 per minute. *QRS* complex is 150 ms, in the form of a complete RBBB and LAFB, *R*-shape in the a*VR* lead, and *r/S* ratio in the V6 lead < 1

a complete RBBB and LAFB with a QRS width of 150 ms. When analyzing the ECG of type 1 VT, with a frequency of 131 bpm (Fig. 2), attention was drawn to the strictly regular alternation of two identical RR intervals (420 and 480 ms) and negative oscillations in the lower leads after the short R-R interval. The first impression of duplicated VT seemed unlikely, as this variant could involve two independent ventricular sources of automaticity operating simultaneously, both at excessively low and similar frequencies (~60 and 75 bpm). The presence of reciprocal LV VT was more probable, with

two channels of impulse propagation, similar to the figure of eight configuration, in which the conduction time through one of the channels is longer than that through the other channel, with retrograde conduction of the impulses to the atria in a ratio of 2:1.

Moreover, type 2 VT recorded on the same day was strictly regular, with a frequency of 157 bpm and similar characteristics of the QRS complexes of type 1 VT, but without regular fluctuations of the *R-T* intervals (which was considered to be propagation of the impulse along a single reentry loop). Additionally, no *P*-wave could be detected.

In the intervals between hemodynamically significant VT paroxysms, frequent ventricular extrasystoles were recorded, the morphology of which was similar to the QRS complexes in the VT circuit (Fig. 4).

EchoCG showed hypercontractility of the basal and mid-LV segments associated with local akinesia of the apex (Merlon's sign) and significant wall masses,

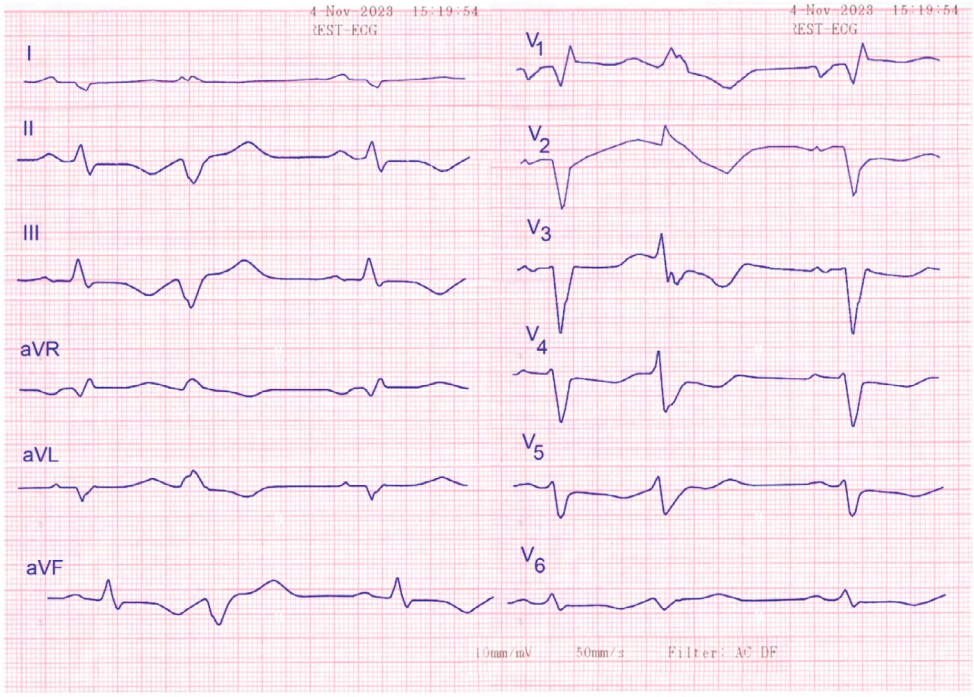


Fig 4. Sinus tachycardia ~100 per minute. Left ventricular extrasystole with complete compensatory pause, having the form of a complete RBBB and LAFB, similar to the form of *QRS* complexes in tachycardia. Overload of the left atrium

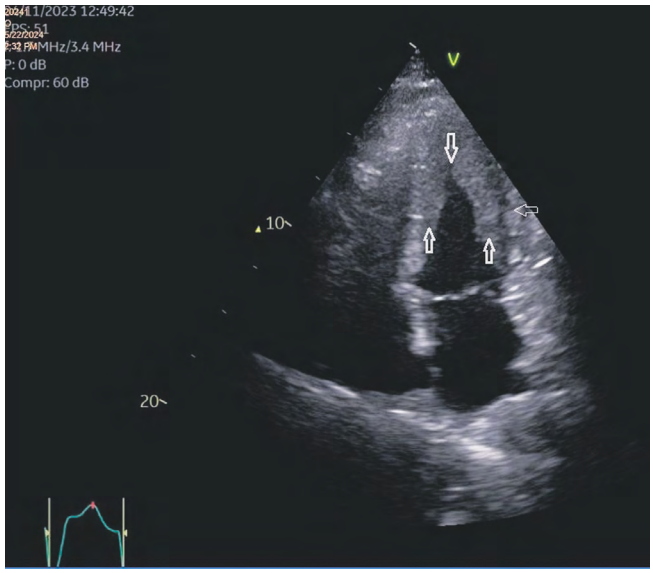


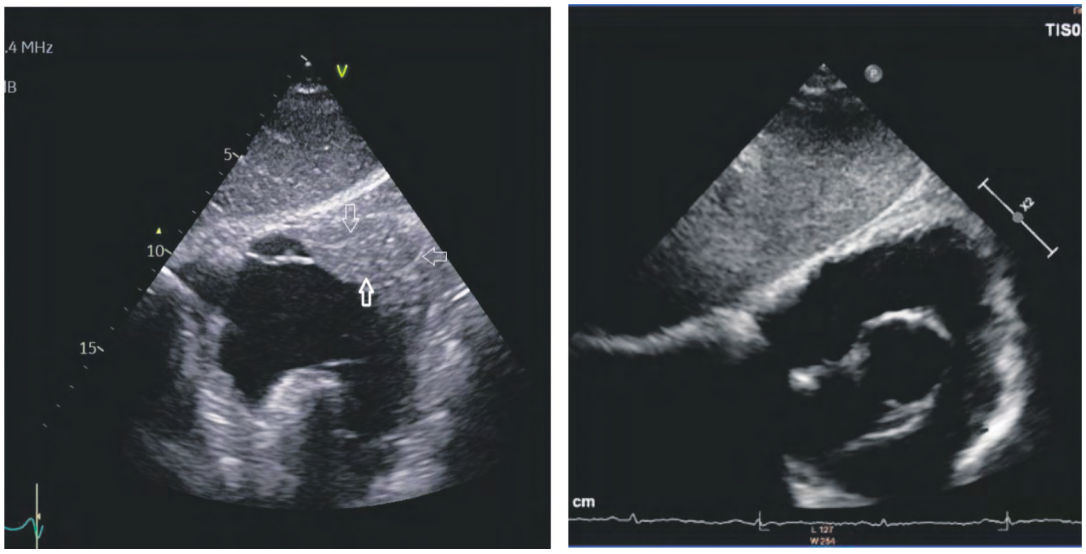
Fig. 5. Echocardiogram of Loeffler endomyocarditis of the LV. Four-chamber view, apical approach. The arrows indicate wall masses in the area of the akinetic apex and in the projection of myocardium with preserved local contractility. The border between the myocardium and myocardial projections is clearly visible. The wall masses and myocardium have different densities, and there is an obvious boundary between them. Vertical arrows indicate extensive wall masses initially believed to be thrombus; horizontal arrow indicates LV myocardium

which were initially believed to be extensive thrombotic deposits. These masses were localized in the area of the fixed apex and in the protrusion of myocardium with preserved contractility (Fig. 5). A similar condition was observed in the region of the outflow tract and the apex of the right ventricle (Fig. 6). LV systolic function was preserved. No echocardiographic evidence of severe diastolic dysfunction was noted.

Based on the echocardiographic data, Loeffler endomyocarditis was diagnosed. In the hospital, the patient received anti-inflammatory therapy with glucocorticosteroids (in doses not exceeding the dose of prednisolone of 1.0 mg/kg intravenously) and antiarrhythmic therapy with amiodarone; however, the disease progressed. On hospitalization day 22, another VT paroxysm developed into ventricular fibrillation and then to asystole. Resuscitation was unsuccessful.

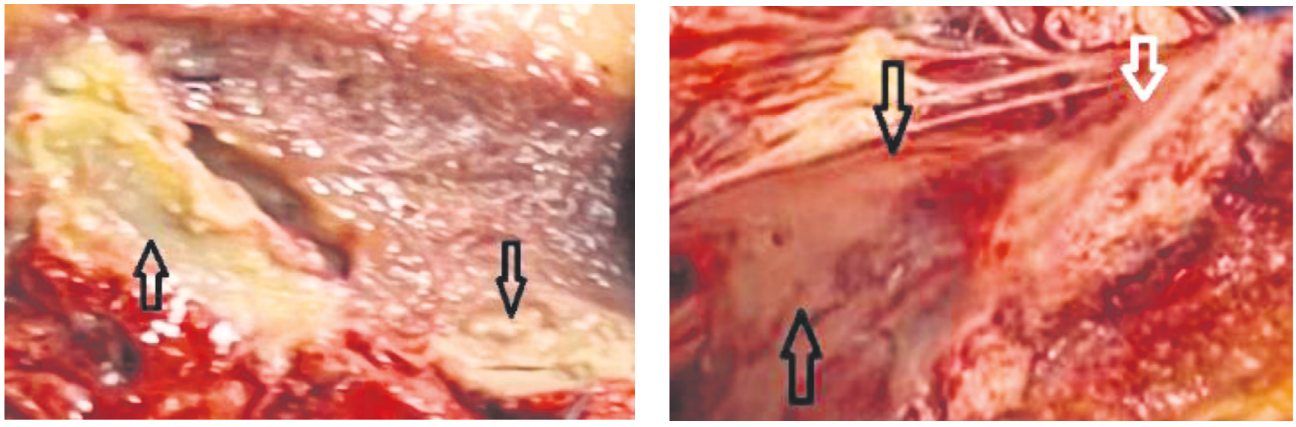
Pathologic examination confirmed the diagnosis of Loeffler endomyocarditis. Macroscopically, large areas of inflammation and marked thickening of the endocardium of the left and right ventricles, with evidence of inflammation extending into the myocardium, were found. Two adjacent foci of muscle necrosis were noted in the apical part of the LV (Fig. 7).

Clinically, no mural thrombus was found on pathologic–anatomic examination. What was believed to be a thrombotic mass in the wall was actually an inflamed, friable, and significantly thickened (edematous) endocardium.



a *b*

Fig. 6. Echocardiographic changes in the right ventricular outflow tract: *a* — short-axis view at the level of the aortic valve, subcostal approach, reveals parietal masses located in the outflow tract of the right ventricle, indicated by the lower arrow. The arrows above and to the right delineate the myocardium of the right ventricle and a clear boundary between the myocardium and the parietal deposits. For comparison; panel *b* — displays the same section from a healthy individual,demonstrating a non-thickened right ventricular myocardium absent of pathological parietal masses



a *b*

Fig. 7. Macroscopic sections in the LV apex: *a* — two adjacent foci of necrosis of pale yellow color (black arrows); *b* — thickened, inflamed endocardium of pale pink color. Intact endocardial areas are marked with black arrows and damaged areas with white arrows

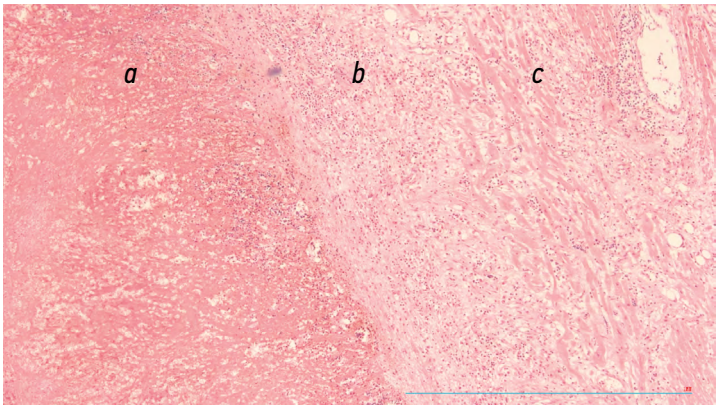


Fig. 8. Histological section of the endocardium and adjacent myocardium: *a* — endocardium; *b* — focus of necrosis; *c* — myocardium. At the border of the endocardium and myocardium, signs of necrosis of both endocardium and adjacent myocardium are noted. In the zone of endocardial necrosis, the presence of necrotic tissue, fibrin, and hemolyzed blood elements is observed. In the adjacent myocardium, similar changes are found: signs of necrosis, overgrowth of granulation tissue, and massive infiltration of the whole area with lymphocytes, plasmocytes, and eosinophils

Histological examination revealed eosinophilic infiltration of the endocardium and myocardium (Fig. 8), as well as of the liver, spleen, bone marrow, and lungs.

DISCUSSION

The course of Loeffler endomyocarditis is characterized by three distinct stages: the acute necrotic stage, stage of wall thrombus formation, and stage of endomyocardial fibrosis. The acute stage persists for approximately 5-6 weeks and lacks distinctive symptoms, although fever, sweating, and arrhythmias may be present. Manifestations of the disease become evident at a later stage, exhibiting as recurrent thromboembolic events in the second stage and progressive heart failure in the third stage.

From a clinical perspective, it is noteworthy that the initial registered manifestation of the disease in the patient was a VT that occurred 10 months prior to the onset of the principal events. Prior to this, the patient with CHD had not experienced any ventricular arrhythmias, including coronary events. VT occurred concurrently with hypereosinophilia, with an estimated eosinophil count of 3255/μL. However, the percentage of eosinophils remained within the normal range, which was probably the reason for the underestimation of hypereosinophilia. The presence of granulation tissue in histologic sections indicated that there may have been earlier foci of necrosis at this site, which were eventually replaced by fibrous tissue. On January 10, 2023, myocardial damage was demonstrated by a marked increase in troponin levels in less than 10 hours. In practice, the increase in troponin level is often associated with myocardial damage owing to electrical discharge during ECV (especially during multiple ECV). Nevertheless, currently available data do not show a definitive correlation between ECV and troponin elevation. This observation reinforces the need to identify other causes of myocardial damage. Furthermore, had troponin levels been monitored in January 2023, a further rise in troponin levels would probably have been detected. However, this remains an assumption.

In the present case, the natural organic substrate of VT was fibrotic and necrotic myocardial changes that created conditions for reentry. The most remarkable indicator of reciprocal tachycardia is the near-complete uniformity of *R-R* intervals within the tachycardia chain. Both were recorded in our patient and exhibited absolute regularity. Furthermore, in the first type of tachycardia circuit, the R-R intervals exhibit a strict alternation of 420 and 480 ms, which occurs when impulse conduction is carried out by two loops of reentry, rather than a single one. A similar character of reciprocal tachycardia was previously presented by W.G. Stevenson et al. [10].

The presented types of VT represent two hypostases of a single reciprocal tachycardia originating from the high regions of the interventricular septum (IVS), which is clinically referred to as fascicular ventricular tachycardia (FVT) or verapamil-sensitive left VT. The morphology of the complexes was similar to that in FVT. In tachycardia, a complete RBBB is present, accompanied by a leftward deviation of the electrical axis. In contrast, under sinus rhythm, no initial similar changes are evident. This point of view has been explained; however, it ignores clinical and morphological data, particularly the presence of obvious morphological changes in the apex region, which is a suitable substrate for VT, whereas no changes were found in the high IVS region [10].

The presence of two foci of necrosis and two types of tachycardia showed that two different reciprocal tachycardias have developed, despite the similar morphology of the ventricular complexes. The similarity of the QRS complexes can be explained by the fact that, according to the autopsy results, all fibrotic–necrotic changes were compactly localized in the cardiac apex or that the direction

of the vectors of electrical excitation propagation should be similar.

Clearly, our reasoning was speculative; however, assuming that it is correct and the first VT paroxysm was indeed the manifestation of LEM, no characteristic signs of LEM were detected by EchoCG at that time. Additionally, there was still no reliable diagnostically significant troponin elevation. Later, progressive hypereosinophilia developed (in the final stage, the number of eosinophils reached 23000/μL).

Remarkably, the relative stabilization of the condition (from January 2023 to September 2023) coincided with the course of chemotherapy. The main treatment method for reactive HES is effective therapy of the underlying disease [11]. Thus, our patient had no hemodynamically significant arrhythmias for 5 months after chemotherapy, and the fact that the leukocyte count at the beginning of the second hospitalization was slightly lower than in January may indicate the efficacy of the chemotherapy performed. Thus, chemotherapy may have slowed down the development of advanced clinical manifestations of LEM.

The abovementioned indicates that, in the present case, the reactive paraneoplastic LEM had a chronic wave-like character, with periods of exacerbation followed by periods of relative stabilization due to adequate therapy.

Treatment of LEM may vary depending on the type of HES. The three variants of HES should be differentiated when choosing a treatment option:

1. Primary or clonal: myeloproliferative and myelodysplastic conditions in which eosinophils represent part of a neoplastic clone and/or FIP1L1/PDGFRA mutations are present [12, 13, 14].

2. Reactive: hypereosinophilia is formed in response to exogenous stimuli via IL-3, IL-5, etc. (i.e., allergic conditions, parasitic infections, adverse drug reactions, and inflammatory or neoplastic diseases).

3. Idiopathic hypereosinophilia: after exclusion of clonal and reactive HES.

In the primary variant, treatment is based on the administration of tyrosine kinase inhibitors (primarily imatinib), whereas the first-line treatment of reactive hypereosinophilia in the absence of FIP1L1/PDGFRA mutation is glucocorticoid steroids (GCS). Evidently, the primary course of action in reactive HES should be etiologic therapy. This may involve antiparasitic treatment for worm infestations, chemotherapy for neoplasms, or drug withdrawal in cases of drug hypersensitivity. The recommended starting dose of prednisolone is 1.0 mg/kg of body weight when administered orally and 5 mg/kg when administered intravenously. In critical cases, the total dose of methylprednisolone administered over a 3-day period may reach 1,000 mg. In cases of reactive HES, it is not recommended to prolong aggressive GCS therapy for more than 3–6 months. Imatinib-sensitive mutations should be excluded, even in cases wherein reactive HES is a potential diagnosis, as clonal HES has been demonstrated to exhibit resistance to steroid therapy [13, 14].

In idiopathic HES, mepolizumab, a humanized monoclonal antibody (IgG1, kappa) directed against IL-5, is the recommended treatment [13, 14].

In the event of resistance to initial pharmacological agents, alternative treatments may be considered, including immunosuppressive drugs (e.g., imatinib, hydroxyurea, vincristine, chlorambucil, etoposide, and cytarabine), immunomodulators (e.g., peginterferon alfa-2a and interferon alfa-2b), and interleukin inhibitors (e.g., mepolizumab and benralizumab) [13, 14].

CONCLUSIONS

The main diagnostic sign of Loeffler endomyocarditis is hypereosinophilia. A high level of physician vigilance is warranted for the recognition and differential diagnosis of hypereosinophilia and hypereosinophilic syndrome.

Ventricular rhythm disturbances with hypereosinophilia may be an early manifestation of Loeffler endomyocarditis and precede diagnostically significant troponin elevations and appearance of typical echocardiographic and electrocardiographic signs.

The use of adequate doses of GCS should be preceded by the exclusion of imatinib-sensitive mutations.

The course of reactive (in the present case, paraneoplastic) Loeffler endomyocarditis may be wavy, with periods of relative stabilization during effective therapy of the underlying disease, provided that such therapy is initiated at the early stage of endomyocarditis.

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: Yu.N. Grishkin, V.Yu. Zimina — concept, design, materials processing, data analysis, writing, literature review; P.O. Karchikian — collection, processing, analysis of echocardiographic data, literature review; A.A. Babayan — collection and analysis of daily monitoring data, literature review; O.V. Grigorieva — collection, processing, analysis of pathological and histological data; T.B. Butaev — materials processing, data analysis.

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

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

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: Ю.Н. Гришкин, В.Ю. Зимина — концепция, дизайн, обработка материалов, анализ данных, написание текста, обзор литературы; П.О. Карчикьян — сбор, обработка, анализ данных ЭхоКГ, обзор литературы; А.А. Бабаян — сбор и анализ данных суточного мониторирования, обзор литературы; О.В. Григорьева — сбор, обработка, анализ патологоанатомических и гистологических данных; Т.Б. Бутаев — обработка материалов, анализ данных.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили прижизненное согласие пациента на публикацию медицинских данных и всех сопутствующих изображений.

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