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Трудности диагностики миокардита: клинический случай

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АННОТАЦИЯ

Миокардит нередко является трудным для диагностики заболеванием сердца. Сложности его диагностики объясняются неспецифической симптоматикой или «стёртой» клинической картиной, отсутствием патогномоничных признаков при физикальном обследовании, а также тем обстоятельством, что эндомиокардиальная биопсия, являющаяся «золотым стандартом» диагностики, — это инвазивная процедура, которая проводится по строгим показаниям ограниченному количеству пациентов. Тем не менее, по мере развития лучевой диагностики у клиницистов появилась возможность неинвазивной диагностики симптомов воспалительного поражения миокарда, в том числе отёка и миокардиального фиброза, с помощью магнитно-резонансной томографии сердца.

В статье представлен клинический случай пациента молодого возраста с симптомами острого коронарного синдрома, у которого при этом не было выявлено признаков поражения коронарных артерий. С учётом повышения активности кардиоспецифических ферментов и уровней маркёров воспаления, выраженных изменений на электрокардиографии с положительной динамикой, а также факта перенесённой накануне инфекции, был заподозрен миокардит. В результате проведения магнитно-резонансной томографии сердца в динамике диагноз был подтверждён. Таким образом, показана роль визуализирующей методики для дифференциальной диагностики ишемического и воспалительного поражения сердца.

Ключевые слова: миокардит; магнитно-резонансная томография; электрокардиография; тропонин; клинический случай.

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Difficulties in myocarditis diagnosis: a case report

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ABSTRACT

Myocarditis is often difficult to diagnose. The diagnostic difficulties include nonspecific symptoms or a "vague" clinical picture, absence of pathognomonic signs during physical examination, and endomyocardial biopsy, which is the "gold standard" of diagnosis of myocarditis, being an invasive procedure that is performed under strict indications in certain patients. Nevertheless, as radiology is rapidly developing, clinicians are now able to noninvasively diagnose symptoms of inflammatory myocardial damage, including edema and myocardial fibrosis, using cardiac magnetic resonance imaging. This article presents the clinical case of a young patient with symptoms of acute coronary syndrome, who showed no evidence of coronary artery disease. Myocarditis was suspected because of increased activity of cardiospecific enzymes and high levels of inflammatory markers, pronounced electrocardiography changes with positive dynamics, and recent infection. Magnetic resonance imaging was used to confirm myocarditis diagnosis. Thus, this case study demonstrates the role of imaging techniques in the differential diagnosis of ischemic and inflammatory heart diseases.

Keywords: myocarditis; magnetic resonance imaging; electrocardiography; troponin; case report.

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心肌炎的诊断难题：临床病例

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简评

心肌炎通常是一种难以诊断的心脏病。非特异性症状或不明显的临床表现、体格检查中缺乏病理征象都是诊断困难的原因。心内膜活检（诊断的“金标准”）是一种侵入性手术，这也是诊断困难的原因。只有在严格的适应症下，才会对少数患者实施这种手术。不过，随着放射诊断技术的发展，医生现在有机会对炎症性心肌损伤症状（包括水肿和心肌纤维化）进行非侵入性诊断。这种诊断是通过心脏磁共振成像（MRI）进行的。

文章介绍一个年轻患者的临床病例，该患者有急性冠状动脉综合征的症状。但是没有发现冠状动脉损伤的迹象。观察到的是，心脏特异性酶活性和炎症标志物的水平升高，心电图检查出现明显变化并呈正方向变动。此外，还注意到患者以前曾感染过病毒。考虑到这些情况，怀疑是心肌炎。心脏动态磁共振成像结果证实了这一诊断。由此可见，本文强调了成像技术在鉴别诊断缺血性和炎症性心脏病变中的作用。

关键词：心肌炎；磁共振成像；心电图检查；肌钙蛋白；临床病例。

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BACKGROUND

Acute myocarditis is defined as a sudden inflammatory injury to the heart muscles. Worldwide, approximately 4–14 cases per 100,000 are reported annually, and 1%–7% die from the disease [1]. Therefore, timely diagnosis is extremely important for clinicians to establish prognosis and treatment strategies.

Poorly defined symptoms at onset, lack of specific clinical signs of myocarditis, minimal changes on physical examination [2], and infrequent use of endomyocardial biopsy as the "gold standard for diagnosis" lead to the underdiagnosis of this disease in real-world practice [3].

However, criteria for diagnosing myocarditis, such as the presence of a previous infection confirmed by clinical and/or laboratory data and the clinical and diagnostic signs of recurrent heart muscle injury remain relevant [4].

Clinical signs:

- Chest pain
- Arrhythmias of unknown origin
- Signs and symptoms of heart failure
- Syncopal episodes and/or sudden death prevented (successfully resuscitated)
- Cardiogenic shock: absence of coronary artery (CA) injury and other cardiovascular diseases that could cause this condition

Diagnostic criteria:

- Laboratory criteria, with grade of recommendation (GoR) C and level of evidence (LoE) 5:
  - Increased:
    - Troponins T and I
    - Lactate dehydrogenase
    - Creatine kinase-MB (CK-MB)
    - N-terminal pro-B-type natriuretic peptide (NT-proBNP)
    - Serum cardiac autoantibodies
- Imaging criteria:
  - Electrocardiography (ECG), GoR C, and LoE 4: blockades, arrhythmias, and ST-T changes
  - Electrocardiography (ECG), GoR C, and LoE 4: functional and/or structural changes
  - Gadolinium-enhanced magnetic resonance imaging (MRI) of the heart, GoR A, and LoE 2: myocardial edema and/or late contrast enhancement
  - Morphological criteria, GoR C, and LoE 4: endomyocardial biopsy results

The prevalence of myocarditis is underestimated because of nonspecific symptoms of its onset and/or asymptomatic course. Therefore, every clinical case is interesting for the possibility of diagnosing this disease.

CASE REPORT

Patient M (33 years old) had no comorbidities or family history of cardiovascular disease. He was a non-smoker with a physically active lifestyle. He was admitted to the cardiac intensive care unit for suspected acute coronary syndrome with ST elevation. On admission, he complained of severe retrosternal pain that was "searing and burning."

Case History

Two days before his hospitalization, the described pain appeared for the first time while resting at night. It lasted for more than an hour and then disappeared spontaneously.

A similar pain attack recurred the next night. However, the pain syndrome lasted longer. ECG recorded by the emergency team showed an inferior atrial rhythm. The heart rate (HR) was 55 beats per minute. II, III, aVF, and V5–6 leads showed ST elevation up to 2 mm. aVR and aVL leads showed ST depression (Figure 1).

The patient presented with severe retrosternal pain and ECG changes that were detected before hospitalization. The patient was treated with 10 mg of intravenous morphine, 600 mg of clopidogrel, and 250 mg of acetylsalicylic acid.

Examination

During admission, the patient's consciousness was clear; the patient was cooperative, adequate, and oriented. The

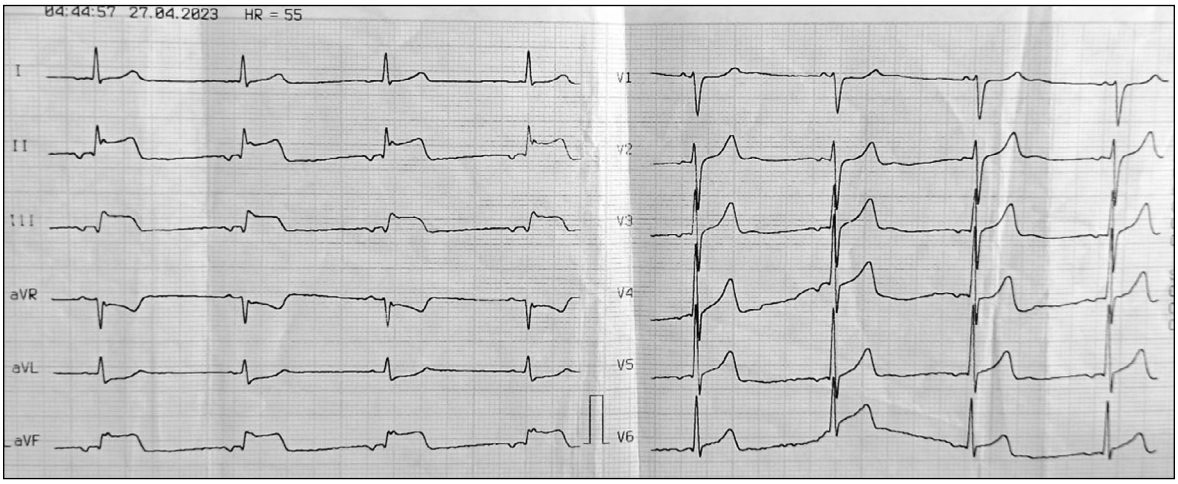

Fig. 1. Pre-hospital ECG of patient M.

skin had normal color and moisture. His vital signs were as follows:

- Body temperature of 36.8°C
- Subcutaneous fat was moderately developed. The body mass index was 27.6 kg/m^2. No peripheral edema
- Resting respiratory rate: 16 breaths/min
- Saturation: 98% in ambient air
- Blood pressure: 130/80 mmHg
- HR: 100 beats /min, the rhythm was normal

The left border of relative cardiac dullness in the fifth intercostal space was 1.5 cm medial to the left midclavicular line. The right border was along the right edge of the sternum. The upper border was in the third intercostal space.

Physiological accentuation of heart sounds was preserved. No heart murmurs were heard. Lung breathing was vesicular. No wheezing was heard. The abdomen was soft and painless. The liver was not enlarged (Kurlov ordinates were 10 × 8 × 7 cm). The spleen was not palpable. Stool and diuresis were normal.

C-reactive protein, antistreptolysin O, troponin I, and aspartate aminotransferase levels were elevated (Table 1).

Based on the clinical picture, ECG changes, and elevated cardiac enzymes on admission, the diagnosis was ST-elevation myocardial infarction. Emergency coronary angiography (CAG) was performed, which showed no stenotic lesions of the coronary bed in the left (Figure 2, *a*) and right (Fig. 2, *b*) coronary arteries. Signs of moderate peripheral vasospasm were observed (Figure 2).

Based on ECHO-CG data, the left ventricular (LV) ejection fraction was 60%. No abnormalities in local contractility were reported. The diastolic function of the left ventricle was not affected. No signs of myocardial hypertrophy of the left ventricle were observed. The cardiac cavities were not dilated. The contractile function of the right ventricular (RV) myocardium was preserved. The systolic pressure of the pulmonary artery was 23 mmHg with grade 0–1 tricuspid regurgitation. Some pericardial thickening was detected.

Table 1. Laboratory data of patient M.

Parameter	Results	Reference
Hemoglobin, g/L	140	130–160
RBC, 10^{12}/L	5.04	4.00–5.00
WBC, 10^{9}/L	6.5	4.00–9.00
Lymphocytes, 10^{9}/L	2.5	2.0–6.5
C-reactive protein, mg/L	10.45*	up to 6
Aspartate aminotransferase, U/L	65*	up to 50
CK-MB, U/L	1.0	up to 25
Antistreptolysin O, GE/mL	738.9*	up to 200
Total anticardiolipin antibodies, IU/L	1.7	up to 12
dsDNA IgG antibodies, IU/mL	2.6	up to 20.0
Antinuclear antibodies (semiquantitative)	0.2	up to 1.0
Troponin I (quantitative), ng/mL	2.6*	up to 0.2
NT-proBNP	54	0–125

Daily ECG monitoring showed the following:

- The main rhythm was sinus one with episodic sinus tachycardia, moderate sinus bradycardia, and sinus arrhythmia.
- The mean HR was 73 beats per minute.
- The maximum mean HR was 111 beats per minute.
- The minimum mean HR was 53 beats per minute.
- Three individual supraventricular extrasystoles occurred; no rhythm pauses lasting >2 s were reported.
- ST elevation was recorded up to 0.2–1.0 mm (in one lead).

Treatment

The patient was treated with angiotensin-converting enzyme inhibitors, mineralocorticoid receptor antagonists,

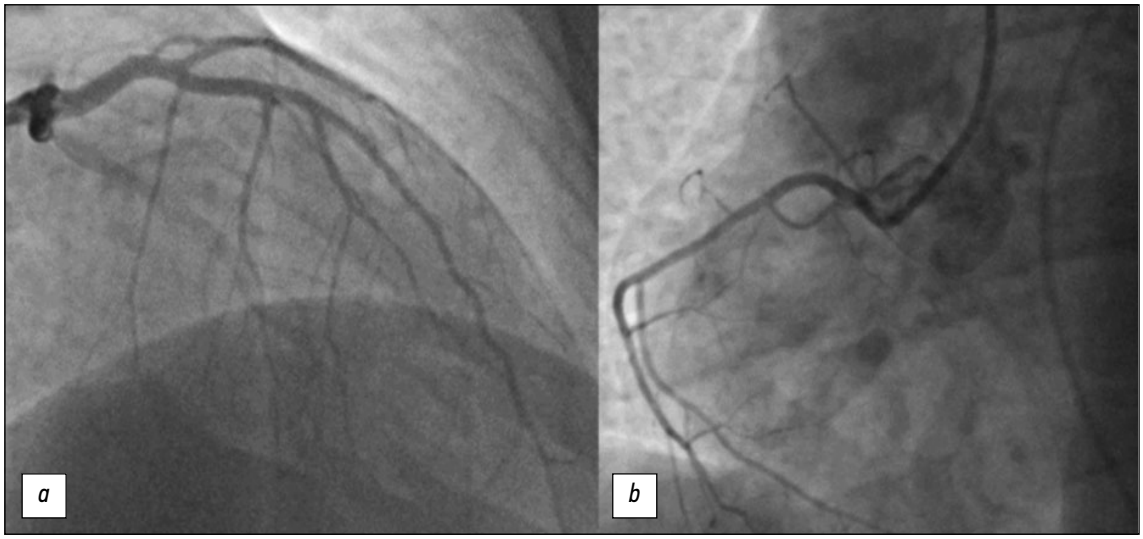

Fig. 2. Coronary angiography of patient M. (*a*) left coronary artery, (*b*) right coronary artery.

and beta-blockers. During therapy, his condition improved, and the chest pain did not recur. Troponin I and inflammatory marker levels normalized within 10 days.

ECG showed positive changes (Figure 3):

- Focal myocardial changes in posterior phrenic and lateral LV walls in qR II, Qr III, and qr aVF leads.
- SR elevation in II, III, aVF, and V4–6 leads to 0.8 mm (over time, regression was >50% of the baseline one) with negative T waves in II, III, aVF, and V5–6 leads.

Clinical diagnosis

Therefore, according to ECHO-CG data, a young patient with severe chest pain had intact coronary arteries and preserved LVEF with no abnormalities in local/diffuse contractility. Moreover, the initial increase in troponin, antistreptolysin O, aspartate aminotransferase, and C-reactive protein levels was noted with significant positive ECG changes, so the diagnosis of myocarditis was discussed.

A detailed interview revealed that 10–12 days before admission, the patient had a sore throat and a fever exceeding 38°C for 3 days. A nasopharyngeal swab was negative for coronavirus infection by polymerase chain reaction. The outpatient antibacterial therapy was reported to reduce fever and sore throat.

Since the infection was chronologically associated with clinical signs of the disease and pseudo-infarction ECG changes in the absence of coronary artery disease and with elevated cardiac enzymes, the possibility of acute myocarditis was high. To confirm the diagnosis, a cardiac MRI was performed using a gadolinium contrast agent.

The contrast-enhanced cardiac MRI did not show any enlargement of the heart chambers.

- An end-diastolic size (EDD) of the left ventricle was 52 mm.
- An indexed end-diastolic volume (EDV) of the left ventricle was 59 (reference, 59) mL/m^2.
- The EDD of the right ventricle was 43 mm.
- The indexed EDV of the right ventricle was 72 (reference, 57–109) mL/m^2).
- The sizes of the left and right atria were 41 and 48 mm, respectively.

The RV myocardium was 3–4 mm thick. No areas of local bulging or dyskinesia of the free RV wall were identified.

The LV myocardium was not hypertrophied: the thickness of the basal and middle segments did not exceed 10 mm, the thickness of the apical segments was 4–5 mm, and the indexed LV myocardial mass was 56 (reference, 41–86) g/m^2. Uniform thickening of the LV myocardium was reported during systole. No abnormalities in segmental contractility of the LV myocardium were detected. No decrease in overall LV contractility was reported with an LVEF of 64%.

No blood clots were found in the heart chambers. First-degree mitral regurgitation and first-degree tricuspid regurgitation were reported. The thoracic aorta had smooth contours and was not dilated, and the blood flow signal in its lumen was uniform. The pulmonary artery and its branches were not dilated. The superior and inferior vena cava were not dilated. In the pericardial cavity, trace amounts of free fluid were found at the level of the basal lateral LV and middle RV segments. The pericardial layers were not thickened, and the contrast agent did not accumulate. No hydrothorax was reported.

Signs of myocardial inflammation were noted including areas of early and delayed contrast enhancement in the basal and middle lateral and lower segments with the transition to the apical lower segment of the left ventricle (Figure 4*a–c*), as well as signs of myocardial edema in these segments (Figure 5*a*).

Conclusion on the cardiac MRI: MR signs of early subacute myocarditis (myocardial areas of early and delayed contrast enhancements in the lower and lateral LV walls with signs of edema).

Based on clinical, historical, laboratory, and instrumental data, the following diagnosis was made: acute infectious and allergic myocarditis, pseudocoronary clinical variant, subacute course; CAG: without stenotic lesions of the coronary arteries.

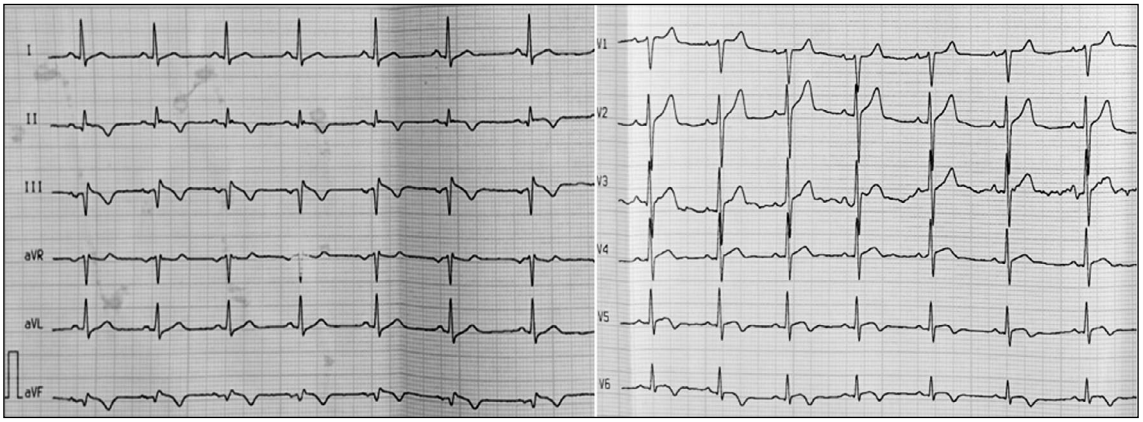

Fig. 3. ECG of patient M. over time.

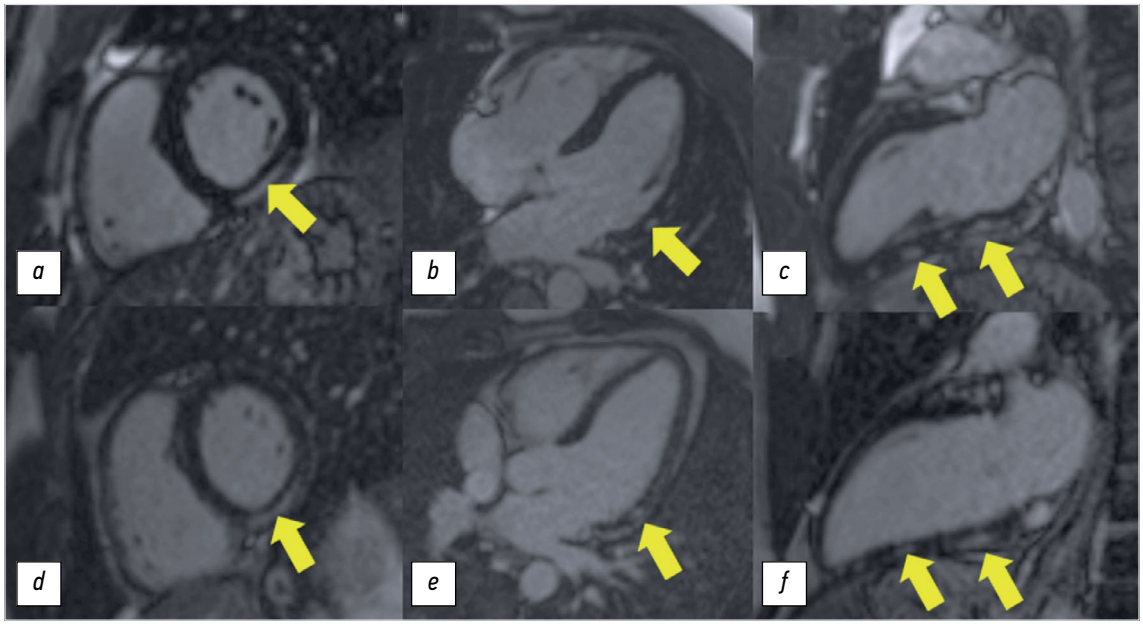

Fig. 4. Cardiac MRI in the delayed gadolinium enhancement mode (7–15 min after the administration of the contrast agent): *a*, *d*, short axis of the left ventricle in the basal segments; *b*, *e*, long axis of the left ventricle, four-chamber projection; *c*, *f*, long axis of the left ventricle, two-chamber projection. Top row, *a–c*, cardiac MRI initially: subepicardial areas of contrast enhancement (yellow arrows) are located in the basal and medial lateral and inferior segments with the transition to the apical inferior segment of the left ventricle; *d–f*, cardiac MRI in dynamics after 1.5 months: areas of delayed enhancement of the same localization and intensity remain.

According to the Mayo Clinic, myocarditis classification [5] using baseline characteristics, the patient had a low risk of cardiovascular complications and a favorable prognosis (Table 2). However, an intermediate-risk group could be considered based on cardiac MRI data.

The patient was discharged with improvement. He was recommended to continue the prescribed therapy and subject to cardiac MRI over time.

After 1.5 months, the contrast-enhanced MRI of the heart showed that the area of subepicardial delayed enhancement of the previous extent remained in the basal and middle inferior segments (Figure 4*d–f*), and no evidence of myocardial edema was found in these segments (Figure 5*b*).

DISCUSSION

The guidelines of the European Society of Cardiology [6] and the Russian clinical guidelines for the diagnosis of myocarditis [4] emphasize the effectiveness of cardiac MRI. For this purpose, the Lake Louise criteria were developed

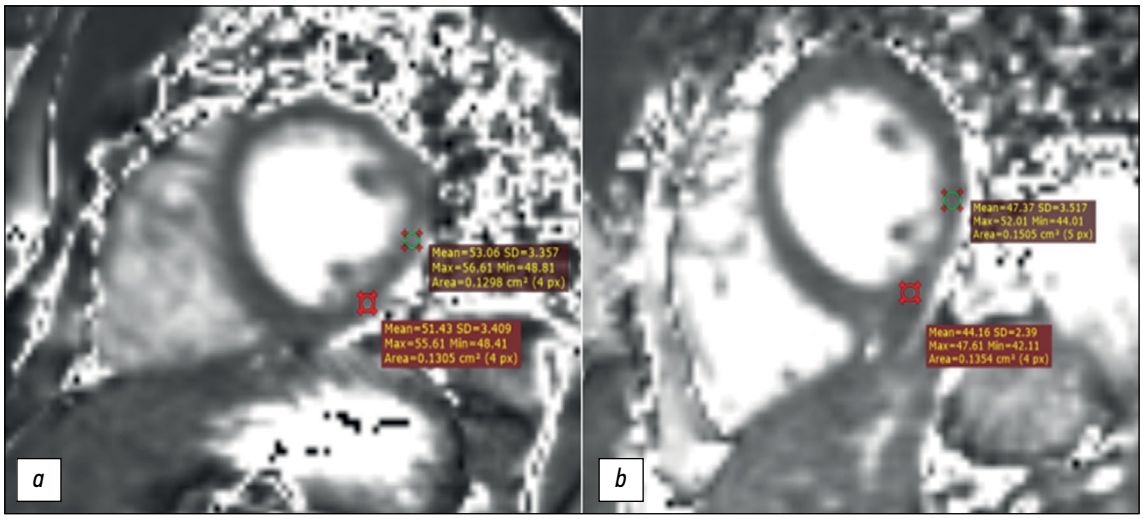

Fig. 5. Cardiac MRI T2 mapping along the short axis of the left ventricle in the basal segments: *a*, cardiac MRI at baseline: there is an increase in T2 parameter (>50 ms) in the inferior and inferolateral segments, indicating the presence of edema; *b*, cardiac MRI after 1.5 months: native T2 parameter within normal values (<50 ms). The numbers indicate the T2 parameter values in msec.

Table 2. Clinical variants of myocarditis onset (Mayo Clinic)

Low risk	Intermediate risk ("gray area")	High risk
Chest pain	Moderate persistent structural and functional changes in the myocardium	Severe persistent LV dysfunction and circulatory decompensation
Supraventricular rhythm disorders	Nonsustained ventricular arrhythmias	Life-threatening arrhythmias
AV block	No life-threatening rhythm and conduction disorders	Persistent AV block
Preserved LVEF Rapid response to therapy (1–4 weeks)	Late gadolinium accumulation in the myocardium without cardiac chamber remodeling	Recurrent syncope
Favorable prognosis	Uncertain prognosis	Unfavorable prognosis

and first published in 2009. They included the assessment of signal hyperintensity on T2-weighted images (T2WI), short T1 inversion recovery sequences, and delayed non-coronary enhancement [7].

As the effectiveness of the original Lake Louise criteria was limited by the subjective nature of the qualitative assessment of the above signs, the criteria were revised in 2018. They have been supplemented with parametric mapping, which allows the quantitative assessment of regional and global myocardial relaxation times T1 and T2 and extracellular volume (ECV). As a result, the new Lake Louise criteria have higher rates of sensitivity and specificity (88% and 96%, respectively) than the old Lake Louise criteria [8].

If at least one criterion in each category is met, cardiac inflammation is confirmed:

- T2WI signs of myocardial edema (myocardial T2WI hyperintensity or high T2 relaxation index) [9]
- T1WI signs of myocardial injury (non-ischemic pattern of delayed contrast enhancement or high T1 relaxation index and/or ECV) [10]

If only one marker is present, myocardial inflammation may be considered in the presence of clinical and/or laboratory manifestations; however, the specificity of MRI in this case is lower. Other signs of heart inflammation include systolic dysfunction (areas of hypokinesia or akinesia) and signs of pericarditis (contrast enhancement of the pericardial layers). The use of these criteria is warranted only when symptoms and signs of inflammatory heart diseases are present and not as a screening technique for asymptomatic patients [11, 12].

In this case report, contrast-enhanced cardiac MRI confirmed the clinical diagnosis. According to cardiac MRI, the patient initially had one of the above criteria for diagnosing myocarditis (myocardial edema on T2 mapping and evidence of non-ischemic myocardial injury on delayed gadolinium enhancement). MRI performed after 1.5 months showed no signs of myocardial edema, indicating a decrease in acute myocardial inflammation.

CONCLUSION

Therefore, contrast-enhanced cardiac MRI is a highly informative imaging modality for myocardial inflammation, for primary diagnosis and long-term assessment, allowing for definitive confirmation of the diagnosis.

ADDITIONAL INFORMATION

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