СЛУЧАЙ ИЗ ПРАКТИКИ

MRI IN DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION IN A PATIENT WITH HYPERTROPHIC CARDIOMYOPATHY

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Purpose. To demonstrate the capabilities of contrast enhanced cardiac MRI in the diagnosis of myocardial infarction (MI) in a patient with a combined pathology (obstructive hypertrophic cardiomyopathy), using the example of clinical observation.

Materials and methods. A clinical observation of a patient M., 61 years old is presented, who was diagnosed with myocardial infarction on the basis of complaints of pressing chest pains and ECG changes. However, during echocardiography, signs of obstructive hypertrophic cardiomyopathy were revealed for the first time, the areas of local contractility reduction (as signs of acute myocardial infarction) were not determined. To clarify the changes in the myocardium of the left ventricle, the patient underwent cardiac MRI, the results of which confirmed the presence of acute infarction and clarified the degree and form of myocardial hypertrophy. To confirm the diagnosis of hypertrophic cardiomyopathy, a genetic study was performed.

Results. During the ECG, indirect signs of myocardial infarction and signs of combined ventricular hypertrophy were revealed, according to echocardiography – there were asymmetrical left ventricular myocardial hypertrophy with the development of left ventricular outflow tract obstruction and left ventricular diastolic disfunction. Contrast-enhanced cardiac MRI revealed asymmetric left ventricular myocardial hypertrophy, left ventricular outflow tract obstruction, as well as pathological accumulation of gadolinium: characteristic of ischemic lesion (acute myocardium infarction), as well as fibrosis in hypertrophied myocardium. A genetic study revealed mutations confirming hypertrophic cardiomyopathy.

Discussion. Diagnosis of heart diseases, including acute myocardial infarction, against the background of severe myocardial hypertrophy is significantly difficult. In the presented case, classical cardiological diagnostic methods did not allow an accurate diagnosis of acute myocardial infarction.

Conclusion. Cardiac MRI allows to identify violations of the structure of the myocardium, as well as to determine the cause of their occurrence, differentiating ischemic and non-ischemic damage. The diagnostic capabilities of standard diagnostic methods, such as ECG and echocardiography, are inferior to the capabilities of cardiac MRI with delayed contrast when assessing the state of the myocardium in difficult situations, especially within a combination of various diseases.

Keywords: cardiac MRI, myocardial infarction, hypertrophic cardiomyopathy.

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МРТ В ДИАГНОСТИКЕ ОСТРОГО ИНФАРКТА МИОКАРДА У ПАЦИЕНТКИ С ГИПЕРТРОФИЧЕСКОЙ КАРДИОМИОПАТИЕЙ

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Цель. Продемонстрировать возможности магнитно-резонансной томографии сердца с контрастированием в диагностике инфаркта миокарда при наличии сочетанной патологии сердца (обструктивной гипертрофической кардиомиопатии) на примере клинического наблюдения.

Материалы и методы. Представлено клиническое наблюдение пациентки М., 61 год, которой, на основании жалоб на давящие загрудинные боли и изменений на ЭКГ, был диагностирован инфаркт миокарда. Однако, при проведении эхокардиографии были впервые выявлены признаки обструктивной гипертрофической кардиомиопатии, а зоны локального снижения сократимости (как признаки острого инфаркта миокарда) не определялись. Для уточнения изменений миокарда левого желудочка пациентке была выполнена магнитно-резонансная томография сердца с отсроченным контрастированием, результаты которой подтвердили наличие острого инфаркта и уточнили степень и форму гипертрофии миокарда. Для подтверждения диагноза гипертрофической кардиомиопатии больной было выполнено генетическое исследование.

Результаты. При проведении ЭКГ были выявлены косвенные признаки инфаркта миокарда и признаки комбинированной гипертрофии желудочков, по данным эхокардиографии – гипертрофия миокарда левого желудочка с развитием обструкции выносящего тракта левого желудочка и нарушением диастолической функции. При МРТ сердца с контрастным усилением определялась ассиметричная гипертрофия миокарда левого желудочка, обструкция выносящего тракта, а также патологическое накопление гадолиния, характерное для острого ишемического поражения (острого инфаркта миокарда) и участки фиброза в гипертрофированном миокарде. При генетическом исследовании были выявлены мутации, подтверждающие гипертрофическую кардиомиопатию.

Обсуждение. Диагностика заболеваний сердца, в том числе острого инфаркта миокарда, на фоне выраженной гипертрофии миокарда значительно затруднена. В представленном случае классические кардиологические методы диагностики не позволили точно поставить диагноз острого инфаркта миокарда.

Заключение. МРТ сердца позволяет выявить нарушения структуры миокарда, а также определить причину их возникновения, дифференцируя ишемическое и не ишемическое повреждение. Диагностические возможности таких стандартных методов диагностики, как ЭКГ, ЭхоКГ уступают возможностям МРТ сердца с отсроченным контрастированием при оценке состояния миокарда в сложных ситуациях, особенно при сочетании различных заболеваний.

Ключевые слова: МРТ сердца, инфаркт миокарда, гипертрофическая кардиомиопатия.

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Hypertrophic cardiomyopathy (HCM) is a genetically determined myocardial disease characterized by hypertrophy of the myocardium of the left and/or right ventricle. The most common cause of HCM is a mutation in sarcomeric proteins, which is transmitted by an autosomal dominant type [1]. According to the literature 15-20% of patients with HCM have signs of atherosclerosis of epicardial coronary arteries [2]. The addition of atherosclerotic lesions of the coronary bed leads to a significant deterioration in the prognosis of the underlying disease. When evaluating the results of multicenter studies, it was noted that in 10% of cases, a combination of HCM and coronary heart disease (CHD) is possible [3, 4, 5].

One of the known complications in patients with hypertrophic cardiomyopathy is acute myocardial infarction (AMI) [2]. Pathogenetic mechanisms of MI development in patients with HCM may be different: ischemia may be caused by obstructive lesions of epicardial coronary arteries (type 1 MI) or occur in the absence of obstructive lesions of epicardial coronary arteries (mainly type 2 MI) [5].

In this article we present a clinical case of a patient with 1st type acute myocardial infarction and hypertrophic cardiomyopathy, she was diagnosed with HCM after the development of AMI.

Case report.

Patient M., 61 years old, female, for a long time has experienced arterial hypertension with rises in blood pressure (BP) up to 250/120 mmHg. The patient has had chest pain for 3 years, but she did not seek medical help. Five years ago she was diagnosed with type 2 diabetes mellitus.

In June 2022, for the first time, a prolonged anginal attack has developed, accompanied by ECG changes: ST segment depression up to 2 mm, negative T-wave in the I standard lead, in aVL, left thoracic leads, which were regarded as ischemic dynamics. The patient was urgently hospitalized in the regional vascular center. Laboratory findings showed an increase in the level of troponin T in dynamics and a diagnosis of acute myocardial infarction without ST segment elevation was made according to the criteria of the Fourth universal definition of MI [6]. During emergency coronary angiography diagonal artery stenosis was detected – 60%, right coronary artery stenosis – 40%, subtotal

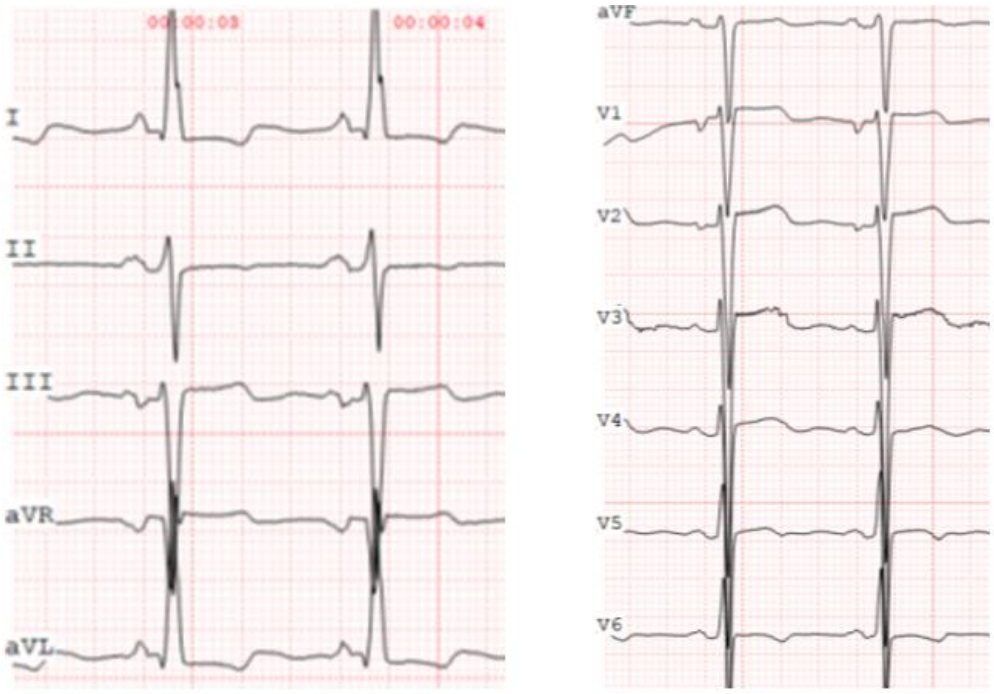

Fig. 1 (Рис. 1)

Fig. 1. ECG.

Sinus rhythm, heart rate 71 bpm. Deviation of the electrical axis of the heart to the left. Signs of LV hypertrophy (Cornell index 36 mm), signs of an LA enlargement (expansion of the P wave to 0.12 s)..

Рис. 1. ЭКГ.

Ритм синусовый ритм, ЧСС-71 уд/мин. Отклонение электрической оси сердца влево. Признаки гипертрофии ЛЖ (Корнельский индекс 36 мм), признаки увеличения ЛП (расширение зубца Р до 0,12 с).

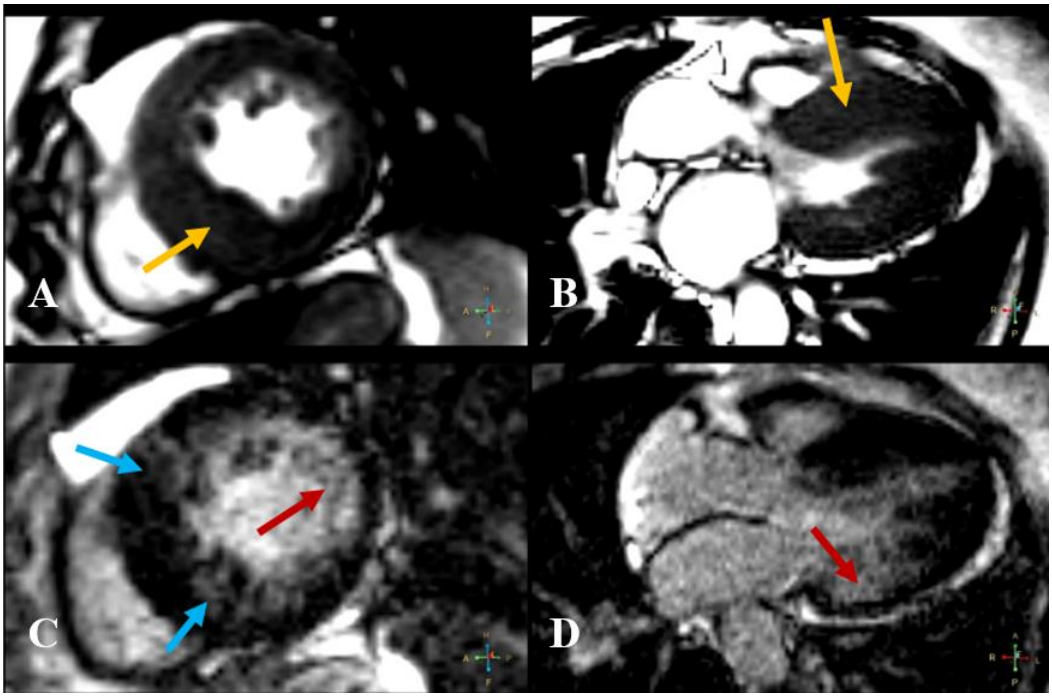

Fig. 2 (Рис. 2)

Fig. 2. cMRI of the heart.

A, B – cine-MRI (A – short LV axis, B – long LV axis); C, D – delayed contrast (Inversion Recovery) (C – short LV axis, D – long LV axis). LV myocardial hypertrophy is detected by cine-MRI (yellow arrows); contrast scans demonstrate the zone of subendocardial accumulation of contrast agent – the zone of acute myocardial infarction (red arrows) and the zones of intramyocardial accumulation (blue arrows).

Рис. 2. МРТ сердца.

А, В – кино-МРТ (А – короткая ось ЛЖ, В – длинная ось ЛЖ); С, D – отсроченное контрастирование (Inversion Recovery) (С – короткая ось ЛЖ, D – длинная ось ЛЖ). На кино-МРТ определяется гипертрофия миокарда МЖП (желтые стрелки); на томограммах с отсроченным контрастированием красные стрелки указывают на зону субэндокардиального накопления контрастного препарата – зону острого инфаркта миокарда, голубые стрелки – на зону интрамиокардиального накопления.

stenosis of the posterolateral artery, in connection with which stenting of the posterior lateral coronary artery was performed, after which anginal pain was relieved. After the restoration of full blood flow through the narrowed coronary artery and the appointment of conservative therapy, the patient's condition improved, intense chest pain did not recur, however, the patient was still bothered by chest pain, shortness of breath with a small load, general weakness. During echocardiography, attention was drawn to a significant thickening of the interventricular septum (IVS) up to 2 cm, as well as the absence of violations of local contractility, preserved global left ventricle (LV) contractility. Taking into account the persistent complaints, the absence of areas of violation of local contractility (as signs of myocardial infarction in echocardiography), the patient was transferred for examination and clarification of the diagnosis to the E.I. Chazov Research Centre of Cardiology.

Patient had complained of chest pains of varying intensity, both against the background of moderate intensity of physical exertion and at rest, shortness of breath, general weakness. On the ECG, a sinus rhythm with a heart rate of 71 beats/min was recorded, signs of LV hypertrophy (Cornell index 36 mm at normal less than 20 mm for women), signs of an LA enlargement (expansion of the P wave to 0.12 s with a norm of up to 0.1 s) (fig. 1).

According to cardiac ultrasound, there were no clear areas of abnormalities of local contractility, symmetrical hypertrophy of the myocardium of the left ventricle (LV) was defined (the thickness of the interventricular septum in the basal segment was up to 18 mm, in the middle segment – 20 mm, the posterior wall of the left ventricle – 17 mm) with the development of significant LVOT obstruction. Global LV myocardial contractility was satisfactory (ejec-

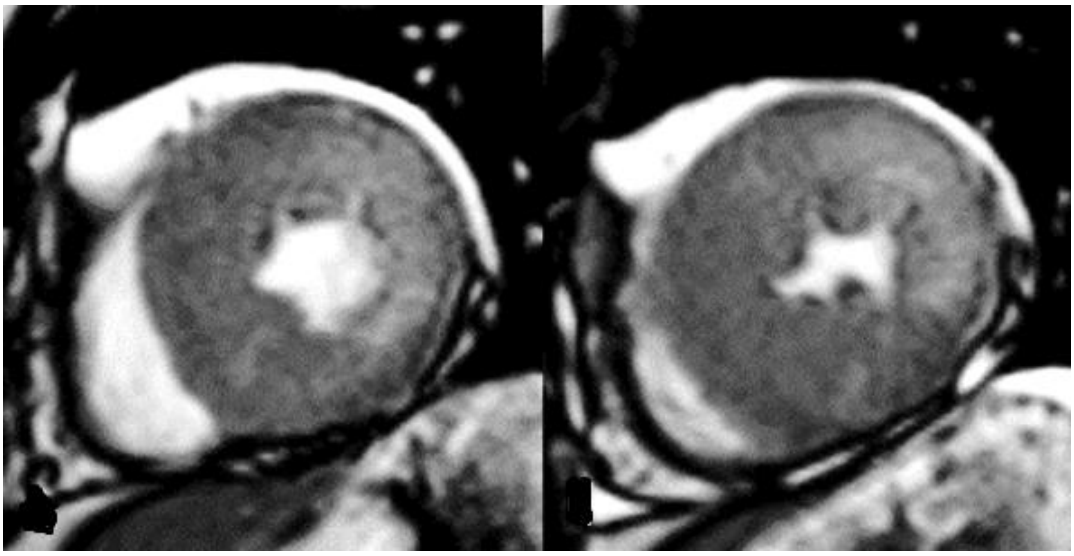

Fig. 3 (Рис. 3)

Fig. 3. Cardiac MRI, T2-weighted images two-chamber projection, short axis, basal and middle segments.

Areas of increase in the MR signal characteristic of edema are indicated by an arrow.

Рис. 3. МРТ сердца, Т2-взвешенные изображения, двухкамерная проекция, короткая ось, базальный и средний сегменты.

Зоны повышения МР-сигнала, характерные для отека, указаны стрелкой.

tion fraction >60% according to Simpson). Thus, according to ECG and echocardiography, convincing data for a myocardial infarction wasn't obtained. Since the presence of MI in the anamnesis is a serious unfavorable factor relative to the prognosis of the disease, a cMRI with contrast enhancement was performed. According to MRI data, asymmetric hypertrophy of the myocardium of interventricular septum (IVS) was determined throughout its entire length (thickness in the basal segment up to 21 mm, in middle – 22 mm, in the apical – 15 mm), uneven thickening of the anterior and lower walls (in the basal segment – up to 15 mm and 13 mm, in the middle and apical segments – up to 10-12 mm). No thickening of the lateral wall was detected (myocardial thickness up to 8-10 mm). There weren't abnormalities of segmental contractility of the LV myocardium. Signs of obstruction of the LVOT due to anterohistolic movement of the mitral valve leaflet and hypertrophy of the anterior LV wall in the basal segment were determined.

After the introduction of the contrast agent, it's pathological accumulation of two types was determined. In the middle segment and, partially, in the adjacent parts of the basal and apical segments of the lateral and anterior walls of the LV, subendocardial accumulation of contrast agent was detected, which is characteristic of ischemic lesions. In addition, single small intramyocardial accumulation sites were detected in the anterior and lower septum regions of the middle segment of the LV (on the border with the free wall of the right ventricle), which most likely reflects the presence of fibrosis zones in the hypertrophied myocardium (fig. 2). The T2-weighted images in the same zone showed an increase in the MR signal, which was regarded as a manifestation of edema (fig.3).

According to the results of the examination, the patient had significant hypertrophy, the severity of which did not correspond to that of a patient with corrected arterial hypertension, as well as focal myocardial lesion of an ischemic nature in the acute (less than 4 weeks) stage, which confirmed the diagnosis of AMI.

To confirm the diagnosis of HCM, a genetic study was performed in which a mutation was detected in the gene encoding the sarcomere contractile protein – myosin-binding protein C (MYBPC3) (fig. 4). The most common mutations in the genes encoding myosin (MYH7), myosin-binding protein C (MYBPC3), actin (ACTC), troponin (TNNI3, TNNT2, TNNC) are at the basis of the development of HCM. About half of the cases of HCM are due to pathogenic variants in the MYH7 and MYBPC3 genes [5].

Thus, the results of the examination allowed us to make the following diagnosis: coronary heart disease: acute non-ST-elevation my-

ocardial infarction. Atherosclerosis of the coronary arteries: diagonal artery stenosis of 60%, right coronary artery stenosis of 40%, subtotal posterolateral artery

stenosis. PTCI of the posterior lateral artery. Hypertrophic cardiomyopathy, obstructive form.

Concomitant diseases: Hypertension of stage III, grade 3. Type 2 diabetes mellitus. Cerebrovascular disease. Dyscirculatory encephalopathy of the 2nd degree.

The patient was discharged in a satisfactory condition under the supervision of a cardiologist, endocrinologist at the place of residence.

Discussion.

Hypertrophic cardiomyopathy is the most common genetically determined heart disease, with an estimated prevalence in the general population of 1:500 (0.2%). The disease is characterized by extreme heterogeneity of the clinical course, the most common and well-recognized clinical complications of HCM are arrhythmic sudden death, progressive heart failure and atrial fibrillation.

Concomitant atherosclerosis in epicardial coronary arteries is observed in 15-25% of patients with HCM (mainly in the older age group of patients). Myocardial infarction (MI) can develop both type 1 (with atherothrombosis) and type 2

(without atherothrombosis) [5, 7, 8]. Factors leading to myocardial ischemia in patients include:

- relative coronary insufficiency (decrease in the reserve of coronary blood flow in severe hypertrophy);
- ischemia of the subendocardial layers of the myocardium due to compression of intramural coronary arteries;
- perivascular fibrosis, while intramural coronary arteries cannot expand during exercise, when the myocardial oxygen demand increases and an increase in coronary blood flow is required;
- congenital pathology of the coronary arteries – "myocardial muscle bridges" and intramural location of branches of large coronary arteries.

Myocardial ischemia due to coronary heart disease is an underestimated component of the process of hypertrophic cardiomyopathy [2]. There are few descriptions of myocardial infarction in hypertrophic cardiomyopathy in the

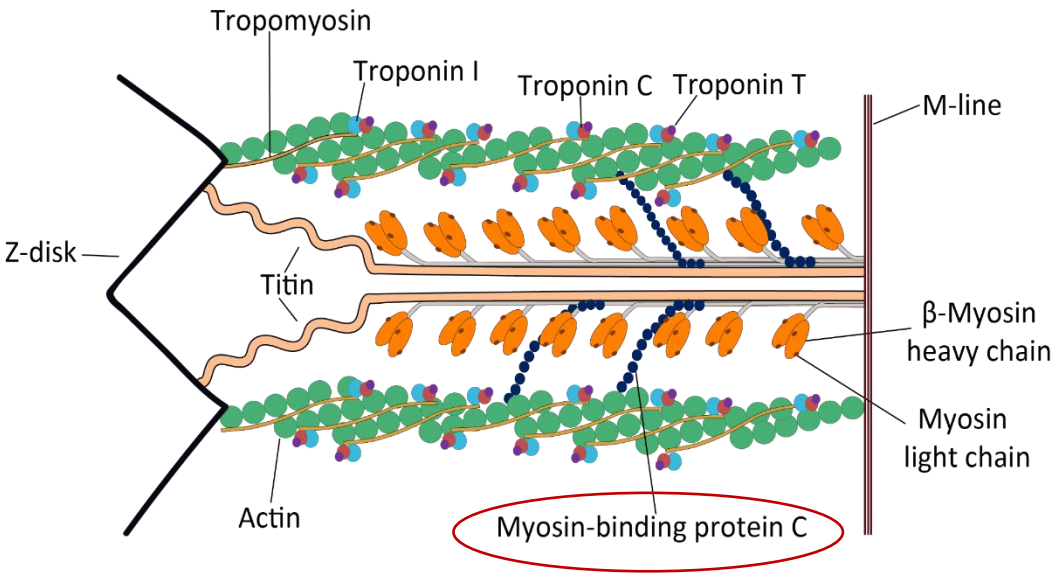

Fig. 4 (Рис. 4)

Fig. 4. Scheme.

The structure of the cardiac sarcomere with tropomyosin. Cardiac myosin binding protein-C binds heavy chains of myosin into thin filaments and titin into elastic filaments. Phosphorylation of this protein provides reduction modulation (the illustrative material is borrowed from publicly available Internet resources that do not contain references to the authors of these materials and any restrictions for their borrowing).

Рис. 4. Схема.

Строение сердечного саркомера с тропомиозином. Сердечный миозин-связывающий протеин-С связывает тяжелые цепи миозина в тонкие филаменты и титина в эластичные филаменты. Фосфорилирование данного протеина обеспечивает модулирование сокращения (иллюстративный материал заимствован из общедоступных ресурсов интернета, не содержащих указаний на авторов этих материалов и каких-либо ограничений для их заимствования).

literature, most of which tell about type II MI in young patients with the absence of obstructive coronary artery disease [9-11].

This article describes a clinical case of a combination of HCM and coronary artery disease with obstruction of coronary arteries that led to non-ST elevation acute myocardial infarction. The diagnosis of acute myocardial infarction was established on the basis of presence of an anginal attack in the patient, an increase in troponin levels and ischemic changes on the ECG. At the same time, during echocardiography, the patient had no violations of local contractility and during emergency coronary angiography, no occlusion or
thrombosis of any coronary artery was detected. Taking into account the presence of myocardial hypertrophy detected during echocardiography, the severity of which did not correspond to the severity of arterial hypertension, which has been medically corrected over the past 10 years, the patient underwent a contrast enhanced MRI with two goals: to confirm the presence of acute ischemic myocardial damage and to characterize myocardial hypertrophy.

The appointment of contrast MRI as a reference method was due to the unique capabilities of MRI not only to noninvasively assess the structure of the myocardium, identifying its damage, but also to determine the genesis of damage. A distinctive feature of the method is the high tissue resolution, which makes it possible to detect various myocardial lesions, including small ones located intramiocardially and subendocardially [5, 12]. Gadolinium-containing contrast agent used for contrast MRI, which has an extracellular distribution character, allows not only to identify areas of necrosis and fibrosis, but also to divide the causes of the lesion into ischemic and non-ischemic [1, 4, 13]. In addition, MRI has a unique ability to differentiate acute and non-acute lesions based on signal changes in T2-weighted images and T2-maps during mapping [14]. In the described case, two different patterns of accumulation of contrast agent were revealed: characteristic for ischemic injury (subendocardial) in the LV lateral wall and for non-ischemic lesion (focal intramiocardial) in the lower parts of the LV, which is typical for HCMP [13]. The presence of edema in the zone of subendocardial accumulation indicated the acute nature of the changes, which corresponded to the approximate time of myocardial infarction.

The results obtained after long-term observation indicate that the addition of atherosclerotic lesions of the coronary bed leads to a significant deterioration in the condition of patients and the prognosis of the underlying disease, myocardial ischemia is an unfavorable prognostic marker of hypertrophic cardiomyopathy and is associated with left ventricular remodeling, the development of systolic dysfunction, sudden cardiac death (SCD) and higher overall mortality [2, 3]. Thus, of course, an important factor is the rapid and accurate diagnosis of MI and HCM.

Conclusion.

Contrast enhanced cMRI confirmed the diagnosis of AMI in a patient with severe myocardial hypertrophy, in which it was impossible to determine the infarction zone by traditional cardiological methods (ECG, echocardiography). The diagnosis of HCM in a patient with a long history of arterial hypertension was confirmed by genetic analysis. This clinical case demonstrates the precision and high informative value of contrast enhanced cMRI in the examination of patients with myocardial hypertrophy. The use of the technique allows one study to obtain complete information about the morphology of the myocardium, the function of the heart, the presence of changes in
the structure of the myocardium, to determine the ischemic or non-ischemic nature of the detected changes, as well as the period of their occurrence.

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The Author(s) declare(s) that there is no conflict of interest.

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