DOI: 10.20514/2226-6704-2023-13-5-352-359
УДК: [616.127-005.8:616.132.2-009.861]-073.75-085
EDN: HJMWGV

Д.Ю. Гамаюнов*[1], А.Н. Калягин[1,3], Н.М. Балабина[2], А.В. Синьков[1], Е.С. Чуйко[1,3], Е.Р. Киселева[3], К.Б. Гайнутдинов[3], А.В. Соржеев[3], Е.О. Быков[3]

[1] — ФГБОУ ВО «Иркутский государственный медицинский университет» Министерства здравоохранения Российской Федерации, кафедра пропедевтики внутренних болезней, Иркутск, Россия
[2] — ФГБОУ ВО «Иркутский государственный медицинский университет» Министерства здравоохранения Российской Федерации, кафедра поликлинической терапии и общей врачебной практики, Иркутск, Россия
[3] — ОГБУЗ «Иркутская городская клиническая больница № 1», Иркутск, Россия

ИНФАРКТ МИОКАРДА 2 ТИПА НА ФОНЕ КОРОНАРНОГО ВАЗОСПАЗМА И ИНВАЗИВНАЯ ТАКТИКА ЕГО ДИАГНОСТИКИ И ЛЕЧЕНИЯ

D.Yu. Gamayunov*[1], A.N. Kalyagin[1,3], N.M. Balabina[2], A.V. Sinkov[1], E.S. Chujko[1,3], E.R. Kiseleva[3], K.B. Gajnutdinov[3], A.V. Sorzheev[3], E.O. Bykov[3]

[1,2] — Irkutsk State Medical University, Irkutsk, Russia
[3] — Irkutsk Municipal Clinical Hospital No1, Irkutsk, Russia

Type 2 Myocardial Infarction on the Background of Coronary Vasospasm and Invasive Tactics of Its Diagnosis and Treatment

Резюме

В настоящее время инфаркт миокарда 2 типа представляет довольно значимую проблему, как в отношении диагностики, так и в отношении лечения. Инфаркт миокарда без обструктивного поражения коронарных артерий встречается у 5-10 % пациентов. Оптимальные стратегии диагностики и лечения пациентов с повреждением миокарда, связанным с нетромботическими механизмами, еще не определены. В статье описано клиническое наблюдение развития инфаркта миокарда 2 типа на фоне вазоспазма, а также диагностическая и лечебная тактика в данной клинической ситуации. **Основные положения:** пациент 22 лет находился в кардиологическом отделении в связи с впервые в жизни возникшим болевым синдромом за грудиной и повышением температуры тела до 37,5˚С. Из анамнеза: активные занятия бодибилдингом, прием тестостерона в инъекционной форме. На электрокардиограмме были обнаружены изменения по типу трансмуральной ишемии миокарда без характерной для инфаркта миокарда динамики. Тропонин I (количественный тест) — 2,1 нг/мл при референсных значениях лаборатории 0,010-0,023 нг/мл. Проводился диагностический поиск в отношении инфаркта миокарда и острого перикардита. На эхокардиографии обнаружены зоны локального нарушения сократимости. С целью дифференциальной диагностики была проведена коронароангиография, в ходе которой выявлен динамический стеноз задней нисходящей артерии. Решение о стентировании сосуда принято не было. Данные проведенного обследования свидетельствовали в пользу инфаркта миокарда без обструкции коронарных артерий (2 типа). С учетом отсутствия окклюзионно-стенотических поражений коронарных артерий, наличия вазоспазма назначен один антитромбоцитарный препарат, статины в средней дозе, изосорбида динитрат, антагонист кальциевых каналов, ингибитор ангиотензинпревращающего фермента. **Заключение.** Инвазивная тактика позволила с большей вероятностью диагностировать инфаркт миокарда 2 типа и назначить наиболее оптимальную медикаментозную терапию.

Ключевые слова: *инфаркт миокарда, коронарный вазоспазм, коронароангиография, антагонисты кальциевых каналов*

*Контакты: Данил Юрьевич Гамаюнов, e-mail: d.gamayunov@mail.ru
*Contacts: Danil Yu. Gamayunov, e-mail: d.gamayunov@mail.ru
ORCID ID: https://orcid.org/0000-0001-9348-9025

Конфликт интересов
Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования
Авторы заявляют об отсутствии финансирования при проведении исследования

Статья получена 18.05.2023 г.
Принята к публикации 03.08.2023 г.

Для цитирования: Гамаюнов Д.Ю., Калягин А.Н., Балабина Н.М. и др. ИНФАРКТ МИОКАРДА 2 ТИПА НА ФОНЕ КОРОНАРНОГО ВАЗОСПАЗМА И ИНВАЗИВНАЯ ТАКТИКА ЕГО ДИАГНОСТИКИ И ЛЕЧЕНИЯ. Архивъ внутренней медицины. 2023; 13(5): 352-359. DOI: 10.20514/2226-6704-2023-13-5-352-359. EDN: HJMWGV

Abstract
Currently, type 2 myocardial infarction is a rather significant problem, both in terms of diagnosis and treatment. Myocardial infarction without obstructive coronary artery damage occurs in 5-10 % of patients with a myocardial infarction. Optimal strategies for the diagnosis and treatment of patients with myocardial damage associated with non-thrombotic mechanisms have not yet been determined. The article describes a clinical observation of type 2 myocardial infarction on the background of vasospasm, as well as diagnostic and therapeutic tactics in this clinical situation. **The main provisions:** the patient was 22 years old in the cardiology department due to the pain syndrome behind the sternum for the first time in his life and an increase in body temperature to 37.5 C. From anamnesis: active bodybuilding, taking testosterone in injectable form. The electrocardiogram revealed changes in the type of transmural myocardial ischemia without the dynamics characteristic of myocardial infarction. Troponin I (quantitative test) — 2.1 ng/ml at laboratory reference values of 0.010-0.023 ng/ml. A diagnostic search was conducted for myocardial infarction and acute pericarditis. For the purpose of differential diagnosis, coronary angiography was performed, during which dynamic stenosis of the posterior descending artery was revealed. The decision to stent the vessel was not made. Echocardiography revealed areas of local contractility disorders. The data of the examination showed in favor of myocardial infarction without coronary artery obstruction (type 2). Taking into account the absence of occlusive-stenotic lesions of the coronary arteries, the presence of vasospasm, 1 platelet aggregation inhibitor, medium-dose statins, isosorbide dinitrate, calcium channel blocker, angiotensin-converting enzyme inhibitor was prescribed. **Conclusion.** Invasive tactics made it more likely to diagnose type 2 myocardial infarction and prescribe the most optimal drug therapy.

Key words: *myocardial infarction, coronary vasospasm, coronary angiography, calcium channel blockers*

Conflict of interests
The authors declare no conflict of interests

Sources of funding
The authors declare no funding for this study

Article received on 18.05.2023
Accepted for publication on 03.08.2023

For citation: Gamayunov D.Yu., Kalyagin A.N., Balabina N.M.et al. Type 2 Myocardial Infarction on the Background of Coronary Vasospasm and Invasive Tactics of Its Diagnosis and Treatment. The Russian Archives of Internal Medicine. 2023; 13(5): 352-359. DOI: 10.20514/2226-6704-2023-13-5-352-359. EDN: HJMWGV

DAPT — dual antiplatelet therapy, PDA — posterior descending artery, MI — myocardial infarction, BMI — body mass index, CAG — coronary angiography, ACS — acute coronary syndrome, EMS — emergency medical service, CVDs –coronary vascular diseasess, FC — functional class, CHF — chronic heart failure, HR — heart rate, ECG — electrocardiography, EchoCG — echocardiography

Introduction

Myocardial infarction (MI) is a disease where cardiomyocyte necrosis is caused by imbalance between the oxygen demand and supply [1].

There are several types of MI, depending on the cause of the ischemic damage and necrosis. Type 1 MI is caused by atherothrombosis development in coronary artery and occlusive blood clot or distal embolization as an atherosclerotic plaque complication. Type 3 MI is diagnosed in the presence of clinical manifestations of ischemia, typical electrocardiogram (ECG) changes and sudden death of the patient, but in the absence of a possibility to measure necrosis markers or before their blood activity elevation. Type 4 MI is associated with percutaneous coronary intervention, while type 5 MI is caused by coronary artery bypass grafting [2].

Type 2 MI is a result of an absolute reduction in oxygen supply to myocardium and/or hypoperfusion due to a higher demand of myocardium in oxygen in the absence of acute atherothrombosis, but in the presence of another factor [1]. There are numerous causes of type 2 MI, including tachyarrhythmia, marked hypoxia, hypotension, coronary spasm, coronary embolism, coronary artery dissection and some other causes [3].

Diagnostics and management of type 2 MI are challenging [3], since the estimated incidence varies greatly — from 1.6 % to 74 % [1]. MI without coronary artery obstruction is diagnosed in 5–10 % of patients [4]. In a majority of cases, patients in this category are older, they have more comorbidities and lower peak troponin levels vs. patients with type 1 MI; this disease mostly affects women [5]. According to Raphael C.E. et al., prognosis is likely to be associated with a trigger. It is assumed that prognosis in arrythmia-caused MI is more favourable than in MI caused by hypoxia, hypotension or anemia [6].

Due to ischemia, clinical presentation of type 2 MI may hardly have any prominent differences vs. type 1 MI. At the same time, clinical signs of the underlying cause in type 2 MI make symptoms interpretation even for challenging [3]. The significance of the problem is accentuated also by the fact that in a number of cases the MI type is not identified. Clinical cases of type 2 MI are very often classified as MI without ST-segment elevation, despite significant differences in the nosetiology, management and outcomes. Type 2 MI is associated with higher 30-day mortality rates (from all causes) (13.5 % vs. 2.9 %) and repeated hospital admission rates (17.7 % vs. 13.9 %) as compared to type 1 MI [7].

Lack of markers specific to various types of MI was a trigger for clinical studies aimed at the search for parameters, the level of which is more sensitive to a certain MI type. Bormann et al. conducted clinical studies of the diagnostic significance of C-reactive protein (CRP). It was found out that type 2 MI patients (n=55) had significantly elevated CRP levels vs. type 1 MI patients (n=199) (0.6 vs. 0.3, p = 0.02) [8].

It is worth noting that even if the exact or a suspected cause is known, the use of specific algorithms for diagnostics and management of type 2 MI is challenging [9]. The optimal therapeutic and diagnostic approaches for myocardium damage associated with non-thrombotic mechanisms have not been identified yet, and the currently available scales do not provide for a reliable classification of short-term and long-term risks [3, 5]. The practicability of coronary examination in type 2 MI is being studied in the randomised controlled study of early coronary angiography (CAG) as compared to conservative therapy in the presence of criteria compatible with type 2 MI [10].

In this article, we are presenting a case study of type 2 MI development associated with vasospasm as well as a diagnostic and therapeutic strategy for such clinical situation.

Case Study

22-year-old patient B. was delivered to the Admission Room by the emergency medical service (EMS) team. According to the patient, at night, when the patient was sleeping, for the first time in his life he had squeezing retrosternal pain which made him wake up. Pain syndrome was accompanied by pale skin and lasted for a long time (more than 1 hour). Body temperature elevated to 37.5 °C. Pre-hospital ECG demonstrated sinus rhythm and heart rate (HR) of 80 bpm and ST-segment elevation in I, II, aVL, aVF, V_4-V_6. Pain was arrested with 2 doses of sublingual isosorbide dinitrate spray. The EMS team administered heparin intravenously (5000 units) and gave the patient acetylsalicylic acid (250 mg). Suspecting acute coronary syndrome (ACS), the EMS team transported the patient to the Admission Room of Irkutsk City Clinical Hospital No. 1.

During initial examination, the patient did not complain of chest pain. Collection of the family history of cardiovascular diseases (CVDs) was challenging: according to the patient, his father had MI at the age of 30 years (but the patient is not sure). The patient denies smoking, abuse of alcohol, drug abuse. The patient denies CVDs, including arterial hypertension, and comorbidities. In 2020, the patient had COVID-19 and was vaccinated approximately one year ago. During history taking, it was revealed that the patient was a body builder. For the last two weeks, he had been injecting intrevenous testosterone enanthate (500 mg once weekly) for muscle gains.

Objective findings: moderately severe condition, active position; the patient is lucid, calm expression on his face, talkative. Proportional, normosthenic body build. Body weight — 88 kg, height — 177 cm, body mass index (BMI) — 28.1 kg/m^2. Skin is of normal colour, moderately moist; skin tightness is preserved. Scalp hair is uniform; body hair is excessive. Nail plates are smooth, slightly round, rose-pink. Visible mucous membranes are rose-pink and moist. The adipose tissue is moderate. No oedema. Peripheral lymph nodes are not palpable. Muscle development is satisfactory; shoulder muscles, biceps and triceps are hypertrophic. Muscle tone is satisfactory; 5 points on a 5-point muscle strength scale. When palpated, muscles are painless. The skeleton is proportional, without deformities. When palpated, bones are painless. Joint shape and overlying skin are normal; when palpated, joints are painless; active and passive joint movements are full-fledged. The chest in normosthenic. When palpated, the chest is painless. When percussing, the lung border is normal; auscultatory, breathing is vesicular, without vesicular murmur. Oxygen saturation is 98 %. The cardiac border is normal; auscultatory, cardiac sounds are clear, the rhythm is normal. The heart rate is 91 bpm and corresponds to the a. radialis pulse wave frequency. Blood pressure (BP) is 116/70 mm Hg. No digestive and urinary abnormalities. Body temperature is 37.3 °C.

Laboratory test results

Complete blood count upon admission shows leucocytosis (15.7×10^9/L), all other parameters are within the reference range. No blood chemistry abnormalities were found. Coagulation profile: fibrinogen — 4.2 g/L, activated partial thromboplastin time — > 120 s, INR — 1.05, Quick's value along — 103 %. Troponin I — 2.1 ng/mL (reference range: 0.010–0.023 ng/mL). SARS-CoV-2 express test (nasopharyngeal swab): negative.

Instrument-aided test results

ECG upon admission: sinus rhythm with heart rate of 84 bpm. Persistent ST-segment elevation in I, II, aVL, aVF, V_4-V_6 (1 mm) (Fig. 1). If compared to EMS ECG: no changes. Chest X-ray (frontal view): no focal, infiltrative changes in lungs; the roots are anatomical, not dilated, sinuses are unobstructed, heart shadow is not dilated.

For provisional diagnosis it was necessary to differentiate between acute MI and acute pericarditis.

Echocardiography (EchoCG) was performed: left ventricular ejection fraction (EF) was 50 % (Simpson); areas of hypokinesia were seen in the bottom, posterolateral walls in apical and mid-segments.

Taking into account long-lasting anginal pain which was later arrested, elevated troponin I level, no ECG changes, areas of hypokinesia and absence of pericardial effusion on EchoCG, CAG was performed for additional examination and differential diagnosis subject to voluntary consent from the patient and administration of a loading dose of ticagrelor (180 mg).

Following CAG, RCA dominance was observed. First angiocardiograms of the right coronary artery showed the posterior descending artery (PDA) as a stump (Fig. 2). Following intracoronary nitroglycerine injection, PDA could be seen entirely (Fig. 3); no occlusive and stenotic involvement of other coronary arteries was observed (Fig. 4). Taking the dynamic nature of the PDA stenosis into account, it was decided that vascular stenting was not required.

After CAG, the patient was transfered to ICU. He did not have any major complaints. Auscultatory, breathing is vesicular; without vesicular murmur; oxygen saturation: 97 % without additional oxygenation. Heart sounds are muffled; the rhythm is normal; heart rate is 94 bpm. BP — 117/73 mm Hg. ECG recording: sinus rhythm with heart rate of 72 bpm; signs of incomplete right His bundle branch block. Development of bottom and posterolateral MI (ST-segment is close to isoline in I, II, aVL, aVF, V_4-V_6; negative T-wave in II, aVF, V_6; increased negative T-wave amplitude in III; development of biphase T-wave in V_4-V_5) (Fig. 5). Blood troponin I was measured qualitatively over time. The result was positive. CRP level was elevated to 221.3 mg/L. Lipid profile: total cholesterol — 4.12 mmol/L, triglycerides — 0.59 mmol/L, HDL cholesterol — 0.69 mmol/L, LDL cholesterol — 2.77 mmol/L. Urinalysis results: unremarkable.

Taking into account complaints and past medical history, post-CAG ECG changes, elevated troponin I level and positive troponin qualitative measurements, CAG and EchoCG results, the following clinical diagnosis was made: "Myocardial infarction of bottom, posterolateral wall of left ventricle without coronary artery obstruction dated 14 April 2022. Emergency CAG: angiographic confirmation of PDA vasospasm, recanalization following intracoronary nitroglycerine injection. Killip I. CHF I with preserved EF (50 % on S), FC I."

Taking into account absence of occlusive and stenotic coronary artery involvement on CAG, presence

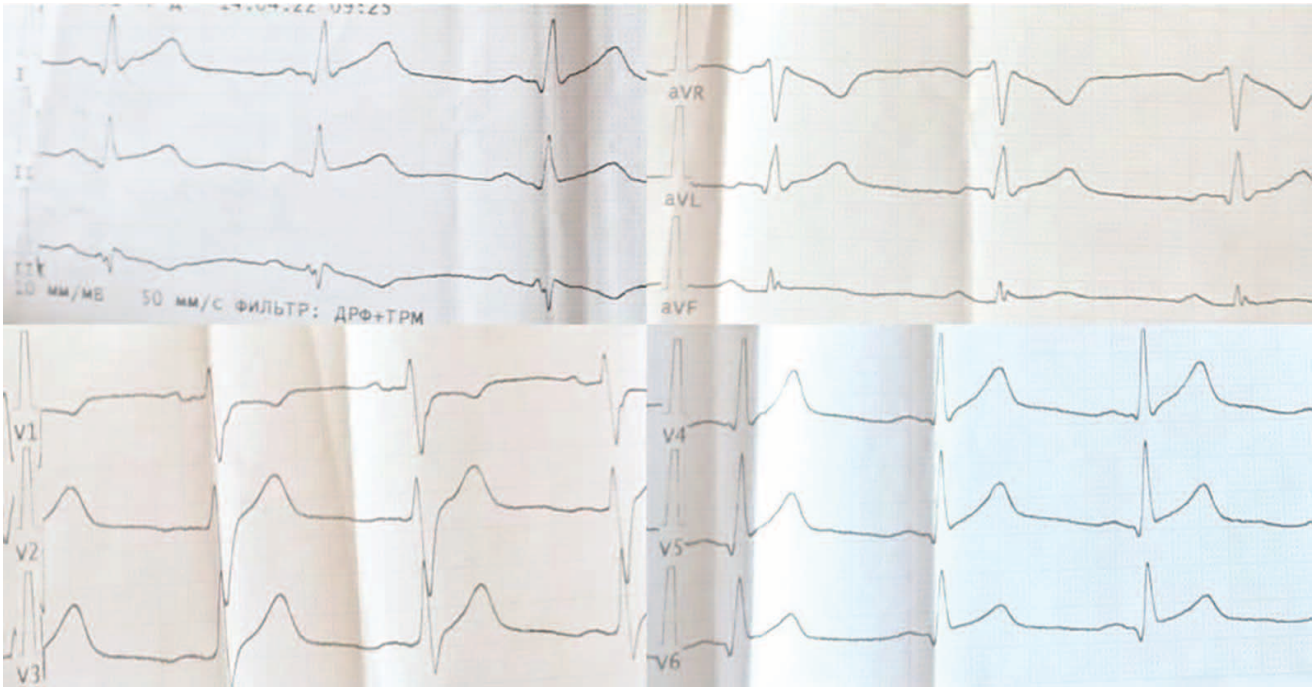

Figure 1. *ECG of the patient on admittance*

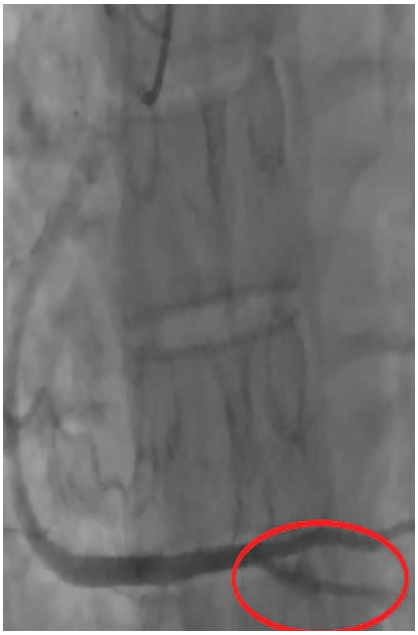

Figure 2. *The first angiogram of the right coronary artery. The posterior descending artery is contrasted in the form of a stump*

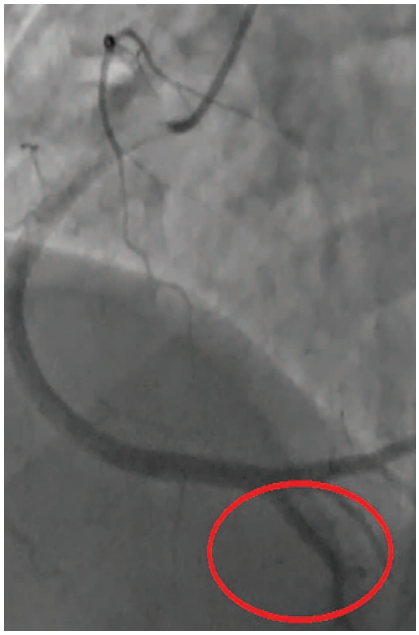

Figure 3. *Angiogram of the right coronary artery after intracoronary injection of nitroglycerin. The posterior descending artery is contrasted on all extent*

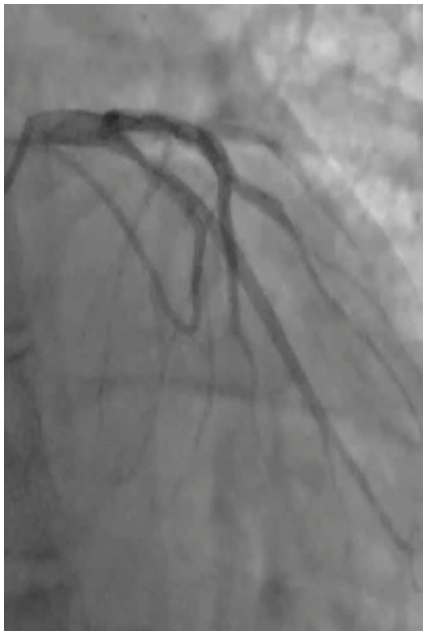

Figure 4. *Angiogram of the left coronary artery. Occlusive-stenotic lesions were not detected*

of vasospasm, it was decided to prescribe one antiplatelet drug (Clopidogrel 75 mg/day), medium statin doses (rosuvastatin 20 mg/day), prolonged-release nitrates (isosorbide dinitrate 20 mg/day) and calcium channel antagonist (amlodipine 2.5 mg/day) to prevent vasospasm.

Following positive changes in patient's condition, in 24 hours he was transferred to the Cardiology Unit. Perindopril 2.5 mg/day was added to the therapy regimen. During follow-up, positive changes were noted, mobility improved, blood white cell levels normalised, there were no changes in complete blood count, blood biochemistry and coagulation profile. ECG: typical MI developments. Holter ECG monitoring was performed: rare ectopic discharge (2 single supraventricular complexes), no pathological pauses, impaired atrioventricular conduction, episodes of diagnostically significant ST-events were recorded. Brachiocephalic artery duplex scanning did not reveal any stenosis. 6-min walk distance test results are consistent with functional class (FC) I of chronic heart failure (CHF).

Also, the patient was consulted by endocrinologist who recommended to discontinue testosterone enanthate.

The patient was discharged in satisfactory condition with therapeutic and occupational recommendations. Prescription: clopidogrel 75 mg/day for 12 months, amlodipine 2.5 mg/day, perindopril 2.5 mg/day, atorvastatin 20 mg/day, isosorbide dinitrate 20 mg/day.

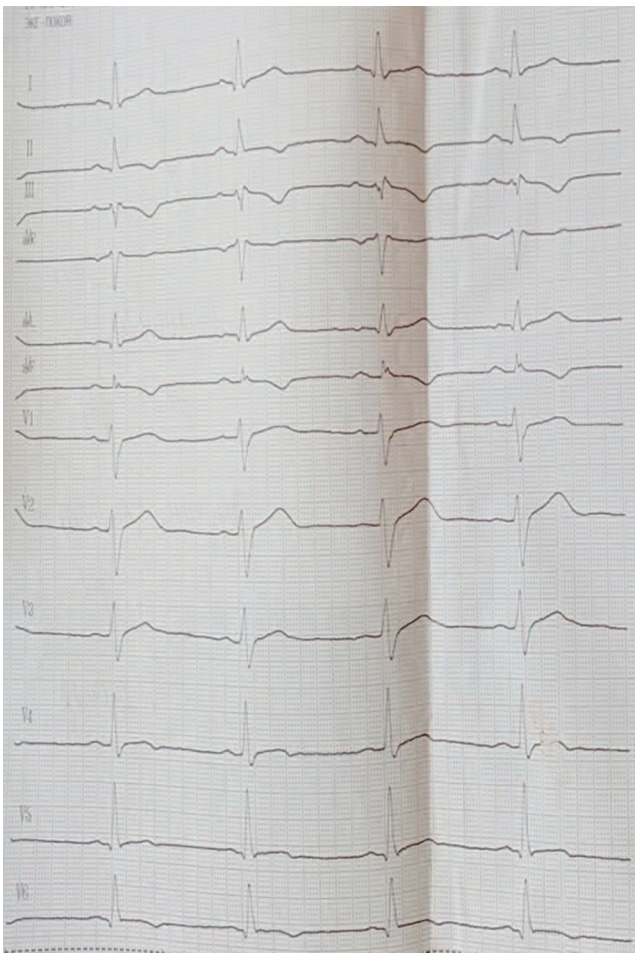

Figure 5. *ECG after coronary angiography. Dynamics of lower and posterolateral myocardial infarction*

Additional scheduled examinations were recommended. Taking into account the patient's young age, absence of atherosclerotic coronary artery involvement, an antiphospholipid syndrome examination was recommended (antiphospholipid antibodies — Ig M and Ig G). Considering vasospasm and suspected endothelial dysfunction in MI pathogenesis, we recommended to have blood tested for homocysteine and an extended test for folate cycle enzymes genes (MTHFR — methylenetetrahydrofolate reductase gene, MTR — methionine synthase gene, MTRR — methionine synthase reductase gene) in order to identify their mutations if homocysteine levels are elevated.

Discussion

Currently, type 2 MI diagnosis is somehow challenging. Because of the nature of pain syndrome, fever and ECG pattern, we were considering ACS and acute pericarditis. Clinical manifestations are also not specific for this condition. Auscultation did not reveal pericardial murmur, although this sign was observed in less than 33 % of acute pericarditis cases [11]. Subfebrile fever can occur both in acute pericarditis (if it is caused by an infection) and MI (probably as a manifestation of resorption necrotic syndrome) [12]. Usually elevated troponin I levels confirm suspected MI; however, they can show pathological processes including pericarditis [13]. Besides, elevated troponin I levels are not useful in differentiating MI types [3, 5]. Areas of hypokinesia in the bottom, posterolateral walls in apical and mid-segment during EchoCG evidence focal myocardial involvement, a sign typical of ischemic damage. However, impaired local contractility does not allow differentiating between type 1 and type 2 MI.

Taking into account patient's medical history, it is obvious that there are no CVDs; also, the young age makes it doubtful that the diagnosis is ACS.

Lack of specific signs for differential diagnosis and suspected ACS with elevated ST-segment necessitated CAG [2]. According to DeFilippis A.P. et al., CAG is the golden standard for identification of coronary anatomy and coronary trombosis; besides, it can help differentiate to some extent between type 1, type 2 MI 2 and acute myocardial damage [5]. Absence of occlusive and stenotic coronary artery involvement made it possible to rule out atherothrombosis and, hence, type 1 MI. DPA spasm seen during CAG and controlled by nitroglycerine injection was a probable cause of acute myocardium ischemia and necrosis. It can be assumed that the vasospasm could be caused by testosterone use by the patient, as there are some evidences in literature. According to Seara F.A.C. et al., vasospasm with atherosclerosis, hypercoagulation and increased thrombogenicity is seen as a probable cause of myocardial ischemia in persons consuming synthetic testosterone products [14]. There are some evidences that increased homocysteine levels and genetic defects in folate cycle enzymes which can cause homocysteine accumulation in the body are risk factors for cardiovascular pathology. In their case control study, Nedelcu C. et al. demonstrated a strong correlation between plasma homocysteine levels and first acute MI in young patients; therefore, homocysteine can be seen as a possible risk factor for MI [15]. Mechanisms of homocysteine effect on the vascular wall include endothelial dysfunction, direct impact on platelets, smooth muscle cell proliferation, oxidative HDL modification [16].

At present, relevant is the information on antiphospholipid syndrome and its role in arterial and venous blood-clotting. Lóczi L. et al. reported a high risk of MI in such patients. MI with antiphospholipid syndrome has a number of specific features: relatively young age, usually absence of signs of coronary artery atherosclerosis, a high risk of recurrent blood-clotting complications. These features are typical of the present case, therefore, the patient should undergo an additional scheduled examination for antiphospholipid syndrome [17].

Changes in ST-segment and T-wave after CAG were indicative of acute ischemic changes in the myocardium. Available imaging methods have limited capabilities in differential diagnosis of MI types. There are evidences that magnetic resonance tomography can identify conditions associated with non-MI myocardial damages [5].

According to DeFilippis A.P. et al., in the absence of a certain alternative cause in a majority of patients presenting with signs of acute ischemic damage of the myocardium, type 1 MI should be suspected, and surgery should be performed in accordance with the approved guidelines for type 1 MI. In the absence of atherothrombosis, alternative causes should be considered, including type 2 MI [5]. Based on the clinical presentation, medical history, ECG changes, EchoCG and CAG results, as well as confirmatory positive result of troponin test, type 2 MI was diagnosed which was caused by angiographically confirmed vasospasm.

The therapeutic approach for type 1 MI patients is well-known; however, there is no reliable information on the management of other types of MI [9]. Prescription of prolonged-release nitrates was necessary not for symptomatic effect, but rather for pathogenic action: vasodilation and reduction of the pre- and afterload on the myocardium [2]. The information on dual antiplatelet therapy (DAPT) is dubious. In MINOCA observational study, during the 4.1-year follow-up of patients with MI without coronary artery obstruction who were treated with DAPT, the risk of unfavourable cardiac events (death from all causes, hospital admission for MI, ischemic

stroke and heart insufficiency) was 10 % lower (OR 0.90; 95 % CI 0.74–1.08) vs. patients who were not treated with DAPT. However, the benefit from DAPT therapy was minor, while the rate of hospital admissions for bleeding grew by 33 % [4]. In this case, one antiplatelet — platelet P_2Y_{12}-receptor inhibitor Clopidogrel — was prescribed. Also, according to MINOCA study, the identifiable risk was 18 % lower (OR 0.82; 95 % CI 0.73–0.93) in patients treated with ACE inhibitors/angiotensin II receptor blockers, 23 % lower (OR 0.77; 95 % CI 0.68–0.87) in patients treated with statins, by 14 % lower in patients treated with beta-blockers (OR 0.86; 95 % CI 0.74–1.01) vs. patients who did not receive these medicinal products [4]. However, according to the clinical guidelines of the Russian Society of Cardiology, beta-blokers are not recommended in suspected coronary artery spasm [18]. The patient was prescribed atorvastatin and perindopril. Taking into account vasospastic origin of MI, amlodipine, a dihydropyridine calcium channel blocker, was prescribed to prevent vasospasm [18].

Conclusion

Therefore, despite limited data and lack of a specialised algorithm for diagnosis and management of specific MI types, except for type 1 MI, the use of invasive diagnostics in this case study allowed diagnosing type 2 MI and prescribing the most optimal drug therapy.

Вклад авторов:

Все авторы внесли существенный вклад в подготовку работы, прочли и одобрили финальную версию статьи перед публикацией

Гамаюнов Д.Ю. (ORCID ID: https://orcid.org/0000-0001-9348-9025): ведение пациента; сбор, анализ и интерпретация данных; обзор публикаций по теме статьи; написание текста рукописи; взаимодействие с редакцией в процессе подготовки публикации и печати

Калягин А.Н. (ORCID ID: https://orcid.org/0000-0002-2708-3972): ведение пациента; разработка концепции и дизайна публикации; обзор публикаций по теме статьи; написание текста рукописи; проверка критически важного интеллектуального содержания; окончательное утверждение рукописи для публикации; взаимодействие с редакцией в процессе подготовки публикации и печати

Балабина Н.М. (ORCID ID: https://orcid.org/0000-0001-7430-4558): обзор публикаций по теме статьи; написание текста рукописи; проверка критически важного интеллектуального содержания

Синьков А.В. (ORCID ID: https://orcid.org/0000-0002-7242-9346): разработка концепции и дизайна публикации; обзор публикаций по теме статьи; коррекция рукописи

Чуйко Е.С. (ORCID ID: https://orcid.org/0000-0002-9838-6970): ведение пациента; разработка дизайна публикации; обзор публикаций по теме статьи; проверка критически важного интеллектуального содержания

Киселева Е.Р. (ORCID ID: https://orcid.org/0000-0002-5587-5344): ведение пациента; разработка концепции и дизайна публикации; обзор публикаций по теме статьи; коррекция рукописи

Гайнутдинов К.Б. (ORCID ID: https://orcid.org/0000-0003-4507-0005): ведение пациента; обзор публикаций по теме статьи; написание текста рукописи

Соржеев А.В. (ORCID ID: https://orcid.org/0000-0002-7598-9103): ведение пациента; сбор, анализ и интерпретации данных; обзор публикаций по теме статьи

Быков Е.О. (ORCID ID: https://orcid.org/0000-0002-2317-2767): ведение пациента; предоставление иллюстративного материала

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Gamayunov D.Yu. (ORCID ID: https://orcid.org/0000-0001-9348-9025): case management; data collection, analysis and interpretation; review of publications on the topic of the article; writing the text of the manuscript.

Kalyagin A.N. (ORCID ID: https://orcid.org/0000-0002-2708-3972): case management; article concept and design development; review of publications on the topic of the article; writing the text of the manuscript; verification of critical intellectual content; final approval of the manuscript for publication; interaction with the editors in the process of preparing a publication for printing.

Balabina N.M. (ORCID ID: https://orcid.org/0000-0001-7430-4558): review of publications on the topic of the article; writing the text of the manuscript; verification of critical intellectual content.

Sinkov A.V. (ORCID ID: https://orcid.org/0000-0002-7242-9346): article concept and design development; review of publications on the topic of the article; correction of the manuscript.

Chujko E.S. (ORCID ID: https://orcid.org/0000-0002-9838-6970): case management; article design development; review of publications on the topic of the article; verification of critical intellectual content.

Kiseleva E.R. (ORCID ID: https://orcid.org/0000-0002-5587-5344): case management; article concept and design development; review of publications on the topic of the article; correction of the manuscript.

Gajnutdinov K.B. (ORCID ID: https://orcid.org/0000-0003-4507-0005): case management; review of publications on the topic of the article; correction of the manuscript.

Sorzheev A.V. (ORCID ID: https://orcid.org/0000-0002-7598-9103): case management; data collection, analysis and interpretation; review of publications on the topic of the article.

Bykov E.O. (ORCID ID: https://orcid.org/0000-0002-2317-2767): case management; provision of illustrative material

Список литературы / References:

1. Sandoval Y., Jaffe A.S. Type 2 Myocardial Infarction: JACC Review Topic of the Week. J Am Coll Cardiol. 2019; 73(14): 1846-1860. doi: 10.1016/j.jacc.2019.02.018.
2. Острый инфаркт миокарда с подъемом сегмента ST электрокардиограммы. Клинические рекомендации 2020. Российское кардиологическое общество, Ассоциация сердечно-сосудистых

хирургов России. Российский кардиологический журнал. 2020; 25(11): 4103. DOI: 10.15829/29/1560-4071-2020-4103.
2020 Clinical practice guidelines for Acute ST-segment elevation myocardial infarction. Russian Journal of Cardiology. 2020; 25(11): 4103. doi:10.15829/1560-4071-2020-4103 [in Russian].

3. Chapman A.R, Sandoval Y. Type 2 Myocardial Infarction: Evolving Approaches to Diagnosis and Risk-Stratification. Clinical Chemistry. 2021; 67(1): 61-69. doi: 10.1093/clinchem/hvaa189.
4. Lindahl B., Baron T., Erlinge D. et al. Medical Therapy for Secondary Prevention and Long-Term Outcome in Patients With Myocardial Infarction With Nonobstructive Coronary Artery Disease. Circulation. 2017; 135(16): 1481-1489. doi: 10.1161/CIRCULATIONAHA.116.026336.
5. DeFilippis A.P., Chapman A.R., Mills N.L. et al. Assessment and Treatment of Patients With Type 2 Myocardial Infarction and Acute Nonischemic Myocardial Injury. Circulation. 2019; 140(20): 1661-1678. doi: 10.1161/CIRCULATIONAHA.119.040631.
6. Raphael C.E., Roger V.L., Sandoval Y. et al. Incidence, Trends, and Outcomes of Type 2 Myocardial Infarction in a Community Cohort. Circulation. 2020; 141(6): 454-463. doi: 10.1161/CIRCULATIONAHA.119.043100.
7. Hawatmeh A., Thawabi M., Aggarwal R. et al. Implications of Misclassification of Type 2 Myocardial Infarction on Clinical Outcomes. Cardiovasc Revasc Med. 2020; 21(2): 176-179. doi: 10.1016/j.carrev.2019.04.009.
8. Bormann J., Psyrakis D.A., von Jeinsen B. et al. Myeloid-related protein 8/14 and high-sensitivity cardiac troponin I to differentiate type 2 myocardial infarction. Int J Cardiol. 2020; 304: 144-147. doi: 10.1016/j.ijcard.2020.01.043.
9. Лысенко М.А., Ванюков А.Е., Потешкина Н.Г. и др. Вазоспазм, как причина инфаркта миокарда 2 типа. Тактика ведения пациента за рамками рекомендаций. Российский кардиологический журнал. 2017; (9): 93-98. doi: 10.15829/1560-4071-2017-9-93-98.
Lysenko M.A., Vanyukov A.E., Poteshkina N.G. et al. Vasospasm as a cause of type 2 myocardial infarction. Tactics outside the guidelines. Russian Journal of Cardiology. 2017; (9): 93-98. doi: 10.15829/1560-4071-2017-9-93-98 [in Russian].
10. Lambrakis K., French J.K., Scott I.A. et al. The appropriateness of coronary investigation in myocardial injury and type 2 myocardial infarction (ACT-2): A randomized trial design. Am Heart J. 2019; 208: 11-20. doi: 10.1016/j.ahj.2018.09.016.
11. Adler Y., Charron P., Imazio M. и др. Рекомендации ESC по диагностике и ведению пациентов с заболеваниями перикарда 2015. Российский кардиологический журнал. 2016; (5): 117-162. doi: 10.15829/1560-4071-2016-5-117-162.
Adler Y., Charron P., Imazio M. et al. 2015 ESC guidelines for the diagnosis and management of pericardial diseases. Russian Journal of Cardiology. 2016; (5): 117-162. doi: 10.15829/1560-4071-2016-5-117-162 [in Russian].
12. Внутренние болезни. Сердечно-сосудистая система: учеб. пособие. Г.Е. Ройтберг, А.В. Струтынский. 6-е изд., перераб. и доп. М.: МЕДпресс-информ. 2019; 904 с. ISBN 978-5-00030-658-1.
Internal diseases. Cardiovascular system: training manual. G.E. Roytberg, A.V Strutynskiy. 6nd ed. M.: MEDpress — inform. 2019; 904 p. [in Russian].
13. Chauin A. The Main Causes and Mechanisms of Increase in Cardiac Troponin Concentrations Other Than Acute Myocardial Infarction (Part 1): Physical Exertion, Inflammatory Heart Disease, Pulmonary Embolism, Renal Failure, Sepsis. Vasc Health Risk Manag. 2021; 17: 601-617. doi: 10.2147/VHRM.S327661.
14. Seara F.A. C., Olivares E.L., Nascimento J.H.M. Anabolic steroid excess and myocardial infarction: From ischemia to reperfusion injury. Steroids. 2020; 161: 108660. doi: 10.1016/j.steroids.2020.108660.
15. Nedelcu C., Ionescu M., Pantea-Stoian A. et al. Correlation between plasma homocysteine and first myocardial infarction in young patients: Case-control study in Constanta County, Romania. Exp Ther Med. 2021; 21(1): 101. doi: 10.3892/etm.2020.9533.
16. Minana G., Gil-Cayuela C., Fácila L. et al. Homocysteine and long-term recurrent infarction following an acute coronary syndrome. Cardiol J. 2021; 28(4): 598-606. doi: 10.5603/CJ.a2020.0170.
17. Lóczi L., Kappelmayer J., Tarr T. et al. Antiphospholipid syndrome and the risk of myocardial infarction: current evidence and uncertainties. Kardiol Pol. 2020; 78(1): 6-14. doi: 10.33963/KP.15090.
18. Барбараш О.Л., Дупляков Д.В., Затейщиков Д.А. и др. Острый коронарный синдром без подъема сегмента ST электрокардиограммы. Клинические рекомендации 2020. Российский кардиологический журнал. 2021; 26(4): 4449. doi: 10.15829/1560-4071-2021-4449.
Barbarash O.L., Duplyakov D.V., Zateischikov D.A. et al. 2020 Clinical practice guidelines for Acute coronary syndrome without ST segment elevation. Russian Journal of Cardiology. 2021; 26(4): 4449. doi: 10.15829/1560-4071-2021-4449 [in Russian].