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Роль миокардиальных мостиков в формировании ишемии миокарда: клиническое наблюдение

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АННОТАЦИЯ

Введение. Миокардиальные мостики (ММ) рассматриваются как доброкачественная патология, однако их наличие нередко ассоциировано со стенокардией, инфарктом миокарда, желудочковой тахикардией и внезапной сердечной смертью. Клиническая значимость ММ обусловлена наличием динамического стеноза коронарной артерии, который зависит от частоты и силы сердечных сокращений, трудно визуализируется инструментальными методами, что затрудняет своевременную диагностику и раннее назначение адекватной терапии. Представлен клинический случай сочетанного поражения коронарного русла у пациента 58 лет: ММ передней межжелудочковой артерии (ПМЖА), дающий гемодинамически значимое сужение туннельной артерии, и гемодинамически незначимое атеросклеротическое поражение коронарных артерий. ММ имел типичную локализацию — в среднем сегменте ПМЖА. Диагностика аномалии коронарного русла стала возможной после проведения коронароангиографии: ММ ПМЖА стенозировал туннельный сегмент до 80% и явился причиной приступов стенокардии. Проведено стентирование ПМЖА стентом с лекарственным покрытием. Эффективность стентирования можно отнести к особенностям случая, поскольку в лечении данной патологии предпочтение отдается миотомии и аортокоронарному шунтированию как более эффективным. Срок наблюдения за пациентом составил 7,5 года; после проведения стентирования туннельного сегмента ПМЖА приступы стенокардии не возобновились.

Заключение. Описанный клинический случай демонстрирует роль ММ в развитии ишемии миокарда. При своевременной диагностике нарушения коронарного кровотока, успешной реваскуляризации и адекватной фармакотерапии согласно действующим клиническим рекомендациям прогноз благоприятный.

Ключевые слова: *миокардиальный мостик; ишемическая болезнь сердца; ИБС; чрескожное коронарное вмешательство; клинический случай*

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Role of Myocardial Bridging in Myocardial Ischemia: Case Report

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ABSTRACT

INTRODUCTION: Myocardial bridging (MB) is considered a benign pathology, however, its existence is often associated with angina pectoris, myocardial infarction, ventricular tachycardia and sudden cardiac death. The clinical significance of MB is determined by the dynamic stenosis of the coronary artery, which depends on the rate and strength of heart contractions and is poorly visualized by instrumental methods, which impairs timely diagnosis and early administration of the adequate treatment. A clinical case of a combined damage to the coronary bed in a 58-year-old patient is presented: MB of the anterior interventricular artery (AIVA) causing dynamically significant narrowing of the tunnel artery and hemodynamically insignificant atherosclerotic lesion of the coronary arteries. MB was typically located in the mid-segment of the AIVA. The diagnosis of the anomaly of the coronary bed was established after coronary angiography: MB of AIVA led to stenosis of the tunnel segment to 80% and was the cause of angina attacks. Stenting of the AIVA with a drug-coated stent was performed. The effectiveness of the stenting can be referred to a peculiar feature of this case, since in the treatment of this pathology the preference is given to myotomy and coronary artery bypass grafting as more effective methods. The follow-up period was 7.5 years; after stenting of the tunnel segment of the AIVA the anginal attacks did not recur.

CONCLUSION: The described clinical case demonstrates the role of MB in the development of myocardial ischemia. With timely diagnosis of the coronary blood flow disorders, successful revascularization and adequate pharmacotherapy in accordance with the current clinical recommendations, the prognosis is good.

Keywords: *myocardial bridging; coronary heart disease; CHD; percutaneous coronary intervention; case report*

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ECO•VECTOR

LIST OF ABBREVIATIONS

AIVA — anterior interventricular artery
AP — arterial pressure
AV — aortic valve
CA — coronary artery
CAG — coronary angiography
ECG — electrocardiogram
EchoCG — echocardiography
ES — extrasystole
HR — heart rate
LA — left atrium
LV — left ventricle
MB — myocardial bridge, myocardial bridging
MSCT — multispiral computed tomography
MV — mitral valve
RCH — Regional Clinical Hospital
RR — respiratory rate

INTRODUCTION

Myocardial bridging (MB) is a commonest congenital anomaly of coronary arteries (CA, in which a segment of CA is surrounded by the myocardium. The artery covered with myocardium, is called a *tunneled artery*.

MB was first discovered by Reyman in 1737 in autopsy, and its angiographic presentation was described by Portmann and Iwig in 1960. MB most often occurs in the middle segment of the anterior interventricular artery (AIVA). With this, due to anatomic, physiologic and hemodynamic peculiarities, the AIVA is also most vulnerable to atherosclerotic lesion [1]. The existence of MB in the middle segment of the AIVA is an independent predictor of formation of atherosclerotic plague proximal to MB [2].

It is difficult to estimate the prevalence of MB, which is associated with use of various visualization techniques. According to the literature, the prevalence of MB ranges from 3.5% to 58% [3]. Myocardial bridges exist in almost a third of adults, but not all of them lead to clinically significant narrowing of the CA and symptoms of impaired myocardial perfusion. In the work by R. Nakanishi, et al. myocardial bridging was detected in coronary angiography (CAG) in less than 5% of cases [4]. Russian researchers M. N. Kapustinski and I. G. Menshikova in their work note that after examination of 1,640 patients with coronary heart disease, in 4.7% of them MB was detected in CAG, which caused from 40% to 100% compression of the CA and had cause-and-effect relationship with acute coronary syndrome in 85.7% of cases [5]. The provoking tests in patients with angiographically 'normal' CA can increase systolic compression and detect MB in even more number of patients [6, 7].

From the point of view of pathophysiology, MB causes narrowing of the lumen of the artery in systole and absence of the adequate dilation in diastole. Hemodynamic load promotes *formation of an atherosclerotic plaque* in the place of transition of the CA to the tunneled segment. It was found by international researchers that the pressure in the segment proximal to the MB is higher than in the aorta, which creates turbulent blood flow and increases fluid shear stress in vessels that accelerates *development of atherosclerotic lesion proximal to MB* [8]. The intravascular ultrasound and Doppler examination showed that compression of the CA inside MB is not only a systolic event, but also persists in diastole and is associated with reduction of the coronary flow reserve. The reduction of the coronary blood flow in diastole may also favor myocardial hypertrophy, endothelial dysfunction [9] and spasm of coronary arteries.

MB is considered a *benign* pathology, however, its presence is often associated with angina pectoris, myocardial infarction, ventricular tachycardia, sudden cardiac death. The tunneled artery usually does not manifest with symptoms until the third decade of life despite the existence of the anomaly since birth. Clinical manifestations of MB can be atypical without any correspondence between the expression of symptoms and the length or depth of the tunneled segment of the CA or the degree of systolic compression. The ECG data of MB at rest are non-specific [10]. The clinical significance is also determined by the presence of dynamic stenosis of the CA which depends on the heart rate and strength of heart contractions and is poorly visualized by instrumental methods, which impedes the timely diagnosis and early administration of adequate therapy.

Here, we report a clinical case of MB of the AIVA with atherosclerotic lesion of the CA in a patient of 58 years, which illustrates the peculiarities of clinical manifestations, diagnosis and prognosis.

Case Report

Patient L., 58 years old, was admitted to the Regional Clinical Hospital (RCH) of Ryazan on 2014, November 28 with ***complaints*** of compressing retrosternal pain in walking and sometimes at rest, with irradiation to the left arm lasting about 10 minutes and rapidly disappearing after elimination of the load.

Medical history: the patient was suffering from arterial hypertension for about 10 years, was taking antihypertensive drugs irregularly, the maximal level of arterial pressure (AP) was 170/110 mm Hg. Retrosternal

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pain of compressing quality had been present for about 7 years appearing in walking for 300–400 m. Since the end of December 2010, the frequency of pain increased, and it started to occur in 100 m distance walk. In connection with this, in January 2011 the patient was for the first time hospitalized in RCH for unstable angina. Discharged on 2011, January 31 with improvement. Recommendations included intake of lisinopril, amlodipine, acetylsalicylic acid, clopidogrel and atorvastatin. In February 2012, he was re-hospitalized in the RCH with impairment (complaints of compressing chest pain and a feeling of lack of air in walking for up to 100 meters).

Echocardiography (EchoCG, 14/02/2012): moderate myocardial hypertrophy, enlargement of the cavity, type I disorder of the diastolic function of the left ventricle (LV). Dilatation of the left atrium (LA). Moderate insufficiency of the mitral valve (MV), aortic valve (AV). Signs of aortic atherosclerosis. Abnormal location of the chord of the LV.

Holter ECG monitoring (14/02/2012): sinus rhythm, heart rate (HR) 57–122 beats per minute. Single ventricular and supraventricular extrasystoles (ES). A single episode of group supraventricular ES. No ischemic changes.

The patient refused CAG.

Multispiral computed tomography (MSCT) of the CA (15/02/2012) — no data for atherosclerosis of the CA.

The patient was discharged with improvement on 2012, February 20.

Life history: denied bad habits, allergic history without peculiarities. Positive heredity for arterial hypertension (sister, mother — from 47 years of age), father suffered a myocardial infarction at the age of 50, brother had type 2 diabetes mellitus. Past diseases and operations: acute respiratory viral infections, appendectomy, duodenal ulcer (last exacerbation 8 years ago).

The present impairment started on 2014, November 28, when angina attacks became more frequent again, and tolerance to exercise decreased.

On admission (28/11/2014) general condition satisfactory, clear consciousness. No edema. Height 180 cm, weight 98 kg, body mass index 30.25 kg/m^2. Respiratory rate 16 per minute, vesicular breathing in lungs, no crackles. Heart rate (HR) 60 beats per minute. AP — 160/90 mm Hg. Boundaries of the deep cardiac dullness displaced to the left by 1.5 cm. In auscultation, the heart sounds diminished, rhythm regular. The abdomen soft, painless. Stools once a day. Costovertebral symptom negative on both sides. Urination free, painless, diuresis sufficient.

Clinical blood analysis: erythrocytes — 4.7 × 10^{12}/l, hemoglobin — 145 g/l, platelets — 139 × 10^9/l, leukocytes — 6.8 × 10^9/l, eosinophils — 1%, polymorphonuclear neutrophils — 60%, lymphocytes — 24%, monocytes — 12%.

Clinical urine analysis: without pathology.

Biochemical blood test: total protein — 70 g/l, total bilirubin — 10.0 μmol/l, conjugated bilirubin — 2.0 μmol/l, unconjugated bilirubin — 8.0 μmol/l, aspartate aminotransferase — 18 Un/l, alanine aminotransferase 22 Un/l, urea — 4.8 mmol/l, creatinine — 103 μmol/l, potassium — 4,1 mmol/l, sodium — 140.0 mmol/l, cholesterol — 4.17 mmol/l, low density lipoproteins — 2.82 mmol/l, high density lipoproteins — 1.08 mmol/l, triglycerides — 2.37 mmol/l.

Glomerular filtration rate calculated on CKD-EPI — 69 ml/min/1.73m^2.

Troponin I negative.

ECG: with sinus rhythm, alterations of the myocardium of the lower wall of the LV were recorded as downsloping ST segment depression with negative T wave in III, aVF leads (Figure 1).

EchoCG (03/12/2014): hypertrophy of the myocardium and dilatation of the LV. Impaired diastolic function of the LV. Enlargement of the LV, of the right atrium. Moderate insufficiency of the MV, AV. Mild pulmonary hypertension. Signs of atherosclerosis of the ascending section of the aorta, calcification of fibrous ring of the AV.

Holter monitoring of ECG (05/12/2014): sinus rhythm with HR 65–139 beat/min. Rare supraventricular ES (in total 12). Subendocardial alterations of ischemic character: an episode of ST segment displacement significant in II, V5, V6 (Figure 2). Total duration of episodes of ischemic ST displacement — 14 minutes, during episodes of Ischemic ST displacement the patient two times experienced retrosternal pain.

Coronary angiography (11/12/2014). Left type of circulation. The left CA trunk without changes. MB in the middle AIVA segment narrowing the lumen of the artery to 80% in systole (Figure 3). Stenosis of the orifice of the 1st diagonal branch to 60%, stenosis of the middle segment of circumflex artery to 50%. The right CA without changes.

Percutaneous transluminal coronary angioplasty and ***endoprosthetics*** of MB of the AIVA with drug-coated stent Endevaor 3.5 × 24 mm was performed. Postdilatation with balloon 3.0–20 mm. Blood flow TIMI III.

In the course of treatment, the condition improved, no pain in the heart. AP within 120/80 — 130/80 mm Hg. HR at rest 60–65 beat/min.

Final clinical diagnosis:

Main disease: Coronary heart disease: unstable angina with outcome to I functional class stable exertion angina. Stenotic myocardial bridging of the AIVA, endoprosthetics of the AIVA on 2014, December 11.

Underlying disease: III stage essential hypertension, controlled arterial hypertension, very high risk of cardiovascular complications.

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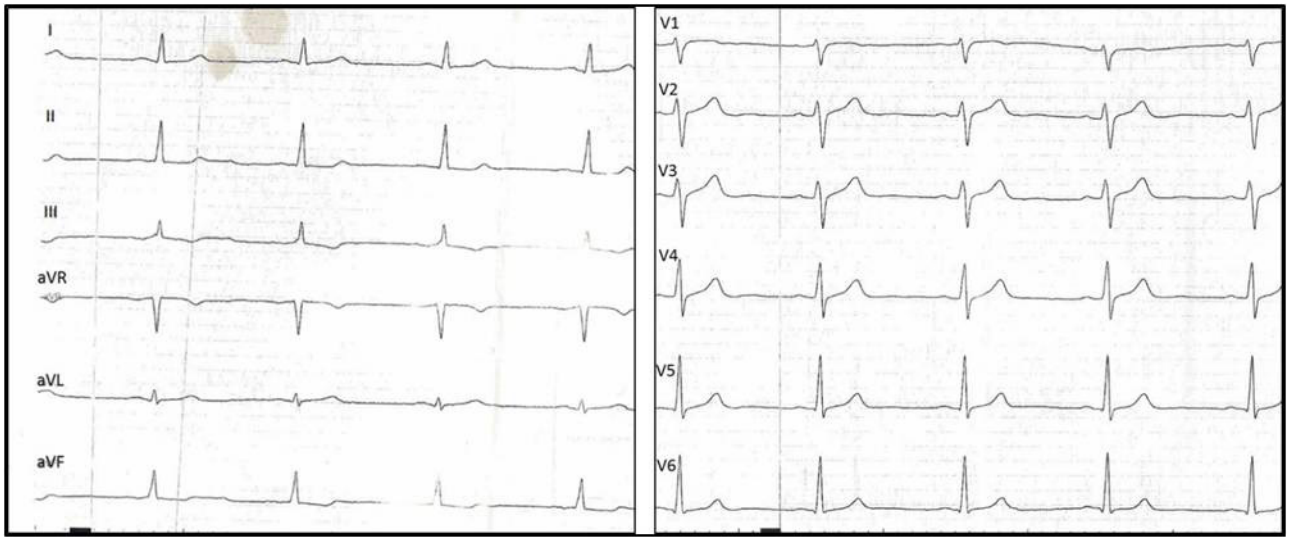

Fig. 1. Electrocardiogram of patient L. on admission 2014, November 28.

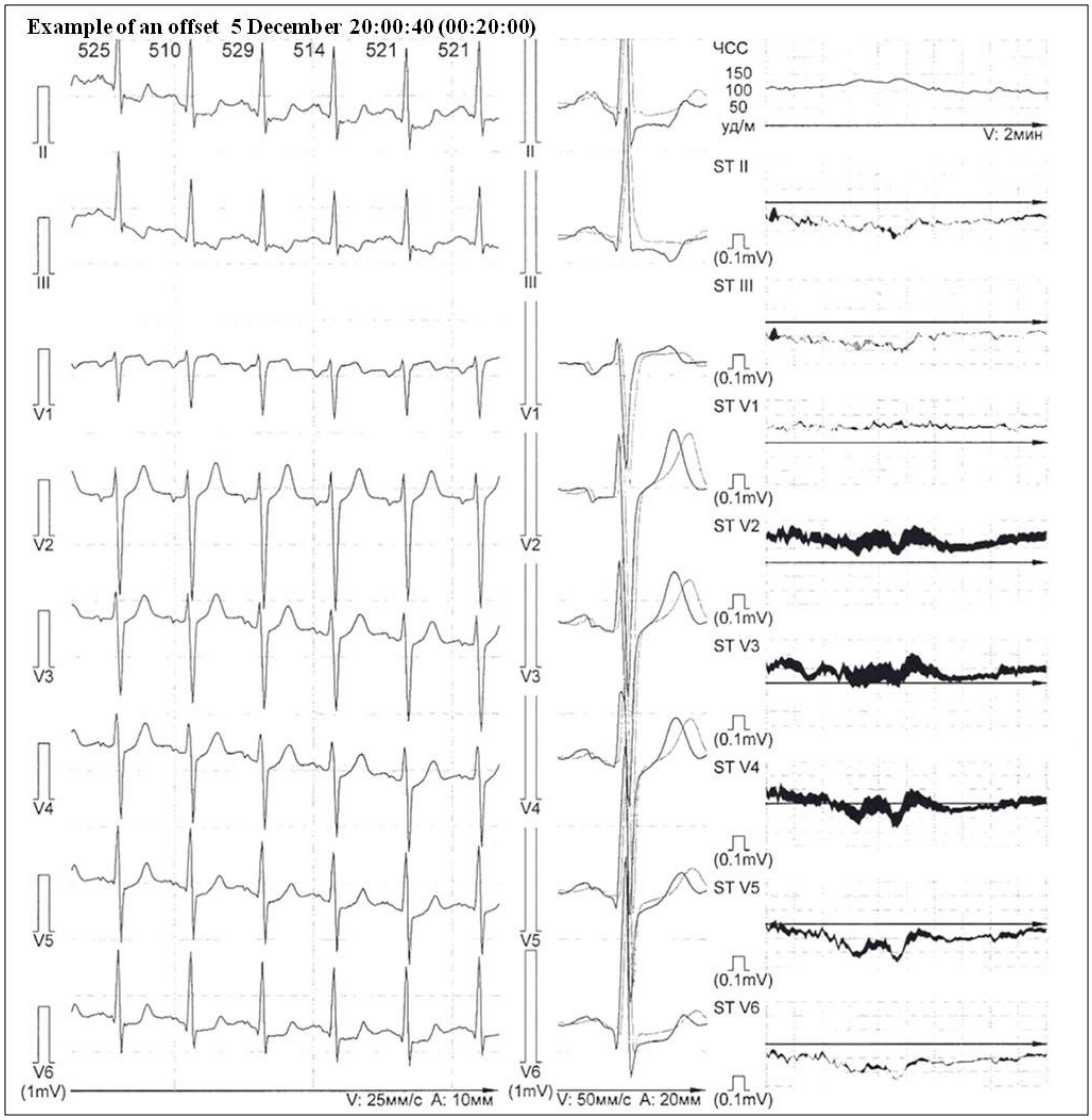

Fig. 2. Holter monitoring of electrocardiogram of patient L. (05/12/2014): subendocardial alterations of ischemic character.

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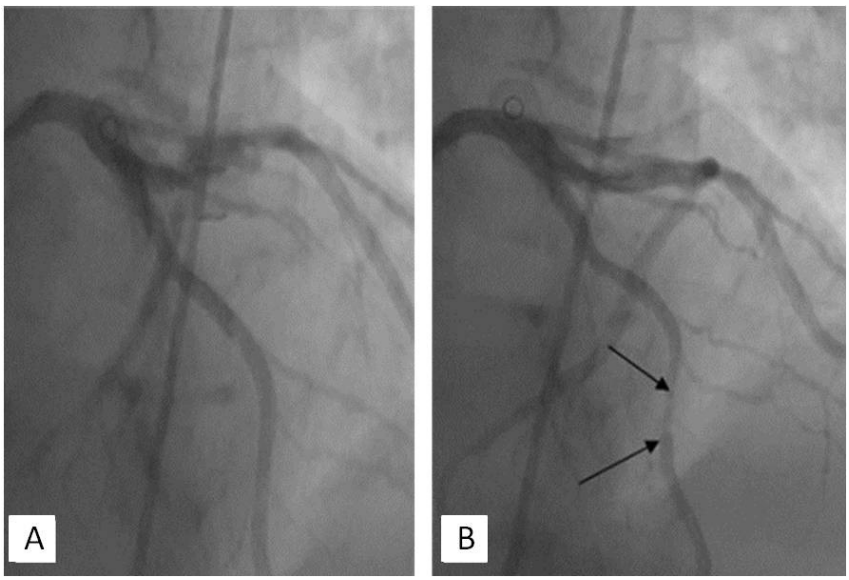

Fig. 3. Coronary angiography (11/12/2014) of patient L. A myocardial bridge in the middle segment of the anterior interventricular artery (diastole (A), systole (B)) with compression of the artery in systole to 80% (arrows).

Complications of the main disease: I stage chronic heart failure, II functional class.

Comorbid diseases: I degree obesity. Duodenal ulcer in remission stage.

Recommended medical treatment (all *per os*): perindopril 10 mg/day, amlodipine 10 mg/day, acetylsalicylic acid (in combination with magnesium hydroxide) 75 mg/day, clopidogrel 75 mg/day (12 months), atorvastatin 40 mg/day.

The follow-up period was 7.5 years; after stenting of the tunneled segment of the AIVA, angina attacks did not resume. Figure 4 shows the patient's ECG of 2022, June 17: sinus rhythm, QRST without negative dynamics.

DISCUSSION

In discussion of the clinical case, it should be noted that location of MB in the middle segment of the AIVA is the most typical location of this CA anomaly [11]. The clinical picture in the described case was characterized by typical attacks of exertion angina, while i*n most cases MB does not manifest clinically*. The patient had the following traditional risk factors for cardiovascular diseases: male gender, age, I degree obesity, positive heredity. ECG taken at rest was non-informative in diagnosis of ischemia, but in Holter ECG monitoring episodes of ST segment depression below isoline were recorded during anginal pain attacks. MSCT of the CA conducted in 2012 appeared non-informative in diagnosis of MB, no data for coronary atherosclerosis were neither obtained. However, according to the literature, *the probability for detection of MB in MSCT of CA is higher than in traditional CAG* [12]. Perhaps, in our clinical observation, non-informative character of MSCT of CA could be associated with proficiency of the personnel, with correctness of the procedure (for example, the quality of the test with breath holding).

Today, *CAG is considered a universally recognized diagnostic standard for MB*. MB is identified by transient systolic compression of a branch of the CA, the phenomenon termed *milking effect*. Systolic compression of the involved epicardial CA is the main angiographic finding.

It was possible to visualize MB in patient L. in invasive CAG, which revealed not only hemodynamically significant stenosis of the AIVA to 80% in systole due to the presence of MB, but also hemodynamically insignificant stenoses of the CA (stenosis of the orifice of the 1st diagonal branch to 60%, stenosis of the middle segment of the Cx artery to 50%). A similar combination of MB and atherosclerotic stenoses of the CA is described by S. A. Chepurnenko, et al. in their work; however, in this study only hemodynamically insignificant stenoses due to MB were found: in 27.27% of cases the lumen of the CA tunneled segment narrowed in myocardial contraction by 40%–50%, in 63.63% of cases — by 30% and less [11].

It is also important to discuss the patient management tactics in case of MB detection. The asymptomatic MB with hemodynamically insignificant stenosis of the CA without atherosclerotic lesion of the CA requires observation of a patient, and in case of presence of atherosclerotic

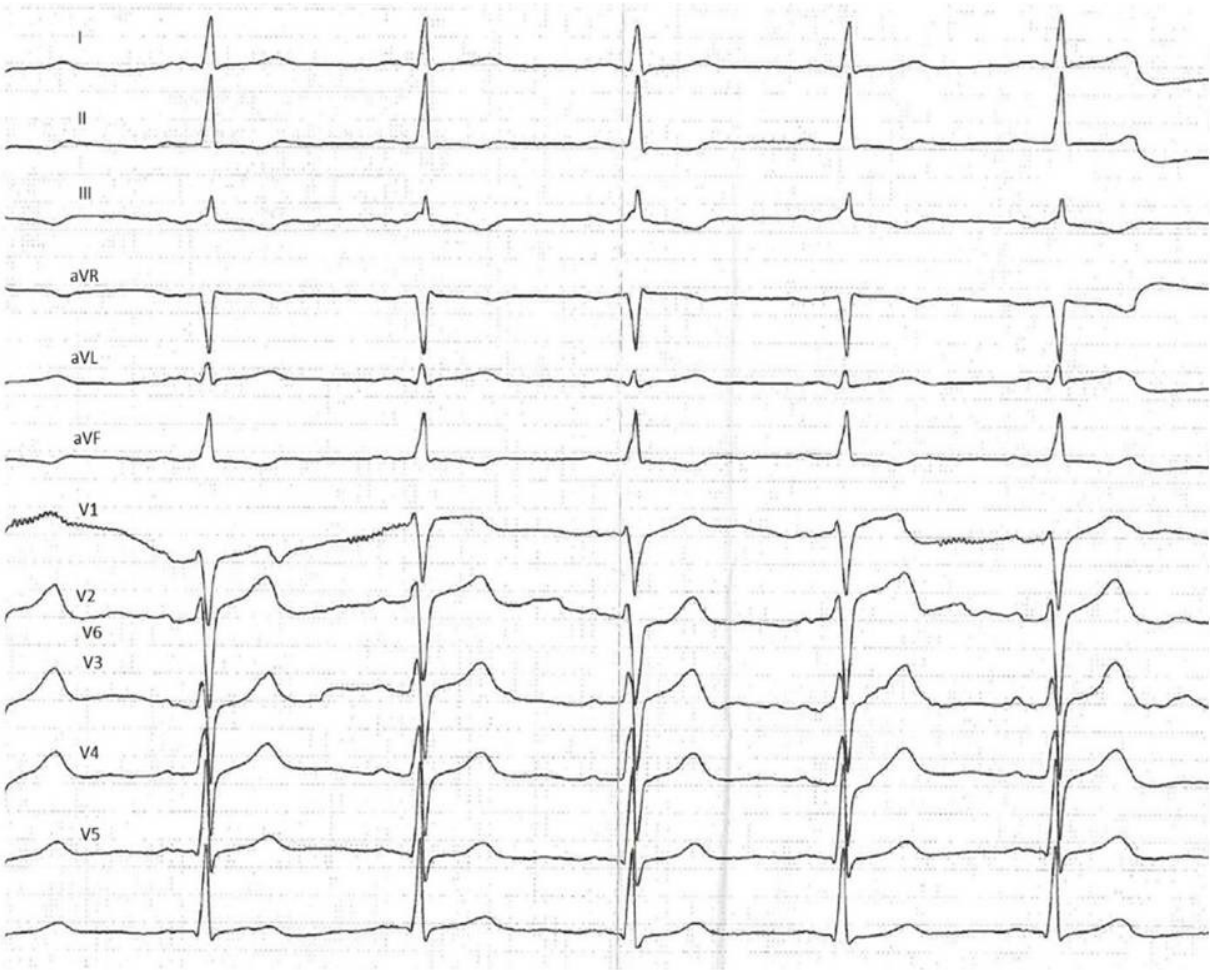

Fig. 4. Electrocardiogram of patient L. in dynamics (17/06/2022).

lesion the treatment is required according to the current Clinical Recommendations taking into account the fact that, *according to the results of CAG, nitrates increase the extent of systolic narrowing of the tunneled segment of the CA and may worsen clinical symptoms*. No randomized studies were conducted for comparison of pharmacological treatment with percutaneous coronary intervention in patients with MB. A method of choice is pharmacological treatment with β-blockers or antagonists of slow calcium channels. To eliminate MB-associated stenosis of the CA, it is recommended the 2nd generation of drug-coated stents be used and bioresorbable stents avoided; of critical significance in stenting of the tunnel segment is intravascular imaging to prevent excessive or insufficient opening of the stent [13, 14]. Insertion of a stent in the tunneled segment of the CA can lead to perforation of the CA, restenosis of the stent, its destruction, etc. [14–17]. However, stenting can be effective in evident atherosclerotic lesion of the CA with hemodynamically significant narrowing of MB segment or in the absence of good distal seating area for stent implantation [15, 17]. *Patients with MB symptoms, reluctant to pharmacotherapy, should undergo surgical treatment*, such as myotomy and coronary artery bypass grafting [18]. Myotomy is a preferred procedure due to its safety and satisfactory results [19].

Observational study of the frequency of the intrastent restenosis after implantation of a drug-coated stent in patients with MB with use of optic coherent tomography showed that *stent restenosis is associated with the severity of initial systolic stenosis in MB*. Here, mild stenosis of MB is harmless and is effectively eliminated with a drug-coated stent which partially or fully covers the tunneled segment of the CA.

In the clinical case under discussion, a drug-coated stent was used, which led to complete disappearance of anginal attacks during 7.5 years of postoperative follow-up of the patient.

Of the drugs that affect the prognosis, adherence to statin therapy for coronary heart disease is the least

[20], but it is adherence to drug therapy that permits to slow down progression of the atherosclerotic process in the CA. A long-term prognosis for patients with isolated MB is usually favorable. Thus, five-year survival rate of 81 patients with MB (mean age 46 years) was 97.5%, and none of the two deaths was associated with MB [21]. In another group consisting of 61 patients (mean age 50 years) with MB in the AIVA, 11-year survival rate was 98%, and none of the deaths was provoked by the presence of MB [22]. Certainly, a combined lesion of the coronary bed: MB which gives a hemodynamically significant narrowing of the tunnel artery, and hemodynamically insignificant atherosclerotic lesion of the CA, makes the prognosis more serious.

CONCLUSION

The described case of a combined lesion of the coronary bed in a 58-year-old patient: a myocardial bridge of the anterior intraventricular artery resulting in hemodynamically significant narrowing of the artery to 80% and a hemodynamically insignificant lesion of coronary arteries demonstrated the role of a myocardial bridge in the development of myocardial ischemia.

The effectiveness of stenting of the tunneled artery can be referred to peculiarities of the case, since in surgical treatment of this pathology the preference is given to myotomy and coronary artery bypass grafting as more effective methods. A long follow-up period (7.5 years) with absence of angina attacks shows a good prognosis in case of timely diagnosis of coronary artery anomalies and adequate pharmacotherapy in accordance with the current clinical recommendations.

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Финансирование. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.
Конфликт интересов. Авторы заявляют об отсутствии конфликта интересов.
Согласие на публикацию. В статье использованы обезличенные клинические данные пациента в соответствии с подписанным ими информированным согласием.
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