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КЛИНИЧЕСКИЙ СЛУЧАЙ УСПЕШНОГО ЛЕЧЕНИЯ ДИССЕКЦИИ КОРОНАРНОЙ АРТЕРИИ И ТРОМБОЗА ЛЕВОГО ЖЕЛУДОЧКА У МОЛОДОГО МУЖЧИНЫ — НОСИТЕЛЯ ГЕНА ФАКТОРА V ЛЕЙДЕНА С ТРАНЗИТОРНОЙ ИШЕМИЧЕСКОЙ АТАКОЙ

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A clinical case of successful treatment of coronary artery dissection and left ventricular thrombosis in a young man carrier of factor V Leiden gene and transient ischemic attack

Резюме

Одной из причин транзиторной ишемической атаки или ишемического инсульта являются объемные образования левых отделов сердца, в том числе, интракардиальные тромбы. Одним из предрасполагающих факторов тромбообразования является наличие Лейденской мутации, как наиболее распространенной формы наследственной тромбофилии в европейской популяции. В то же время, одной из причин острой сосудистой катастрофы может являться спонтанная диссекция коронарных артерий. Особую сложность представляет дифференциальная диагностика, требующая дополнительных методов обследования (внутрисосудистое ультразвуковое исследование, оптическая когерентная томография) помимо проведения коронарографии. Представленный клинический случай описывает диагностику и успешное лечение спонтанной диссекции передней межжелудочковой артерии, тромбоза левого желудочка, выявление мутации гена фактора V Лейдена у молодого мужчины с анамнезом транзиторной ишемической атаки.

Ключевые слова: *тромб левого желудочка; опухоль сердца; образование сердца; тромбэктомия; транзиторная ишемическая атака*

Конфликт интересов

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

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Abstract

One of the causes of transient ischemic attack or ischemic stroke are mass formations in the left heart. One of the predisposing factors for thrombosis is the presence of the Leiden mutation, as the most common form of hereditary thrombophilia in the European population. At the same time, spontaneous dissection of the coronary arteries (SCA) can be one of the reasons of an acute vascular accident. Of particular difficulty is differential diagnosis, which requires additional examination methods (intravascular ultrasound, optical coherence tomography) in addition to coronary angiography. The presented clinical case describes the diagnosis and successful treatment of spontaneous anterior interventricular artery dissection, left ventricular thrombosis, detection of factor V Leiden gene mutation in a young man with a history of transient ischemic attack.

Key words: *left ventricular thrombus; heart tumor; heart formation; thrombectomy; transient ischemic attack*

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The authors declare no conflict of interests

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IVUS — intravascular ultrasound, DB — diagonal branch, CAG — coronary angiography, LV — left ventricle, MRI — magnetic resonance imaging, ACS — acute coronary syndrome, AIVA — anterior interventricular artery, SCAD — spontaneous coronary artery dissection, TIA — transient ischemic attack, PE — pulmonary embolism, EF — ejection fraction, HR — heart rate.

Introduction

Thrombotic mass lesions located in left heart chambers are one of the causes of transient ischemic attacks (TIA) or ischemic strokes. Depending on the clot morphology, remote risks for embolic and cardiovascular events increase up to 22–37 % [1, 2]. Leiden mutation is one of the predisposing factors for clotting. It is one of the most common forms of hereditary thrombophilia in the European population [3]. Leiden mutation belongs to "classic" hereditary thrombophilias with antithrombin deficiency. Deep vein thrombosis and pulmonary embolism (PE) are considered the most common manifestations of thrombotic complications, though other thrombotic locations are possible, including cerebral, portal, and hepatic ones [4, 5].

At the same time, spontaneous coronary artery dissection (SCAD) may be considered one of the causes of vascular accidents. According to the scientific literature, 1–4 % of all ACS cases are associated with SCAD [6, 7]. Meanwhile, true SCAD incidence is unknown, accounting for non-specific clinical signs and difficult diagnosis. The disease is more common among young females; less than one third of cases is reported in males [8, 9]. The purpose of Inspire International Community supported by the The Women Heart Support Community is the collection and distribution of data regarding the female patients suffered from SCAD. Coronary angiography (CAG) in SCAD reveals the following: intimal flap in the arterial lumen, contrast extravasation, true and false vascular lumina (double vascular lumen), vascular lumen shrinkage due to hematoma. The J. Saw classification defines three SCAD types: type 1 with evident arterial wall contrasting; type 2 with diffuse stenosis of variable degree; type 3 mimicking atherosclerosis [10]. However, CAG data are not always enough for correct diagnosis. Differential SCAD search may require additional examination methods (intravascular ultrasound (IVUS), optical coherence tomography). Clinical cases with predisposing factors and patient comorbidities are especially difficult. Single SCAD cases associated with factor V (Leiden) mutation are described in the scientific literature. Hereby we present a proper case study describing the treatment of spontaneous anterior interventricular artery dissection with left ventricular (LV) thrombosis and TIA in a young man carrying the mutation of factor V (Leiden) mutation gene.

Case Study

The male patient A., 36 years old, was hospitalized on January 10, 2022 into the Cardiac Surgery Department No. 1 of the Federal State Budget Institution "Federal Center of Cardiovascular Surgery", Ministry of Health of the Russian Federation (Astrakhan), complaining of dyspnea on mild physical exertion, worsening fatigue.

History: the patient has been sick for about a year, when he developed dyspnea on physical exertion, pain in calf muscles during prolonged walking, worsening fatigue. According to the patient (no medical documents available), he was hospitalized in 2017–2018 due to deep vein thrombosis of the right upper extremity, right great saphenous vein; he was treated conservatively with short-term oral anticoagulants. The patient's current condition was worsening since January 5, 2022, when during car driving he felt weakness in the left extremities and the left side of the face. The symptoms regressed 10–15 min later; the neurologist considered this a TIA. The patient underwent transthoracic echocardiography at place of residence, where the mass lesion was detected in the LV. The patient was referred for hospitalization to the Federal State Budget Institution "Federal Center of Cardiovascular Surgery", Ministry of Health of the Russian Federation (Astrakhan) with the diagnosis of mass LV lesion. On admission the patient took the following medications: rivaroxaban 20 mg, acetylsalicylic acid 100 mg, clopidogrel 75 mg. The patient had significant cardiovascular family history — his father had a stroke. Bad habits: 10-year smoking history (previously) with a 5-year break; at the time of hospitalization, the patient was smoking e-cigarettes. The patient did not suffer from COVID-19.

Preliminary diagnosis on admission: LV lesion. Coronary artery disease (confirmation required). TIA (January 5, 2022). Chronic heart failure, Stage 2A, NYHA Functional Class II.

On admission, the overall condition was satisfactory. The patient's consciousness was clear. Hypersthenic constitution (body mass index 34.3 kg/m^2). The skin was clear, of physiological color, without cyanosis. Body temperature: 36.7 °C. Oxygen saturation: 98 %. Pulmonary auscultation revealed vesicular breathing with no rales; respiratory rate was 16/min. Cardiac auscultation revealed regular rhythm with the heart rate (HR) of 68/min; cardiac sounds were muffled, no pathological murmurs were auscultated. The pulse in radial arteries was symmetric, of satisfactory filling; no deficit was reported. Blood pressure: 140/90 mm Hg in both arms. No carotid bruits were detected. The tongue was moist and clear. The abdomen was soft and nontender on superficial palpation. No peripheral edema was detected.

The complete blood count on admission revealed lymphocytosis (48.4 %). The homocysteine level in blood was 13.3 mmol/L (reference values 5.46–16.2 mmol/L); anti-phospholipid antibodies did not exceed 10 U/mL. Antithrombin III activity was 85 % (reference values 83–128 %), Protein C concentration 124 % (reference values 70–140 %), free Protein S concentration 97.4 % (reference values 74.1–146.1 %). The urinalysis on admission revealed proteinuria (0.38 g/L).

Transthoracic echocardiography (January 10, 2022): end-diastolic LV volume 85 mL; end-systolic LV volume 40 mL; LV ejection fraction (EF, Simpson) 55 %; right ventricle: basal part 3 cm; left atrium 3.3 cm; left atrial volume 42 mL. Cardiac chambers were not enlarged. Global myocardial contractility was normal. Local contractility disorders of LV segments: apical LV hypokinesia. A mobile hyperechogenic pedunculated lesion sized

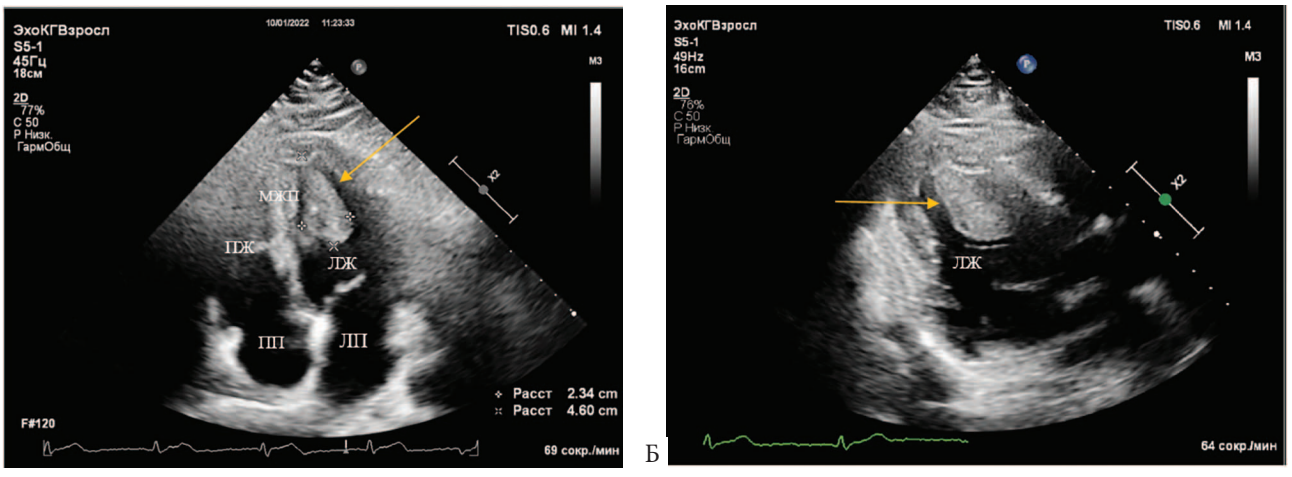


Figure 1. *Transthoracic echocardiography of patient*

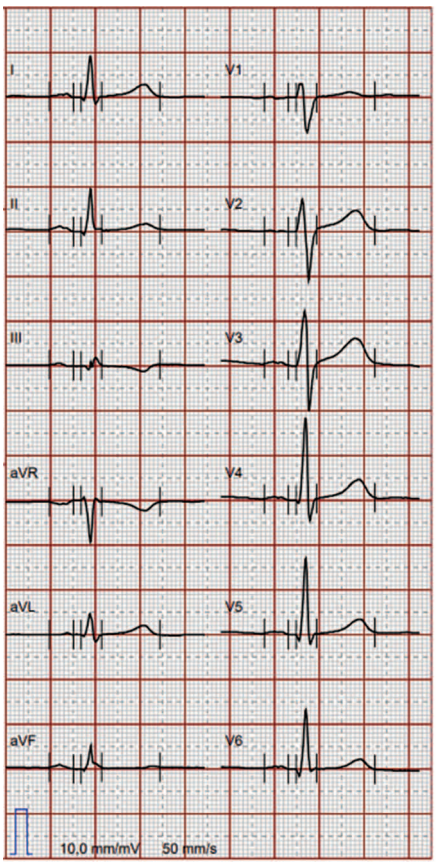
Note. Transthoracic echocardiography. a — apical approach, four-chamber position in the diastolic phase. In the LV cavity, a mobile hyperechogenic mass on a pedicle is visualized, attached to the apex, 4.6 x 2.3 cm in size. b — parasternal approach, position along the long axis in the diastolic phase

4.6 x 2.3 cm and attached to the apex was located in the LV cavity (thrombus? tumor?). Diastolic LV function was not impaired. Systolic right ventricular function was not impaired. Systolic pressure in the pulmonary artery 24 mm Hg. Pericardial and pleural cavities were normal (Fig. 1).

Electrocardiography (January 10, 2022): sinus rhythm with HR 76 beats per minute. Electrical axis of the heart was horizontal (Fig. 2).

GAG (January 10, 2022): eccentric atherosclerotic plaque 40 % in the middle third of the anterior interventricular artery (AIVA) (Fig. 3).

The patient underwent cardiac magnetic resonance imaging (MRI) with intravenous contrast enhancement (January 11, 2022): cine-MRI revealed no decrease in the global LV contractility (EF 56 %). Apical hypokinesis was detected; no other segmental LV contractility disorders were observed. Enhanced trabecularity of anterior and lateral LV walls at the level of middle and apical segments not reaching criteria for trabecular myocardium was detected. The interventricular septum was not thickened at the level of basal and middle segments.



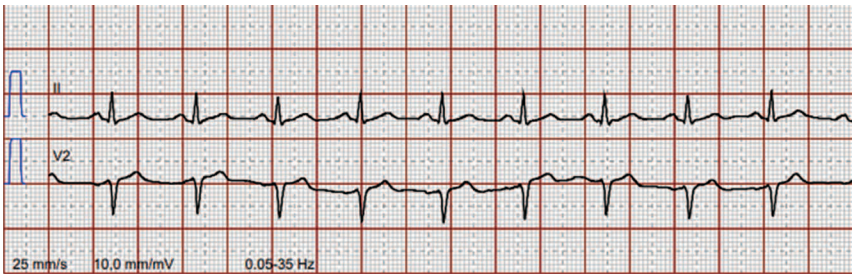


Figure 2. *Electrocardiography of patient*
Note. Sinus rhythm with a heart rate of 76 beats per minute. The electrical axis of the heart is horizontal

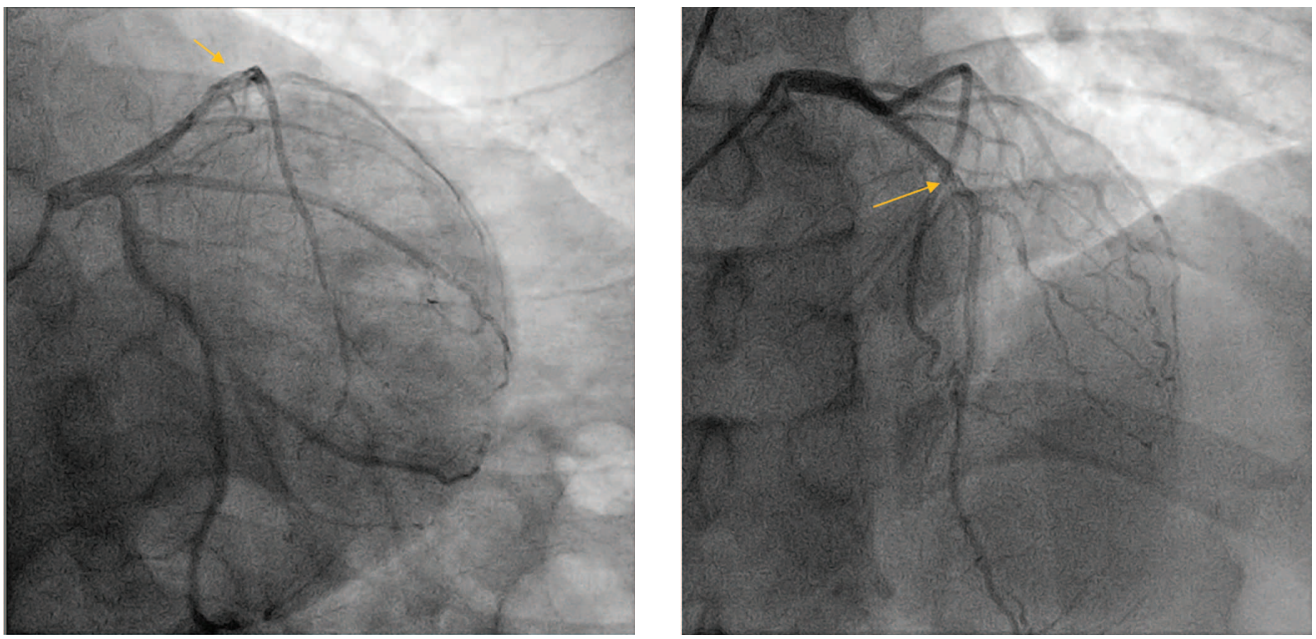


Figure 3. *Coronary angiography of patient at admission*
Note. An eccentric atherosclerotic plaque of 40 % is determined in the anterior interventricular artery (AIA) in the middle third

Myocardial hypertrophy of the apex (1.06 cm) and inferior wall (0.8 cm) of the apical LV segment. A clot sized 4.4×2.4 cm was detected attached to the LV apex. After contrast administration, its transmural accumulation was confirmed in the apex and anterior LV wall (apical segment) (Fig. 4).

Brain MRI (January 11, 2022) was ordered to exclude ischemic foci in the patient's brain. According to its results, no mass lesions, ischemic changes, and intracerebral hemorrhages were detected.

Due to the high risk of embolic complications, thrombectomy (January 13, 2022) was selected.

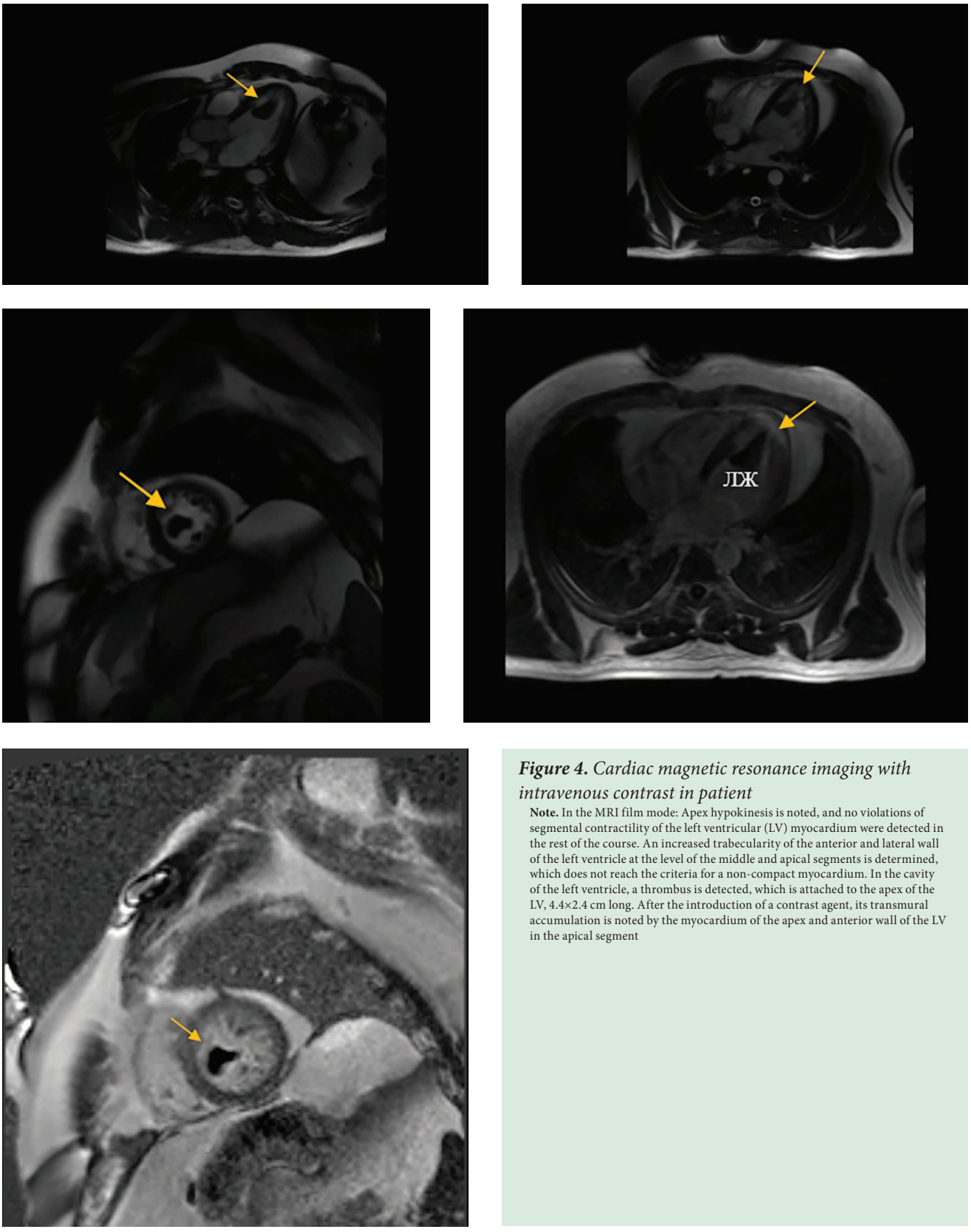


Figure 4. *Cardiac magnetic resonance imaging with intravenous contrast in patient*

Note. In the MRI film mode: Apex hypokinesis is noted, and no violations of segmental contractility of the left ventricular (LV) myocardium were detected in the rest of the course. An increased trabecularity of the anterior and lateral wall of the left ventricle at the level of the middle and apical segments is determined, which does not reach the criteria for a non-compact myocardium. In the cavity of the left ventricle, a thrombus is detected, which is attached to the apex of the LV, 4.4×2.4 cm long. After the introduction of a contrast agent, its transmural accumulation is noted by the myocardium of the apex and anterior wall of the LV in the apical segment

Intraoperative transesophageal echocardiography (January 13, 2022): LV ejection fraction (Simpson) 55 %. Cardiac chambers were not enlarged. Global myocardial contractility was normal. Apical LV hypokinesis was detected. A mobile hyperechogenic lesion on a thin pedicle sized 4.6×2.3 cm and attached to the apex was located in the LV cavity. Mild mitral and tricuspid regurgitation was detected (Fig. 5).

The mass tumor-like lesion was removed from LV via the right-sided mini-thoracotomy in the 4th intercostal space (Fig. 6). Intraoperative revision: a solid piriform LV lesion lacking a capsule, 4 cm long, 1–2 cm wide, fixed with a solid fibrotic based to the myocardium of the LV apex. The lesion was excised within the limits of normal tissues; the section demonstrated the sarcotic structure with solid fibrotic areas.

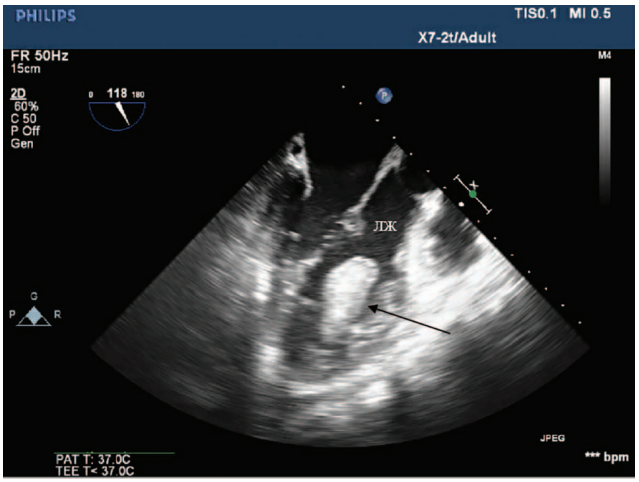


Figure 5. *Intraoperative transesophageal echocardiography of patient*

Note. In the cavity of the left ventricle, a mobile hyperechoic formation on a thin stalk is located, attached to the apex, 4.6×2.3 cm in size

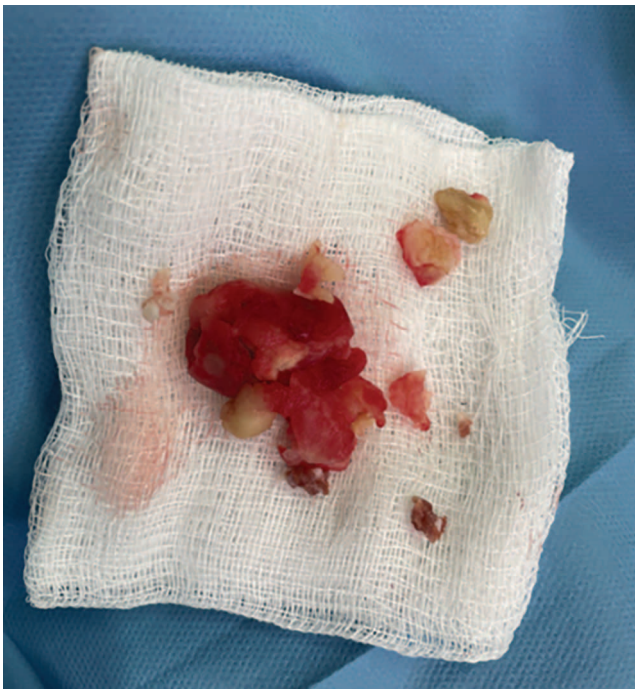


Figure 6. *Intraoperative view of the left ventricular mass*

The histological material was sent to the pathology center for examination. The following conclusion was made: "thrombotic masses with focal organization".

Accounting for thrombotic masses based on MRI and the histology results, CAG was analyzed again to verify the ischemic origin of clinical signs — AIVA dissection was suspected. It was decided to order the intravascular ultrasound (IVUS).

The repeated coronary angiography (Day 6 after the admission) detected a prolonged area of duplicated lumen with narrowing up to 75 % (Fig. 7A) in the AIVA and second diagonal branch. IVUS revealed a duplicated dissection lumen, 45–50 mm long, which was partially thrombotic and formed against the background of atherosclerotic plaque; true arterial lumen in distal areas was completely obturated with the IVUS catheter (Type 1 chronic AIVA dissection with thrombus recanalization) (Fig. 7B). AIVA was stented with drug-eluting stents (PROMUS, Boston Scientific, Massachusetts, USA) 2.75 x 32 mm and 3.5 x 32 mm (Fig. 8). The follow-up CAG confirmed the elimination of AIVA stenosis, with the artery patent along the whole length and preserved distal circulation.

Accounting for the disease history, clinical signs, and examination results, the patient was tested for the gene of Factor V Leiden mutation — a polymorphism variant predisposing to impaired folate cycle (heterozygous form).

Based on the data obtained, the following diagnosis was verified in the patient: "Thrombophilia (Factor V Leiden heterozygote)".

On Day 8, the patient was discharged home with the following clinical diagnosis:

Main disease: LV lesion (thrombus). ICD-10 code: D15.1

Main disease: Non-stenotic atherosclerosis of coronary arteries. Dissection of AIVA and the diagonal branch (DB) (Type 1 based on IVUS data). ICD-10 code: I25.1

Complication of the main disease: TIA (cardioembolic subtype) in the territory of the right middle cerebral artery (January 5, 2022). Heart failure with preserved EF (EF 55 %), Stage 2A (Strazhesko-Vasilenko staging), Functional Class II (NYHA). ICD-10 code: I50.0

Surgery: Excision of the mass LV lesion on January 13, 2022. Percutaneous transluminal coronary angioplasty of AIVA with the implantation of PROMUS stents 2.75 x 32 mm and 3.5 x 32 mm (January 16, 2022).

Concomitant diseases: Venous thromboses of the right upper and lower extremities (in 2017, 2018). Thrombophilia (Factor V Leiden heterozygote).

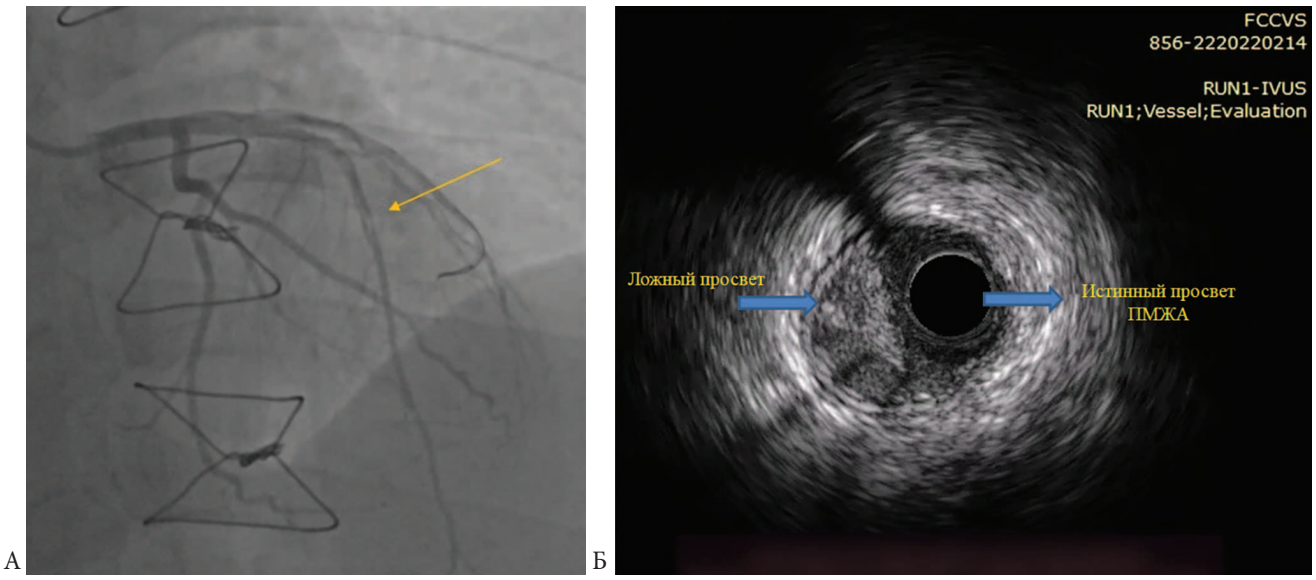


Figure 7. *Coronary angiography and IVUS after surgery (on the 6th day from admission) of Patient A., 36 years old*
Note. А — coronary angiography in the postoperative period (dissection of the anterior interventricular artery type 1 with recanalized thrombus; Б — intravascular ultrasound (IVUS)

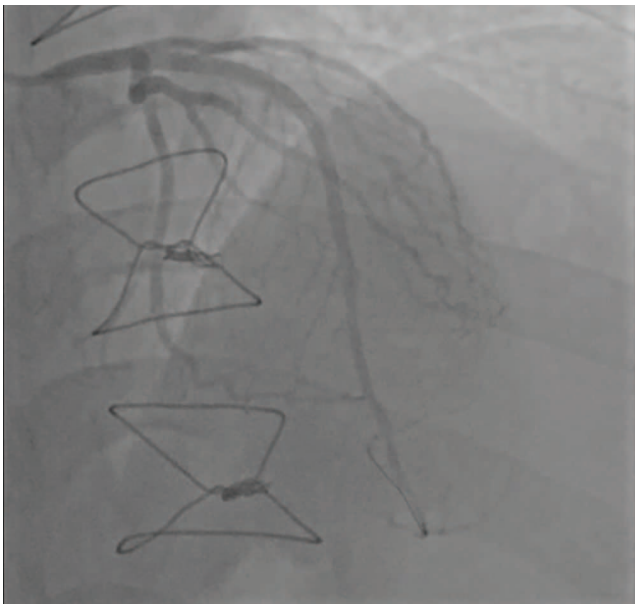


Figure 8. *Control coronary angiography after, anterior interventricular artery stenting of Patient A., 36 years old*
Abbreviations: IVUS, intravascular ultrasound, AVA, anterior interventricular artery

The patient was given the following recommendations on drug therapy: triple antithrombotic therapy for 6 months (rivaroxaban 20 mg + acetylsalicylic acid 75 mg + clopidogrel 75 mg QD), followed by dual antithrombotic therapy for another 6 months (clopidogrel 75 mg + rivaroxaban 20 mg QD), then rivaroxaban monotherapy 20 mg QD should be continued; nebivolol 7.5 mg QD, fixed perindopril 5 mg + indapamide 1.25 mg QD with BP monitoring; spironolactone 25 mg QD (for 1 month with potassium level and glomerular filtration rate monitoring, and subsequent decision on treatment adjustment); rosuvastatin 40 mg QD (with low-density lipoprotein monitoring).

When the patient was followed up later, thromboembolic complications did not relapse, and the patient was in satisfactory condition.

Subsequently, the patient discontinued the therapy administered himself and developed the acute cerebrovascular accident (7 months after discharging). The treatment is currently resumed to a complete extent.

Discussion

If a mass cardiac lesion is suspected in patients, echocardiography is traditionally used as the primary imaging method [11]. However, the differential diagnosis of cardiac lesions based on echocardiography data is complex, especially in young patients. Though transthoracic echocardiography is highly specific (98 %), it is less sensitive than cardiac MRI (29 % vs. 82–88 %) [12]. Contrast-enhanced cardiac MRI provides an additional valuable diagnostic information, which is especially important when planning the surgical intervention; this method specificity reaches 100 % [13]. According to cardiac MRI, LV thrombosis was detected in our patient. In order to exclude thrombosis development (or formation) as CAD/MI complication (or against the background of ischemic events), CAG was arranged in the patient [14]. However, primary CAG evaluation did not demonstrate overt causes of thrombus formation. SCAD was suspected only with repeated expert evaluation.

The latest decade has demonstrated changes in the concept of clinical SCAD evaluation as a non-atherosclerotic non-traumatic cause of acute coronary syndrome (ACS) and sudden cardiac death [6]. SCAD is an uncommon, but rather well-known clinical condition, which may manifest as ACS, syncope, or heart failure. The prevalence of this condition is 0.1–0.24 % among all patients that undergo CAG for stable coronary artery disease or CAG [7]. It is often difficult to detect the coronary artery dissection only with coronary angiography data (as in our case) — such additional examinations as optical coherent tomography or IVUS are required [6, 7, 10].

SCAD treatment options include conservative treatment, early revascularization with percutaneous intervention (PCI), or coronary bypass. PCI is predominant in isolated single-vessel dissection, coronary bypass is more suitable for patients with the dissection of the left coronary artery or several vessels [9].

AIVA stenting was selected in the case study presented, which led to the restoration of the arterial patency. TIA and LV thrombosis in the differential diagnosis of the patient led to the diagnosis "Thrombophilia (Factor V Leiden heterozygote)". Meanwhile, despite the acute thrombosis, administered anticoagulants, and comorbidities, the genetic test (polymerase chain reaction) helps to detect the Leiden mutation [15].

Single cases with SCAD combinations in patients with the Leiden mutation are described in the literature. Tahir Khan et al. (2013) described a rare SCAD case (in the right coronary artery) with the Factor V Leiden mutation in a 31-year-old male [16]. Stents were implanted in that patient. J.H. Joo et al. (2019) published another SCAD and LV thrombosis case in a 64-year-old female with successful conservative management [17].

Further patient management in the clinical case presented presumes long-term antithrombotic treatment. The patient compliance with the treatment is mandatory for the prevention of possible cardiovascular accidents [18]. Treatment compliance does not exceed 50 % with long-term secondary cardiovascular prevention [19]. From the perspective of patient-oriented approach and treatment continuity, high-tech medical care should be followed with outpatient recommendations at place of residence, which is not always possible in real clinical practice. Despite adequate patient awareness about possible unfavorable events with treatment discontinuation, further follow-up revealed unsatisfactory compliance with the recommendations, which led to the acute cerebrovascular accident. Subsequently the patient resumed the administered treatment and is constantly followed up at place of residence.

Conclusion

The described clinical case raises several important and unsolved issues of interdisciplinary patient management, including the inclusion of SCAD into the differential diagnosis of cardiovascular accidents — this presumes combined diagnosis with IVUS. One should account for the comorbidity of patients, including the history of cardiovascular events. Accounting for the age, risk factors, and the character of cardiovascular diseases, timely diagnosis of thrombophilia improves the prognosis in patients. The treatment of patient with thrombophilia after SCAD and intracardiac thrombosis presumes that the correct and timely cardiosurgical tactics is inseparably associated with conservative therapy providing the prevention of cardiovascular events. Therapy continuation, satisfactory treatment compliance, and monitoring the compliance with recommendations form the basis of the comorbid patient treatment that helps to improve the prognosis. Any successful cardiosurgical treatment will not be able to prevent further relapses in the absence of compliance with long-term conservative therapy, which is literally confirmed by the described clinical case.

Вклад авторов:

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