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## ОСОБЕННОСТИ ДИАГНОСТИКИ И ВЫБОРА ТАКТИКИ ЛЕЧЕНИЯ ВПЕРВЫЕ ВЫЯВЛЕННОГО СИНДРОМА БЛАНДА–УАЙТА–ГАРЛАНДА НА ФОНЕ ТЯЖЕЛОЙ СОПУТСТВУЮЩЕЙ СОМАТИЧЕСКОЙ ПАТОЛОГИИ (КЛИНИЧЕСКИЙ СЛУЧАЙ)

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## Features Of Diagnosis and Choice of Treatment Tactics of The First-Diagnosed Bland-White-Garland Syndrome with Severe Coexisting Somatic Pathology (Clinical Case)

### Резюме

Синдром Бланда-Уайта-Гарланда (БУГ-синдром) представляет собой редкую и опасную врожденную аномалию, являющуюся одной из ведущих причин ишемии и инфаркта миокарда у детей. Без своевременного лечения и коррекции заболевание может привести к крайне тяжелым последствиям: до 90 % пациентов умирают в течение первого года жизни. В случае со взрослыми, пациенты, страдающие этим синдромом, могут столкнуться с различными осложнениями, такими как дисфункция левого желудочка, митральная регургитация, бессимптомная ишемия миокарда и инфаркт, а также высоким риском внезапной сердечной смерти. Учитывая, что заболевание редко диагностируется, важно выявлять БУГ-синдром на ранней стадии для превентивного лечения и предотвращения осложнений.

В данной работе описан уникальный случай впервые выявленного БУГ-синдрома у 27-летней пациентки. Помимо порока, женщина страдала от тяжелой сопутствующей патологии — первичного склерозирующего холангита, который осложнялся циррозом печени и гиперспленизмом. Наличие данных заболеваний в значительной степени усложнило выбор лечебной тактики, требуя персонализированного подхода. В связи с высоким риском развития послеоперационных осложнений, предпочтение было отдано малоинвазивной эндоваскулярной эмболизации левой коронарной артерии. Операция показала хорошие результаты и привела к значительному улучшению состояния пациентки, в том числе к достижению компенсации сердечной недостаточности до функционального класса II (NYHA). Представленный случай демон-

стрирует важность индивидуального подхода в лечении редких врожденных пороков сердца у взрослых, особенно при наличии серьезных сопутствующих заболеваний других органов и систем.

**Ключевые слова:** Синдром Бланда-Уайта-Гарланда, сосудистые мальформации, сердечная недостаточность, эндоваскулярная эмболизация

### 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

### Соответствие принципам этики

Пациент дал согласие на опубликование данных лабораторных и инструментальных исследований в статье «ОСОБЕННОСТИ ДИАГНОСТИКИ И ВЫБОРА ТАКТИКИ ЛЕЧЕНИЯ ВПЕРВЫЕ ВЫЯВЛЕННОГО СИНДРОМА БЛАНДА-УАЙТА-ГАРЛАНДА НА ФОНЕ ТЯЖЕЛОЙ СОПУТСТВУЮЩЕЙ СОМАТИЧЕСКОЙ ПАТОЛОГИИ (КЛИНИЧЕСКИЙ СЛУЧАЙ)» для журнала «Архивъ внутренней медицины», подписав информированное согласие

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### Abstract:

ALCAPA (Anomalous Left Coronary Artery from the Pulmonary Artery) is a rare and dangerous congenital anomaly that is one of the leading causes of myocardial ischemia and infarction in pediatric patients. Without timely treatment and correction, the disease can lead to extremely severe consequences: up to 90 % of patients die within the first year of life. In adults, patients suffering from this syndrome may face various complications such as left ventricular dysfunction, mitral regurgitation, asymptomatic myocardial ischemia and infarction, and a high risk of sudden cardiac death. Given that this disease is rarely diagnosed, it is important to detect it early for preventive treatment and avoidance of serious disorders.

This paper describes a unique case of first-diagnosed ALCAPA syndrome in a 27-year-old female patient. In addition to the malformation, the woman suffered from severe concomitant pathology — primary sclerosing cholangitis, which was complicated by liver cirrhosis and hypersplenism. The presence of these diseases significantly complicated the choice of therapeutic tactics, requiring a personalized approach. Due to the high risk of postoperative complications, preference was given to minimally invasive endovascular embolization of the left coronary artery. The operation showed good results and led to significant improvement of the patient's condition, including the achievement of heart failure compensation up to functional class II (NYHA). This case highlights the importance of an individualized approach in the treatment of rare adult congenital heart disease, especially in the presence of serious concomitant diseases of other organs and systems.

**Key words:** ALCAPA Syndrome, vascular malformations, heart failure, endovascular embolization

### Conflict of interests

The authors declare no conflict of interests

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### Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article « Features Of Diagnosis and Choice of Treatment Tactics of The First-Diagnosed Bland-White-Garland Syndrome with Severe Coexisting Somatic Pathology (Clinical Case)» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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BP — blood pressure, BWG-syndrome — Bland-White-Garland syndrome, LV — left ventricle, MRI — magnetic resonance imaging, FC — functional class, FN — physical activity, CHF — chronic heart failure

## Introduction

Bland-White-Garland (BWG) syndrome, also known as ALCAPA (Anomalous Left Coronary Artery from the Pulmonary Artery), is a rare congenital heart disease characterized by the anomalous branching of the left main coronary artery from the pulmonary trunk. This

disease incidence is 1:300,000, constituting 0.25–0.5 % of all congenital birth defects and 0.5 % of all congenital heart diseases [1].

Adequate myocardial perfusion can be maintained in the neonatal period due to the increased pressure in the left pulmonary artery, which is similar to that in the aorta,

and the disease can be quite asymptomatic. Subsequently, during the first weeks of life, the pulmonary artery pressure decreases, which leads to the inadequate myocardial perfusion. Approximately 90 % cases of the disease manifest within the first 2–3 months of life with myocardial ischemia, left ventricular dysfunction, and mitral regurgitation, with clinical signs including acrocyanosis, tachypnea, tachycardia (infantile type of the BWG syndrome). If the collateral circulation is adequate, the arterial blood retrogradely flows from the right coronary artery to the left one, which also causes the myocardial stealing phenomenon; however, this can be subclinical for a long time (adult type of the BWG syndrome). Concerning symptoms, the leading disease manifestations in this patient group include dyspnea, angina, decreased exercise tolerance, and sudden cardiac death [2–5].

Below we present a clinical case report of the newly detected Bland-White-Garland syndrome in an adult patient. This clinical case was peculiar concerning the severe concomitant pathology that affected the patient management tactics.

## Clinical Case Report

The female patient V., 27 years old, visited the emergency department of the General Republican Medical Center, State Budget Healthcare Institution of the Crimea Republic “N.A. Semashko Republican Clinical Hospital” (Simferopol) complaining of palpitations, dyspnea during brisk walking and when climbing to the 2nd floor, tachycardia during exertion. Physical examination demonstrated hepatomegaly, splenomegaly; the patient had a low height and was underweight (height 148 cm; body weight 43.0 kg; body mass index 19.6 kg/m<sup>2</sup>).

Based on the patient’s words, she had been followed up by the cardiologist since childhood due to minimum

aortic regurgitation and minimum pulmonic stenosis. She was exempted from physical education classes in school due to low exercise tolerance. Intensive skin itching also persisted since early childhood. Since teenage years she periodically noted blood pressure (BP) elevation to 170/80 mm Hg (the patient was adapted to BP 110/70 mm Hg), occasionally taking enalapril. During the first pregnancy (in 2021), she developed BP elevation to 180–190/80 mm Hg, methyldopa was administered; thrombocytopenia ( $34.0 \times 10^9/L$ ) was also detected for the first time then. That pregnancy was complicated by eclampsia, and the delivery was arranged via the cesarean section on Week 34. One month after the delivery, the patient was hospitalized with pulmonary edema of unknown origin; she was discharged to continue diuretics, which she took for some time, but cancelled spontaneously.

Subsequently, since spring 2023, the patient noted worsening general condition and dyspnea with decreasing exercise tolerance. In September 2023 she spontaneously visited the emergency department of SBHI RC “N.A. Semashko RCH”, where she was diagnosed with paroxysmal tachysystolic atrial fibrillation, and the patient was hospitalized to the Department of surgical treatment of complex arrhythmias and pacing. Chemical cardioversion was achieved with amiodarone (450.0 mg). During the hospitalization, the patient underwent echocardiography: left atrium 6.1 cm, left atrial volume 150 mL, left ventricular end-diastolic volume 87 mL, left ventricular end-systolic volume 36 mL, interventricular septum thickness 0.9 cm, left ventricular posterior wall thickness 0.9 cm, left ventricular ejection fraction 58 %, Grade 1 mitral regurgitation, bicuspid aortic valve, Grade 1 aortic regurgitation. The laboratory tests demonstrated three-lineage cytopenia, cholestatic syndrome (Table 1).

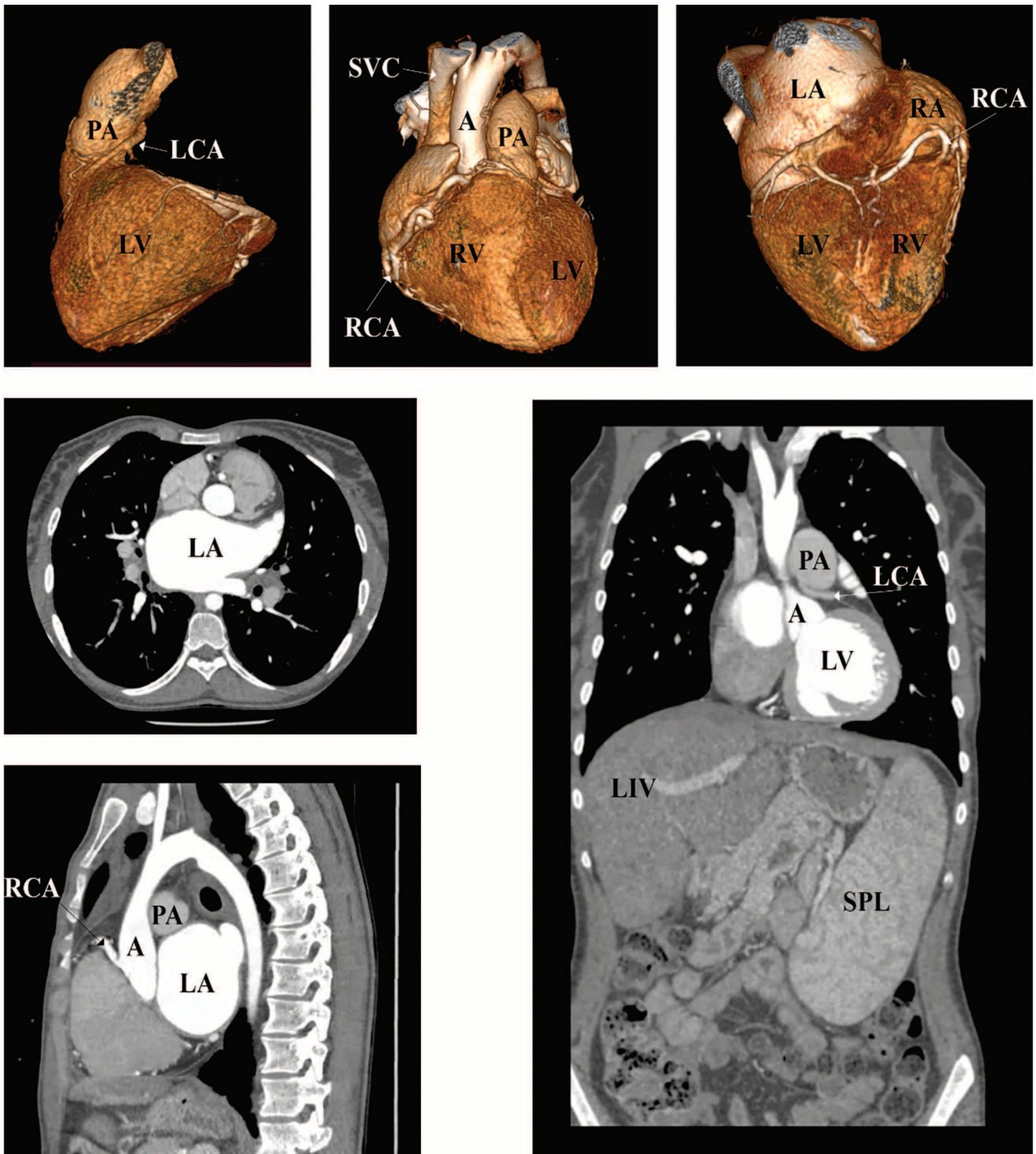
*Table 1. Results of laboratory methods of examination*

Indicator	Result	Standard
<b>Total blood count</b>		
Hemoglobin (g/l)	88,0	120,0-140,0
Hematocrit (%)	29,3	36,0-42,0
Red blood cells ( $10^{12}/l$ )	3,6	3,9-4,7
Platelets ( $10^9/l$ )	94	150-400
Leukocytes ( $10^9/l$ )	1,8	4,0-9,0
<b>Blood biochemical examination</b>		
Alkaline phosphatase (U/L)	128,6	<105,0
GGTP (U/L)	134,2	<32
ALT (U/L)	40,9	32
AST (U/L)	27,3	31
Bilirubin ( $\mu\text{mol}/L$ )	17,0	3,4-17,0

**Abbreviations.** GGTP — gamma-glutamyltranspeptidase, ALT — alanine aminotransferase, AST — aspartate aminotransferase

The patient underwent multislice computed tomography of the chest and abdominal cavity with aortic contrast enhancement: Bland-White-Garland syndrome (anomalous branching of the left main coronary artery from the pulmonary artery); tortuous distal parts of the

right coronary artery (3 mm in diameter) were traced. Severe left atrial dilation was noted. The spleen was significantly enlarged (68x136x177 mm; splenic index 1637); the splenic vein was dilated to 17.5 mm. Liver deformity; local area of a single hepatic vein dilation in S4b (Fig. 1).



**Figure 1.** Multi-slice computed tomography of the abdominal cavity and of the chest organs

Designations: RA — right atrium, LA — left atrium, LV — left ventricle, RV — right ventricle, A — aorta, PA — pulmonary artery, RCA — right coronary artery, LCA — left coronary artery, LIV — liver, SPL — spleen

The patient was discharged with the following treatment recommendations: rivaroxaban 20 mg once daily, metoprolol succinate 50 mg once daily, allapinin 25 mg three times daily. While taking rivaroxaban, the patient developed menorrhagia and discontinued the drug herself. Later, due to significant dizziness, she spontaneously decreased the dose of allapinin to 25 mg daily. After discharge, dyspnea and substernal discomfort with moderate exertion, fatigability, weakness persisted. She was hospitalized to the V.A. Almazov National Medical Research Center (Saint-Petersburg) for examination and discussion of treatment selection.

In January 2024 she was electively admitted to the V.A. Almazov NMRC therapeutic department. Accounting for the syndrome of portal hypertension of unknown origin in the patient, magnetic resonance (MR) cholangiography and liver elastography were arranged to exclude sclerosing cholangitis. Liver elastography: F4 fibrosis based on METAVIR scale. MR-cholangiography: signs of sclerosing cholangitis. MR signs of liver cirrhosis, splenomegaly, gallstone disease, biliary dyskinesia (deformity, impaired colloidal bile properties), ectopic right kidney. The diagnosis of primary sclerosing cholangitis with the outcome of hypersplenism and liver cirrhosis was first established. Constant ursodeoxycholic acid and non selective beta-blockers were recommended for treatment.

Coronary angiography: the left anterior descending and circumflex branches of the left coronary artery were hypoperfused, diffusely altered, without significant stenosis; they filled retrogradely along intersystemic collaterals from the tortuous right coronary artery with a large diameter (~5 mm).

To evaluate the significance of myocardial ischemia, cardiac magnetic resonance imaging (MRI) was arranged: left ventricular (LV) ejection fraction 46%; end-diastolic volume 133 mL; end-systolic volume 59 mL; dilation of left heart chambers; LV walls were not thickened; the LV contractility was moderately decreased due to diffuse hypokinesia; no signs of myocardial edema were demonstrated. No clear pathological contrast enhancement in the LV myocardium was detected on delayed post-contrast images.

During the follow-up, the patient developed short-term symptomatic, but hemodynamically insignificant paroxysmal atrial fibrillation with the maximum ventricular rate (VR) of 110 per min. Holter electrocardiography (ECG) monitoring (treatment: sotalol 120 mg/day) — paroxysmal atrial flutter with predominant conduction 2:1, 3:1, lasting max 2 sec, with the rate of 108–116 (mean 112) bpm. No other significant arrhythmias and blocks were confirmed.

The following clinical diagnosis was established in the patient:

Main disease: Congenital heart disease: Bland-White-Garland syndrome (Anomalous Left Coronary Artery from the Pulmonary Artery). Hypoplasia of the descending thoracic aorta. Bicuspid aortic valve. Grade 1 aortic regurgitation. Grade 1 mitral stenosis. Grade 3 symptomatic hypertension, partially controlled, risk of cardiovascular complications 4, target BP <130/70–79.

Complications: Paroxysmal atrial fibrillation–atrial flutter. CHA<sub>2</sub>DS<sub>2</sub>-VASc 2 points, HAS-BLED 1 point, EHRA 2 points. CHF IIA with moderately reduced left ventricular ejection fraction (46%), FC III.

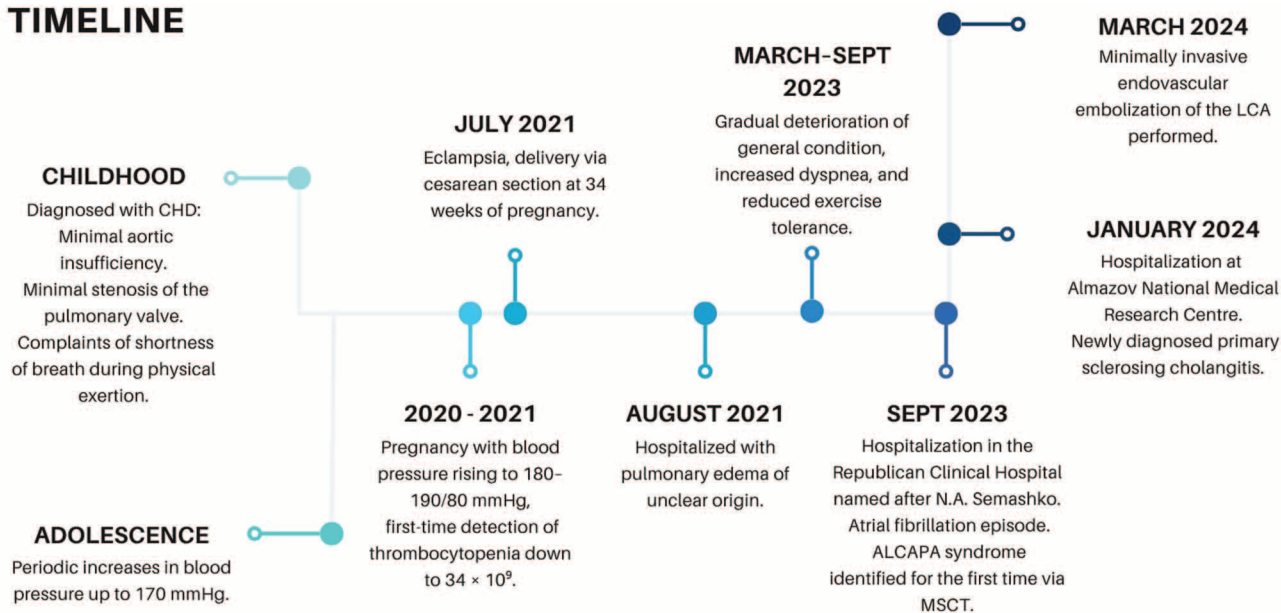
Concomitant diagnosis: Malnutrition. Hiatal hernia. Biliary dyskinesia. Gallstone disease. Small gallstones. Cholestasis. Hepatic fibrosis (METAVIR F4). Splenomegaly. Hypersplenism. Mild iron deficiency anemia. Grade 1 thrombocytopenia. Grade 1 leukopenia.

Accounting for concomitant diseases and associated postoperative risks, the mini-invasive surgery was selected. In March 2024 the patient underwent endovascular embolization of the left main coronary artery branching from the pulmonary artery. Under local anesthesia with the lidocaine solution (1% — 20.0), the femoral vein was punctured and catheterized; the left main coronary artery was embolized with a 7x7 mm occluder. Follow-up angiography: the blood flow in the left coronary artery was collateral from the right coronary artery; no blood flow was detected to the left coronary artery from the pulmonary artery. The postoperative period was uneventful. After the surgery, the patient's condition improved, and the heart failure was compensated at the level of FC II (NYHA). The following drug therapy was recommended for the patient: ursodeoxycholic acid 500 mg daily, folic acid 15 mg daily, valsartan/sacubitril 100 mg daily, eplerenone 25 mg daily, dapagliflozin 10 mg daily, sotalol 160 mg daily, apixaban 5 mg twice daily, ademethionine 400 mg 1 tab. daily for 1 month. See the event time scale in Figure 2.

## Discussion

The BWG syndrome is a disease usually manifesting clinically in childhood and (very rarely) in the adult years. Based on literature data, over 90% patients with this pathology die during the first year of life without surgical interventions [1]. Thus, the late presentation (as in our case) is an exception. Accounting for no specific disease symptoms (most common manifestation is with the decompensated heart failure), the BWG syndrome has to be differentiated with cardiomyopathies and coronary artery disease, especially in younger patients. Apart from clinical examination, it is feasible to include modern imaging methods (i.e. echocardiography, cardiac MRI, CT angiography, coronary angiography) into

## TIMELINE



**Figure 2. Timeline**

**Abbreviations:** CHD — congenital heart defect; BP — blood pressure; PA — physical activity; ALCAPA — anomalous left coronary artery from the pulmonary artery; MSCT — multi-slice computer tomography; LCA — left coronary artery.

the diagnostic algorithm. These diagnostic tools are required for the accurate investigation of the coronary vessel anatomy, evaluation of myocardial perfusion, and surgery planning. In our case the prolonged relative stability was associated with the collateral circulation, promoting the retrograde blood flow from the right coronary artery to the left coronary artery territory. Nevertheless, echocardiography and cardiac MRI results demonstrated left chamber dilation and moderately decreased left ventricular contractility, which confirmed the gradual worsening of hemodynamic disorders with the emergence of diastolic and systolic dysfunction.

The clinical case above describes a rare combination of a congenital heart disease (Bland-White-Garland syndrome) and a severe hepatobiliary pathology represented by the primary sclerosing cholangitis, which manifested with liver cirrhosis and signs of portal hypertension. This combination has been described only in single literature cases, which defines its scientific significance and underlines the need for the analysis of compensation/decompensation mechanisms in similar cases. Accounting for the high risk of intraoperative and postoperative complications (massive profuse hemorrhages, acute kidney injury, infectious complications) during the open cardiac surgery, which rate usually correlates with its scope and duration of cardiopulmonary bypass, the endovascular correction of the congenital heart disease was selected in our situation due to lower operative risks [6, 7]. A mini-invasive embolization of the left main coronary artery seems justified in the setting of significant somatic

diseases — that provided a partial elimination of pathological coronary blood flow, decreasing the left ventricular ischemic burden. Further patient management presumed the correction of antiarrhythmic and hepatoprotective therapy, considering persistent paroxysms of atrial fibrillation and hepatic failure. Further follow-up results will be practically significant for developing the tactics of multidisciplinary management in patients with a similar polymorbid background.

Thus, our case demonstrates the features of diagnosis and treatment in a patient with combined BWG syndrome and primary sclerosing cholangitis complicated with liver cirrhosis and portal hypertension. The clinical situation presented underlines the need for the complex approach to examination and management aimed at controlling the cardiovascular pathology and correcting severe hepatobiliary dysfunctions. This experience may be used when determining therapeutic and surgical strategies in patients with combined rare pathologies, as well as when evaluating risks and planning pregnancy in this patient category.

## Conclusion

Bland-White-Garland syndrome is a rare congenital anomaly of coronary blood flow which manifests most often in infancy. The case described demonstrates the possibility of prolonged compensated course due to collateral circulation, so the patient could live to adult years without surgical correction. The patient

management was complex due to the combination of BWG syndrome with a severe concomitant pathology, including primary sclerosing cholangitis resulting in liver cirrhosis. Modern imaging methods provided the timely disease diagnosis and evaluation of the myocardial ischemia severity. Accounting for the high risk of open surgical intervention, a mini-invasive endovascular method was selected, which resulted in the condition stabilization and compensation of chronic heart failure. This case underlines the importance of early diagnosis of coronary artery anomalies, using the multidisciplinary approach and personalized treatment tactics, especially in patients with a burdened somatic background.

#### Вклад авторов:

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

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#### Author Contribution:

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

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**Radkovskaya M.S.:** collection, analysis and interpretation of data, manuscript writing

**Radkovsky V.A.:** collection, analysis and interpretation of data, manuscript writing

**Morozov N.E.:** collection, analysis and interpretation of data

**Kazantseva O.A.:** collection, analysis and interpretation of data

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