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В.С. Щекин[1], Р.Ф. Рахимова[1], Е.А Лопина[2], Е.А. Бадыкова[1], Г.Д. Дивеева[1], Н.Ш. Загидуллин[1]

[1]— Федеральное государственное бюджетное образовательное учреждение высшего образования «Башкирский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Уфа, Россия
[2]— Федеральное государственное бюджетное образовательное учреждение высшего образования «Оренбургский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Оренбург, Россия

КЛИНИЧЕСКИЙ СЛУЧАЙ МИОКАРДИТА АБРАМОВА-ФИДЛЕРА У ПАЦИЕНТА СТАРЧЕСКОГО ВОЗРАСТА

V.S. Shchekin[1], R.F. Rakhimova[1], E.A. Lopina[2], E.A. Badykova[1], G.D. Diveeva[1], N.Sh. Zagidullin[1]

[1]— Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, Russia
[2]Federal State Budgetary Educational Institution of Higher Education «Orenburg State Medical University» of the Ministry of Health of the Russian Federation, Orenburg, Russia

Clinical Case of Idiopathic Abramov-Fiedler Myocarditis in The Elderly

Резюме

Миокардит Абрамова-Фидлера (идиопатический гигантоклеточный миокардит) относится к числу наиболее злокачественных форм неревматических воспалительных поражений миокарда. Заболевание традиционно диагностируется у лиц молодого и среднего возраста и сопровождается развитием быстро прогрессирующей сердечной недостаточности, жизнеугрожающих аритмий и тромбоэмболических осложнений. Представленный клинический случай имеет особое значение в связи с развитием миокардита Абрамова–Фидлера у пациента старческого возраста, что нетипично для данной нозологии. Пациент 80 лет был госпитализирован с клинической картиной острого коронарного синдрома с подъёмом сегмента ST. При поступлении отмечались интенсивные загрудинные боли, гипотензия, одышка и признаки острой левожелудочковой недостаточности. Лабораторные исследования выявили значительное повышение уровня тропонина и ферментов цитолиза. На электрокардиограмме регистрировался подъём сегмента ST по нижнебоковой стенке левого желудочка, а при коронароангиографии стеноз правой коронарной артерии составил лишь 30 % при сохранённом коронарном кровотоке. Несмотря на проводимую терапию, у пациента развился кардиогенный шок, завершившийся летальным исходом на вторые сутки заболевания. Патологоанатомическое исследование выявило очаги обширного воспалительного поражения миокарда с дистрофо-некротическими изменениями кардиомиоцитов, массивной смешанно-клеточной инфильтрацией и наличием гигантских многоядерных клеток. Иммуногистохимическое окрашивание с использованием антител к CD68 подтвердило макрофагальную природу клеточных элементов инфильтрата, что соответствует критериям гигантоклеточного миокардита. Данный клинический случай демонстрирует диагностические сложности, возникающие при атипичном течении миокардита Абрамова-Фидлера в пожилом возрасте, когда ведущую роль в клинической картине играют признаки, имитирующие острый коронарный синдром. Полученные данные указывают на необходимость высокой настороженности врачей в отношении воспалительных заболеваний миокарда у пациентов старших возрастных групп и подчёркивают значение патоморфологического и иммуногистохимического подтверждения диагноза.

Ключевые слова: *идиопатический миокардит Абрамова-Фидлера, гигантоклеточный миокардит, острый коронарный синдром, пожилой возраст, кардиогенный шок, сердечная недостаточность, морфологическая диагностика, иммуногистохимия, аутопсия, патоморфология*

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

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Информированное согласие не требуется в силу невозможности идентифицировать пациента

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Abstract
Abramov-Fiedler myocarditis (idiopathic giant cell myocarditis) represents one of the most malignant forms of non-rheumatic inflammatory heart disease. It is typically diagnosed in young and middle-aged patients and is characterized by rapidly progressive heart failure, life-threatening arrhythmias, and thromboembolic complications. The present clinical observation is of particular interest due to the development of Abramov-Fiedler myocarditis in an elderly patient, which is uncommon for this condition. An 80-year-old male was admitted with a clinical picture of ST-segment elevation acute coronary syndrome. On admission, he presented with severe retrosternal chest pain, hypotension, dyspnea, and signs of acute left ventricular failure. Laboratory tests revealed markedly elevated troponin and cytolytic enzymes. Electrocardiography demonstrated ST-segment elevation in the inferolateral wall of the left ventricle, while coronary angiography showed only a 30 % stenosis of the right coronary artery with preserved coronary flow. Despite intensive therapy, the patient developed cardiogenic shock and died on the second day of illness. Post-mortem examination revealed extensive myocardial inflammatory lesions with dystrophic and necrotic changes of cardiomyocytes, massive mixed-cell infiltration, and the presence of multinucleated giant cells. Immunohistochemical staining using CD68 antibodies confirmed the macrophage origin of the infiltrating elements, consistent with the diagnosis of giant cell myocarditis. This clinical case highlights the diagnostic challenges of atypical Abramov–Fiedler myocarditis in elderly patients, where the presentation may closely mimic acute coronary syndrome. The findings emphasize the importance of maintaining clinical vigilance for inflammatory myocardial diseases in older individuals and underscore the decisive role of morphological and immunohistochemical confirmation in establishing the diagnosis.

Key words: *idiopathic Abramov-Fiedler myocarditis, giant cell myocarditis, acute coronary syndrome, elderly patient, cardiogenic shock, heart failure, morphological diagnosis, immunohistochemistry, autopsy, pathomorphology*

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Conformity with the principles of ethics
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RVC — Regional Vascular Center, STEACS — ST-elevation acute coronary syndrome, BP — blood pressure, BMI — body mass index, SpO2 — oxygen saturation, RR — respiratory rate, HR — heart rate, CBC — common blood count, ECG — electrocardiogram, CAD — coronary artery disease, CAG — coronary angiography, RCA — right coronary artery

Introduction

The older patients are in the risk group for cardiovascular events, including acute myocardial infarction, acute cerebrovascular accident, pulmonary embolism. With the specific clinical signs, any physician starts working with these patients excluding the typical life-threatening conditions. Besides, several inflammatory diseases (including myocarditis) are mostly diagnosed in middle-aged patients. The post-COVID syndrome often manifested with viral myocarditis and atypical clinical manifestations, although the association with the trigger factor could not always be traced.

The idiopathic Abramov-Fiedler myocarditis is characterized by severe diffuse inflammatory, dystrophic, and degenerative myocardial alterations. Clinical signs of this disease are mainly defined by the rate of dystrophic and necrotic alterations in cardiomyocytes. The viral hypothesis for the Abramov-Fiedler myocarditis is confirmed by statistical data: chronic idiopathic myocarditis develops in 4–9 % patients after the acute viral myocarditis vs. 0.005 % in the general population. Approximately 20 % cases of Abramov-Fiedler myocarditis develop in patients with autoimmune diseases, e.g. Takayasu arteriitis, Hashimoto thyroiditis, Crohn's disease. Antimyocardial antibodies and cellular cytotoxicity are detected, thus confirming the immunopathological inflammation mechanism. The drug therapy of idiopathic myocarditis has low efficacy, and the majority of patients die due to complications.

Typical morphological signs of the Abramov-Fiedler myocarditis include isolated cardiac lesions, a combination of widespread dystrophic, infiltrative-inflammatory

myocardial alterations with diffuse cardiosclerosis, intracardiac thrombi and embolism of systemic arteries [1]. Gross pathology in the Abramov-Fiedler myocarditis reveals floppy walls and distended cardiac chambers with intramural thrombi; the wall section demonstrates mottled myocardial discoloration. The biomaterial microscopy detects muscle fiber hypertrophy, diffuse myolysis fields with the muscle tissue substituted with the fibrous one, and signs of coronariitis (inflammatory infiltrates along the small branches of coronary arteries).

A unique clinical case below describes idiopathic Abramov-Fiedler myocarditis in an older male without an overt history of autoimmune or viral diseases.

Clinical Case Study

The male patient N., 80 years old, was brought by the ambulance to the Regional Vascular Center (RVC) on March 24, 2024 at 7:45 p.m. with the diagnosis of ST-elevation acute coronary syndrome (STEACS).

On admission, the patient complained of sudden-onset substernal burning pain not associated with physical exertion, shortness of breath at rest, cold sweat, blood pressure (BP) drop to 80/40 mm Hg, dull pain in the right subcostal area, nausea and one-time vomiting of food.

Medical history: the patient did not suffer from cardiovascular diseases and did not monitor the BP values. He was not regularly followed up by the cardiologist. A day before the hospitalization, the patient started having the complaints above, but he did not seek medical attention. Due to pain worsening, he called the ambulance team on March 24, 2024. The ambulance staff suspected STEACS. The following medical care was provided for the patient before the hospital: acetylsalicylic acid 250 mg, clopidogrel 300 mg, intravenous heparin 4,000 U, nitroglycerin 0.5 mg, 1 % morphine (1 mL). The patient was hospitalized into the RVC emergency department.

According to the patient, he did not suffer from tuberculosis, venereal or parasitic diseases. In 2004, he was diagnosed with a gastric polyp, but did not receive any treatment. He had a history of frequent common colds, treating himself independently. He had been smoking for many years. The patient denied any allergic reactions to drugs.

Upon the admission to the inpatient department, the patient's condition was moderately severe. The patient's consciousness was clear. The body position was forced (lying on a stretcher). The body temperature was 36.6 °C. The oxygen saturation (SpO2) was 99 %. Physical examination: average constitutional build. The skin color and moisture level were physiological. No edema was detected. The body mass index (BMI) was 24.2 kg/m2. Lung auscultation revealed vesicular breathing with no rales. The respiratory rate (RR) was 16 per minute. Cardiac auscultation revealed a systolic murmur at the apex; cardiac tones were regular, muffled. The BP was 80/40 mm Hg. The heart rate (HR) was 80 beats per minute.

The common blood count (CBC) parameters were within the acceptable limits.

The biochemistry panel revealed increased troponin and CK-MB levels, thus confirming myocardial ischemia. Speaking about other specific biomarkers, elevated AST and LDH levels were also detected, thus indirectly signifying cardiomyocyte injury. In the lipid panel the LDL-C level was over the reference range, confirming a very high risk of adverse cardiovascular events. The detected high CRP level confirmed the active inflammation.

Electrocardiogram (ECG) dated March 24, 2025: sinus rhythm with HR 80 beats per minute, 2 mm ST segment elevation in leads II, III, AVF, V5, V6 (inferolateral wall of the left ventricle).

Chest X-ray was arranged on the same day. Chest X-ray demonstrated signs of hypervolemia in the pulmonary circulation, pulmonary congestion, emphysema, fibrotic alterations, aortic atherosclerosis.

The plain abdominal X-ray was arranged to exclude bowel obstruction — no air-fluid levels were visualized. Pneumatosis of the small and large bowel loops was detected. The abdominal ultrasound revealed hepatomegaly, irregular liver structure, and dilated hepatic veins. Other organs did not demonstrate pathological alterations. A small amount of free fluid was detected in all abdominal cavity regions and over the liver, spleen.

Table 1. *Biochemical blood analysis*

Indicator	Result	Reference values
Th T, (ng/L)	14740	0-200
CK-MB, (U/L)	98,12	0-24
ALT, (U/L)	636	0-35
AST, (U/L)	790,2	0-31
Glucose, (mmol/l)	6,34	3,5-5,5
Total protein (U/L)	62,92	62-83
CRP, (mg/L)	27,06	0-0,3
Chol, (mmol/L)	4,49	2,8-5,5
HDL-C, (mmol/L)	1,26	1,03-1,55
LDL-C, mmol/L	3,19	0-2,6
Triglycercides, (mmol/L)	1,21	0-2,1
LDH, (U/L)	1524,4	135-225

Note: CK-MB — creatine kinase MB fraction, Tn I — troponin I, ALT — alanine aminotransferase, AST — aspartate aminotransferase, CRP — C-reactive protein, HDL-C– high density lipoprotein cholesterol, LDL-C — low density lipoprotein cholesterol, LDH — lactate dehydrogenase

Echocardiography demonstrated a small amount of fluid in the pericardial cavity, 40 % ejection fraction (Simpson's), impaired local contractility (hypokinesis) in the inferolateral wall of the left ventricle.

Based on the clinical manifestations, physical examination and diagnostic investigations, the diagnosis of coronary artery disease (CAD), STEACS of the inferolateral wall of the left ventricle was established. Coronary angiography (CAG) revealed the 30 % stenosis of the right coronary artery (RCA), TIMI III blood flow. Other coronary arteries did not have occlusions. After CAG, the patient was transferred to the intensive care unit according to the current STEACS clinical guidelines.

Upon the admission to the intensive care unit, the patient's condition was stably severe. The patient's consciousness was clear. The body temperature was 36.5 °C. The skin had a normal physiological color. Lung auscultation revealed vesicular, but diffusely weakened breathing; RR was 16 per minute, SpO2 was 97 %. Cardiac auscultation revealed regular rhythm, muffled cardiac tones, and a systolic murmur at the apex. The BP was 110/70 mm Hg, HR was 80 beats per minute.

The patient developed sudden cardiac arrest during the night of 24/25 March, 2025. BP and HR could not be recorded. Asystole was recorded in the ECG. Full-scale resuscitation procedures were arranged within 30 minutes, to no effect. The biological patient's death was confirmed.

The patient's body was referred to the autopsy with the following diagnosis:

Main disease: CAD. Acute ST-elevation myocardial infarction of the inferolateral wall of the left ventricle dated March 24, 2024. CAG (March 24, 2024): 30 % RCA stenosis.

Complications: AHF (Killip IV). Acute left ventricular heart failure. Cardiogenic shock. Asystole.

Concomitant diseases: Chronic kidney disease, stage 3B. Multi-organ failure.

The following diagnosis was established based on the autopsy and pathohistology results:

Main disease: Idiopathic giant-cell (Abramov-Fiedler) myocarditis.

Complications: Diffuse myocardial dystrophy and necrosis. Acute left ventricular heart failure. Acute generalized venous congestion (interstitial-alveolar pulmonary edema, acute nutmeg liver). Cardiogenic shock. Acute prerenal failure.

Concomitant diseases: Essential hypertension (myocardial weight 500 g, thickness of the left ventricular wall 1.8 cm, glomerular hyalinosis, hyalinosis of splenic vessels). COPD (non-specific peribronchial-septal pneumofibrosis, panacinar emphysema). Chronic pancreatitis.

Pathology:

During the autopsy, a yellow-gray circular irregular myocardial focus was detected; it had a transmural (sometimes intramural) localization and a total area of 56 cm2. Signs of severe generalized venous congestion (congestive hyperemia of lungs, kidneys, liver, bowel, and vascular meningeal plexuses) were also found. Pieces for histology were collected and placed into the 10 % buffered neutral formalin during the autopsy. After a 48-hour fixation, the pieces were treated in alcohols with increasing concentrations, after which the specimens were paraffin-embedded, and the 4 μm slices were prepared and stained with hematoxylin-eosin. The immunohistochemistry with anti-CD68 primary antibodies (PG-M1 clone) and the Elabscience 2 Step Plus (Poly-HRp anti-Rabbit/mouse IgG with DAB solution, Cat. No E-IR-R213) were arranged on the Autostainer 360 immunohistostainer (Thermo Fisher Scientific, USA). The prepared glasses were scanned using the Pannoramic 250 (3DHISTECH Ltd., Hungary) with subsequent analysis of histological slices under various magnification using the CaseViewer (3DHISTECH Ltd., Hungary) software.

The heart microscopy revealed widespread involvement of the contractile myocardial parenchyma into the inflammatory process with massive interstitial infiltration with various inflammatory elements (lymphocytes, eosinophils, macrophages, and scattered giant multinucleated cells) (Figure 1). Myocardial fibers underwent significant dystrophic alterations with wavy fibrils, contracted cytoplasm volume, and fibrotic substitution foci. Lungs had signs of acute venous congestion with interstitial-alveolar pulmonary edema, acute centrilobular hepatic congestion, and acute tubular lesions (cytoplasm hydrops, fragmentation of apical epitheliocyte

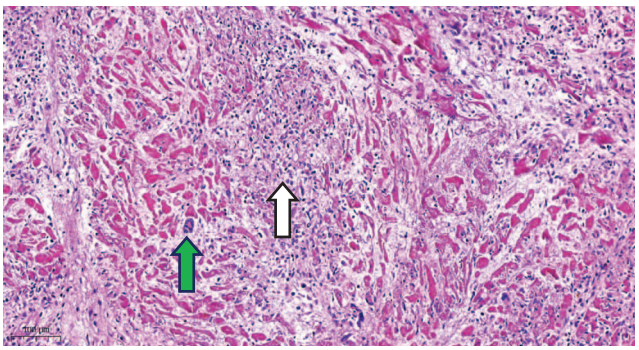

Figure 1. *Micrograph of the intramural myocardium. Pronounced dystrophic-necrotic changes in myofibrils, focal replacement of the parenchyma by connective tissue elements (white arrow) against the background of abundant interstitial mixed cellular infiltration with the appearance of giant multinucleated cells (green arrow). Magnification ×200; hematoxylin and eosin staining*

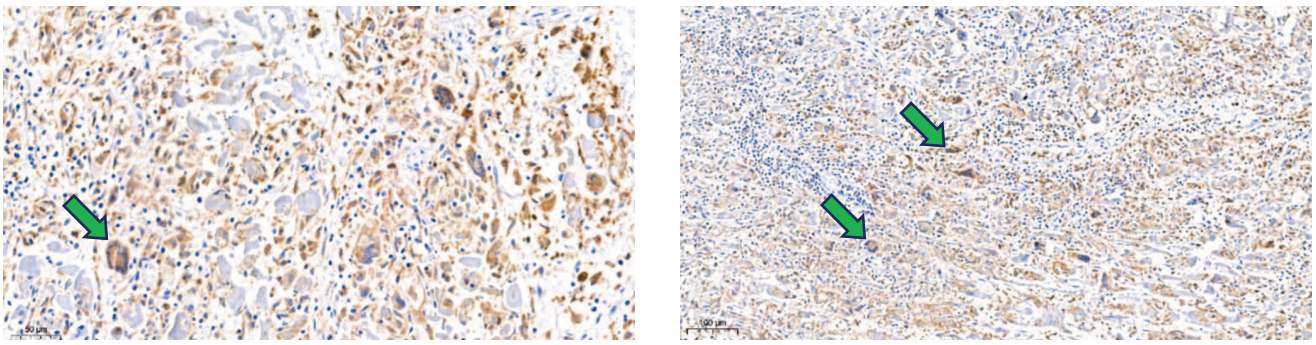

Figure 2. *Photomicrograph of immunohistochemical staining of myocardium for CD68. Persistent cytoplasmic positive staining in multinucleated and mononuclear histiocytic (arrow) inflammatory elements and complete absence of staining in the myogenic component. Immunohistochemical staining for CD68. Magnification ×400, ×200; hematoxylin and eosin staining*

poles in kidneys). To confirm the macrophagal origin of the giant-cell component in the inflammatory infiltrate, immunohistochemistry with anti-CD68 antibodies was arranged, which revealed persistent positive cytoplasmic staining of multinucleated elements, confirming its histiocytic histogenesis (Figure 2).

Based on the autopsy and pathohistology findings, the following diagnosis was established: Idiopathic giant-cell (Abramov-Fiedler) myocarditis complicated with diffuse cardiomyocyte dystrophy and necrosis (dystrophic variant), acute left ventricular heart failure, and acute prerenal failure.

Discussion

The incidence of myocarditis has increased over the past few years, with the current incidence being equal to 10–12 cases per 100,000 population. According to the statistics, myocarditis is more common among young males under 45 years of age, however a more severe disease course is typical for females. The average age of patients with the confirmed diagnosis of idiopathic myocarditis is 42 years [2].

In the case described, the patient belonged to the older age group, which blunted the clinical awareness of physicians concerning inflammatory diseases. The incidence of coronarogenic myocardial diseases of atherosclerotic origin increases with age, displacing other diseases. In our clinical case, the combination of several risk factors (including age, gender, smoking) formed the foundation to suspect the acute coronary syndrome initially.

Abramov-Fiedler myocarditis is considered a very severe form of non-rheumatic myocarditis with high mortality. Young and relatively healthy persons are the typical patients with Abramov-Fiedler myocarditis. Most often patients with the confirmed diagnosis of idiopathic myocarditis had a history of viral or autoimmune diseases, e.g. Coxsackie viral infections, Crohn's disease, Hashimoto thyroiditis, systemic lupus erythematosus, etc. [3].

According to the patient N.'s words, he had frequent common colds which were never verified or treated medically with specific treatment. Due to the atypical or subclinical myocarditis course, the majority of its cases are detected only on autopsy. However, it should be noted that the most typical clinical symptoms for myocarditis include cardiac pain and ventricular arrhythmias or heart blocks. Heart failure quickly progresses in 75 % cases, while 50 % patients develop sustained ventricular tachycardias [4].

A day before the hospitalization and during the short-term hospital stay, our patient had substernal intensive burning pain, but he did not complain of palpitations. No arrhythmias typical for the Abramov-Fiedler myocarditis were diagnosed.

The laboratory and instrumental diagnosis confirming myocarditis is rather difficult and costly. Detected elevated serum levels of cardiospecific enzymes (troponin, CK) may reflect the damaging effects of any factors on cardiomyocytes. The results of instrumental investigations have to be accounted for to confirm the specific diagnosis. Considering our patient, the combination of elevated troponin levels and typical ST segment alterations in the ECG provided the clinical presumption of an acute myocardial infarction; however, CAG demonstrated only 30 % RCA stenosis without its thrombotic occlusion.

The mortality in myocarditis reaches 20–40 % [5]; in the majority of cases, patients die from acute left ventricular failure (ALVF) or ventricular fibrillation. In our case, quick ALVF development (cardiogenic shock) became the cause of death.

The histology of the autopsy material reveals typical dystrophic necrobiotic alterations of cardiomyocytes in the myocardium and the interstitial tissue along with the

widespread inflammatory infiltrate mainly represented by lymphocytes, although giant multinucleated cells are also detected [5]. The microscopy of the patient's biomaterial revealed widespread involvement of the contractile myocardial parenchyma into the inflammatory process with massive interstitial infiltration with various inflammatory elements (lymphocytes, eosinophils, macrophages, and scattered giant multinucleated cells), which coordinates with the general immunohistochemistry results.

In this case myocarditis developed in an elderly patient (80 years old), which is an atypical case. These autopsy data specific for the Abramov-Fiedler myocarditis may be potentially associated with prior viral diseases, including COVID-19. It should be noted that arrhythmias and thromboembolic complications typical for the disease were missing.

Conclusions

The Abramov-Fiedler myocarditis belongs to very severe forms of non-rheumatic myocarditis with high mortality, which usually develops in young patients. The clinical case presented demonstrates a rare disease course in an elderly patient with clinical signs imitating acute coronary syndrome, along with the absence of typical manifestations (significant arrhythmias, intracardiac thrombi). Pathomorphological and immunohistochemistry data helped to verify the diagnosis of giant-cell myocarditis.

The clinical case underscores the need for enhanced awareness of inflammatory diseases in elderly patients, even in the setting of risk factors for the coronary artery disease. Accounting for the possible role of viral infections, including prior COVID-19, in the disease pathogenesis, an advanced-age variant of the Abramov-Fiedler myocarditis should be considered.

Вклад авторов:

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

Щекин В.С.: сбор, анализ, интерпретация данных, разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания, ответственный за все аспекты работы

Рахимова Р.Ф.: сбор, анализ, интерпретация данных, разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания, ответственный за все аспекты работы

Лопина Е.А.: разработка концепции и дизайна, анализ и интерпретация данных, проверка критически важного интеллектуального содержания, ответственный за все аспекты работы

Бадыкова Е.А.: разработка общей концепции и дизайна статьи, проверка критически важного интеллектуального содержания, ответственный за все аспекты работы

Дивеева Г.Д.: сбор, анализ и интерпретации данных, проверка критически важного интеллектуального содержания, подготовка рукописи, ответственный за все аспекты работы

Загидуллин Н.Ш.: сбор, анализ и интерпретации данных, проверка критически важного интеллектуального содержания, ответственный за все аспекты работы, окончательное утверждение рукописи для публикации.

Author Contribution:

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

Vlas S. Shchekin: data collection, analysis, and interpretation; development of the overall concept and study design; manuscript drafting; critical revision of the manuscript for important intellectual content; accountable for all aspects of the work

Rozana F. Rakhimova: data collection, analysis, and interpretation; development of the overall concept and study design; manuscript drafting; critical revision of the manuscript for important intellectual content; accountable for all aspects of the work

Ekaterina A. Lopina: development of the concept and study design; data collection, analysis, and interpretation; critical revision of the manuscript for important intellectual content; accountable for all aspects of the work

Elena A. Badykova: development of the overall concept and study design; critical revision of the manuscript for important intellectual content; accountable for all aspects of the work

Gulnara D. Diveeva: data collection, analysis, and interpretation; critical revision of the manuscript for important intellectual content; manuscript preparation; accountable for all aspects of the work

Naufal Sh. Zagidullin: data collection, analysis, and interpretation; critical revision of the manuscript for important intellectual content; accountable for all aspects of the work; final approval of the version to be published.

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Информация об авторах

Щекин Влас Сергеевич — Заведующий морфологической лабораторией ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения Российской федерации, Уфа, E-mail: vlas-s@mail.ru, ORCID ID: https://orcid.org/0000-0003-2202-7071

Рахимова Розана Фанисовна — Аспирант кафедры пропедевтики внутренних болезней ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения Российской федерации, Уфа, E-mail: r.r-7@mail.ru, ORCID ID: https://orcid.org/0000-0002-2958-616X

Лопина Екатерина Анатольевна — к.м.н., Доцент кафедры госпитальной терапии им. Р.Г. Межебовского, ФГБОУ ВО «Оренбургский государственный медицинский университет» Министерства здравоохранения Российской федерации, Оренбург, E-mail: ekaterina_lopina@mail.ru, ORCID ID: https://orcid.org/0000-0001-7474-7922

Бадыкова Елена Альбертовна — к.м.н., Доцент кафедры пропедевтики внутренних болезней ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения Российской федерации, Уфа, E-mail: lnurova@mail.ru, ORCID ID: https://orcid.org/0000-0002-8167-4271

Дивеева Гульнара Дамировна — к.м.н., Доцент кафедры патологической анатомии ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Уфа, E-mail: diveyevagd@yandex.ru, ORCID ID: https://orcid.org/0009-0003-4814-9779

Загидуллин Науфаль Шамилевич — д.м.н., Заведующий кафедрой пропедевтики внутренних болезней ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения Российской федерации, Уфа, E-mail: zsnaufal@gmail.com, ORCID ID: https://orcid.org/0000-0003-2386-6707

Authors Information:

Vlas S. Shchekin — Head of the Morphological Laboratory Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, E-mail: vlas-s@mail.ru, ORCID ID: https://orcid.org/0000-0003-2202-7071

Rozana F. Rakhimova — Postgraduate student of the Department of Propaedeutics of Internal Diseases Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, E-mail: r.r-7@mail.ru, ORCID ID: https://orcid.org/0000-0002-2958-616X

Ekaterina A. Lopina — PhD, Associate Professor of the Department of Hospital Therapy named after R.G. Mezhebovsky, Federal State Budgetary Educational Institution of Higher Education «Orenburg State Medical University» of the Ministry of Health of the Russian Federation, Orenburg, E-mail: ekaterina_lopina@mail.ru, ORCID ID: https://orcid.org/0000-0001-7474-7922

Elena A. Badykova — PhD, Associate Professor of the Department of Propaedeutics of Internal Diseases Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, E-mail: lnurova@mail.ru, ORCID ID: https://orcid.org/0000-0002-8167-4271

Gulnara D. Diveeva — PhD, Associate Professor of the Department of Pathological Anatomy Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, E-mail: diveyevagd@yandex.ru, ORCID ID: https://orcid.org/0009-0003-4814-9779

Naufal Sh. Zagidullin — Doctor of Medical Sciences, Head of the Department of Propaedeutics of Internal Diseases Federal State Budgetary Educational Institution of Higher Education «Bashkir State Medical University» of the Ministry of Health of the Russian Federation, Ufa, E-mail: zsnaufal@gmail.com, ORCID ID: https://orcid.org/0000-0003-2386-6707

Автор, ответственный за переписку / Corresponding author