



DOI: 10.20514/2226-6704-2024-14-6-467-472

УДК 616.124.2-008.31-073.7-085

EDN: UPHGBH



Д.С. Евдокимов, Е.Г. Быкова, С.А. Болдуева,
Е.Д. Реснянская

ФГБОУ ВО «Северо-Западный государственный медицинский университет
им. И.И. Мечникова» Минздрава России, кафедра факультетской терапии,
Санкт-Петербург, Россия

СЛУЧАЙ РАЗВИТИЯ СИНДРОМА ТАКОЦУБО ПОСЛЕ ПЛАНОВОЙ ЭЛЕКТРОИМПУЛЬСНОЙ ТЕРАПИИ

D.S. Evdokimov, E.G. Bykova, S.A. Boldueva, E.D. Resnyanskaya

North-Western State Medical University named after I.I. Mechnikov,
Department of Faculty Therapy, St. Petersburg, Russia

A Case of The Development of Takotsubo Syndrome After Electropulse Therapy

Резюме

В статье приводится описание клинического наблюдения синдрома такоцубо с развитием кардиогенного шока у пациента 77 лет с персистирующей формой фибрилляции предсердий после проведения плановой электроимпульсной терапии для восстановления синусового ритма. Диагноз синдром такоцубо был подтверждён на основании лабораторно-инструментальных данных: изменений на электрокардиограмме (элевация сегмента ST в отведения V3-4 на 2-3 мм), повышения уровня тропонина (456,8 нг/л), выявленных нарушений сократимости левого желудочка по данным эхокардиографии (акинез всех верхушечных сегментов, передних, передне- и нижне-перегородочных сегментов на срединном уровне, гипокинез остальных сегментов на срединном уровне) с последующим полным восстановлением сократимости левого желудочка в динамике, результатов коронароангиографии (значимых стенозов/тромбозов не выявлено) и данных магнитно-резонансной томографии сердца с гадолинием (отсутствуют признаки миокардита, рубцовых изменений в миокарде).

Представленный клинический случай еще раз подчеркивает важность осведомленности специалистов о возможном риске развития синдрома такоцубо после электроимпульсной терапии, так как это позволит своевременно установить диагноз и инициировать соответствующее лечение. Пациентам с такими факторами риска развития синдрома такоцубо, как с наличие в анамнезе психических или неврологических заболеваний, бронхиальной астмы, хронической обструктивной болезни легких, диффузного узлового зоба, гипо-/гипертиреоза, после проведения кардиоверсии, по-видимому, требуется более тщательное и длительное наблюдение. Подобная тактика, вероятно, позволит своевременно диагностировать это осложнение для предотвращения серьезных последствий, однако требуется дальнейшее изучение этого вопроса.

Ключевые слова: синдром такоцубо, электроимпульсная терапия, фибрилляция предсердий

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 «Случай развития синдрома такоцубо после плановой электроимпульсной терапии» для журнала «Архивъ внутренней медицины», подписав информированное согласие

Статья получена 07.07.2024 г.

Одобрена рецензентом 05.08.2024 г.

Принята к публикации 09.09.2024 г.

Для цитирования: Евдокимов Д.С., Быкова Е.Г., Болдуева С.А. и др. СЛУЧАЙ РАЗВИТИЯ СИНДРОМА ТАКОЦУБО ПОСЛЕ ПЛАНОВОЙ ЭЛЕКТРОИМПУЛЬСНОЙ ТЕРАПИИ. Архивъ внутренней медицины. 2024; 14(6): 467-472. DOI: 10.20514/2226-6704-2024-14-6-467-472. EDN: UPHGBH

Abstract

The article describes a clinical observation of takotsubo syndrome with the development of cardiogenic shock in a 77-year-old patient with persistent atrial fibrillation after planned electrical impulse therapy to restore sinus rhythm. The diagnosis of ST was confirmed based on laboratory

and instrumental data: changes in the electrocardiogram (ST segment elevation in leads V3-4 by 2-3 mm), increased troponin levels (456.8 ng/l), identified left ventricular contractility disorders according to echocardiography (akinesis of all apical segments, anterior, anterior and inferior septal segments at the median level, hypokinesis of the remaining segments at the median level) followed by complete restoration of left ventricular contractility over time, coronary angiography results (no significant stenosis/thrombosis detected) and magnetic resonance imaging data of the heart with gadolinium (no signs of myocarditis, cicatricial changes in the myocardium).

The presented clinical case once again emphasizes the importance of awareness of specialists about the possible risk of developing takotsubo syndrome after electrical impulse therapy, as this will allow timely diagnosis and initiation of appropriate treatment. Patients with such risk factors for the development of takotsubo syndrome as a history of mental or neurological diseases, bronchial asthma, chronic obstructive pulmonary disease, diffuse nodular goiter, hypo-/hyperthyroidism, after cardioversion, apparently require more careful and long-term monitoring. Such tactics will probably allow timely diagnosis of this complication to prevent serious consequences, but further study of this issue is required.

Key words: *takotsubo syndrome, electropulse therapy, atrial fibrillation*

Conflict of interests

The authors declare no conflict of interests

Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article «A Case of The Development of Takotsubo Syndrome After Electropulse Therapy» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

Sources of funding

The authors declare no funding for this study

Article received on 07.07.2024

Reviewer approved 05.08.2024

Accepted for publication on 09.09.2024

For citation: Evdokimov D.S., Bykova E.G., Boldueva S.A. et al. A Case of The Development of Takotsubo Syndrome After Electropulse Therapy. The Russian Archives of Internal Medicine. 2024; 14(6): 467-472. DOI: 10.20514/2226-6704-2024-14-6-467-472. EDN: UPHGBH

BP — blood pressure, CAG — coronary angiography, LV — left ventricle, MRI — magnetic resonance imaging, ACS — acute coronary syndrome, AHF — acute heart failure, TS — Takotsubo syndrome, HF — heart failure, HECGM — Holter electrocardiogram monitoring, AFL — atrial flutter, EF — ejection fraction, AFib — atrial fibrillation, GDS — gastroduodenoscopy, ECV — electrical cardioversion, ECG — electrocardiogram, EchoCG — echocardiography, VR — ventricular rate

Introduction

Atrial fibrillation (AFib) is the most common type of supraventricular tachyarrhythmias globally [1]. This arrhythmia correlates with the 5-fold increase in the stroke risk and the 2-fold mortality risk [2], thus, the rhythm control strategy is considered preferable [3]. Electrical cardioversion (ECV) is considered the most efficient method providing quick sinus rhythm restoration in paroxysmal AFib [3]. According to the clinical guidelines, this procedure is considered safe even in pregnancy [3]. 23% of complications after ECV are related to possible irritation, pain, and/or burns in the places of skin contact with electrodes [1], however, cases of Takotsubo syndrome (TS) development after ECV have been published in the recent years [1].

Takotsubo syndrome is an acute reversible heart failure (HF) with a transient left ventricular (LV) dysfunction, which is often clinically equivalent to the acute coronary syndrome (ACS) [4, 5]. For the first time the term “takotsubo” (“octopus trap” in Japanese) was introduced by Sato H. et al. in 1990, as the LV shape based on echocardiography data is similar to that of the fisher’s trap in this syndrome [4]. The most common TS trigger is the psychoemotional stress; however, in some cases it can be triggered by the new onset or exacerbation of the pre-existing chronic disease, drug product administration, or

the medical intervention [5, 6], including ECV. Based on the latest data, the prevalence of this syndrome associated with ECV is 2.7 cases per 10,000 ECV procedures in AFib [1]. Clinical signs of the transient LV dysfunction manifest within 24-48 hours after ECV with signs of acute HF, up to cardiogenic shock [1].

Let’s get acquainted with the case study of TS after the elective electrical cardioversion in paroxysmal AFib with subsequent circulatory arrest and cardiogenic shock.

Clinical case study

A 77-year-old male was hospitalized electively for additional examination and determining further management tactics.

He had a long history of essential hypertension, with maximum blood pressure (BP) values of 180/90 mm Hg; hypotensive treatment led to BP values of 120/70 mm Hg. The patient denied the history of myocardial infarction, cerebrovascular diseases, diabetes mellitus, angina and dyspnea; he tolerated physical loads satisfactorily, however he noted decreasing tolerance within the previous two months.

The first atrial flutter (AFL) paroxysm was reported in 2011, which was treated with radiofrequency catheter ablation of the cavotricuspid isthmus (2012). In 2018 the

patient developed AFib relapse, which was treated with ECV; the anti-relapse treatment included sotalol (160 mg in the morning, 80 mg in the evening), with concomitant dabigatran 150 mg twice daily, losartan 25 mg/day, spironolactone 25 mg/day. After that, when AFib paroxysm developed 5 years later (on November 15, 2022), cardioversion was provided by amiodarone administration.

A week after that, the patient was electively hospitalized for examination and treatment correction. The Holter ECG monitoring (HECGM) recorded a sinus rhythm with a mean ventricular rate (VR) of 54/min, 130 single ventricular extrasystoles per hour; no significant pauses were detected, with the mean QT (Bazett) duration of 456 ms. Stress-echocardiography (EchoCG) yielded negative results. Mild iron deficiency anemia was detected based on the laboratory tests. During the diagnostic search of anemia etiology, acute gastric ulcer and esophageal candidiasis were detected during gastro-duodenoscopy (GDS). Thus, treatment with rabeprazol, rebamipide, and fluconazole was initiated. As concomitant use of fluconazole and sotalol may lead to prolonged QT interval duration, it was decided to switch sotalol to metoprolol succinate 100 mg. The dose of dabigatran was also reduced (to 110 mg twice daily) for two months due to the increased risks of ulcer hemorrhage.

A year later (in December 2023), during the hospitalization due to acute gastroenteritis, the patient noticed dyspnea on moderate physical exertion and arrhythmic palpitations. The electrocardiogram (ECG) recorded AFib with VR of 120-140 bpm. As COVID-19 infection was detected during this hospitalization (with the polymerase chain reaction method), the patient was discharged to outpatient treatment. However, as AFib persisted, the patient referred to another medical institution to consider the cardioversion tactics.

Complaints of tachyarrhythmias and dyspnea preserved during the elective hospitalization as of January 11, 2024. Physical examination: the patient's consciousness was clear, his condition was relatively satisfactory; skin and visible mucous membranes were normal, no peripheral edema or cyanosis was detected. BP 115/70 mm Hg, VR 116 bpm, arrhythmic pulse; cardiac tones were muffled, but without pathological murmurs. The respiratory rate was 15 per min; on auscultation, the breathing was harsh, but auscultated in all areas, without rales or wheezing. The abdomen was soft and non-tender.

The day after the hospitalization, transesophageal EchoCG was arranged, which revealed no thrombi, with spontaneous Grade 2 contrasting. Based on the clinical and historical data, it was decided to restore the sinus rhythm using ECV, and the patient was transferred to the cardiac intensive care unit.

After the electrical cardioversion (300 J), the ECG demonstrated sinus rhythm with VR of 72/min; however, 2 min later, bradycardia with VR of 25-30/min developed, followed by asystole. The external cardiac massage was started, 1 mg of atropine and 1 mg of epinephrine were administered. This led to cardiac activity restoration, though with preserved hypotension (BP approximately 70/40 mm Hg), due to which vasopressor support was initiated (norepinephrine up to 0.3 µg/kg/min, epinephrine up to 0.03 µg/kg/min).

When evaluating follow-up ECG, 2-3 mm ST segment elevation was recorded in leads V₃₋₄ along with sinus rhythm with VR of 82/min (Fig. 1). The urgent EchoCG detected significantly decreased LV ejection fraction (EF). Evaluation of local kinetic disorders of LV walls was considered incorrect with the significantly decreased global contractility (Table 1). It was decided to arrange coronary angiography (CAG), which yielded no significant stenoses/thromboses. After CAG, ECG revealed sinus rhythm with HR of 82/min without significant ST deviation. Elevated troponin levels (Table 2) were reported, which were most likely due to myocardial damage (ECV, external cardiac massage, CAG) and hemodynamic disorders.

The common blood count demonstrated neutrophilic leukocytosis ($15.77 \times 10^9/L$) and elevated platelet count of $476 \times 10^9/L$. Biochemistry: hyperglycemia up to 7.3 mmol/L (N 4.1-5.9), creatinine — 111 µmol/L, C-reactive protein — 16.98 mg/L (N 0-5), potassium — 5.88 mmol/L (N 3.5-5.1), decreased albumin to 26.6 g/L (N 35-52) and total protein to 45.5 g/L (N 66-83). Thyroid-stimulating hormone: 1.76 µIU/mL (N 0.35-4.94). Lipid profile: total cholesterol — 3.71 mmol/L, low-density lipoproteins — 2.21 mmol/L, triglycerides — 1.16 mmol/L. The urinalysis and coagulation panel were normal. All blood parameters normalized with the treatment administered by Day 14.

The EchoCG repeated next day (January 13, 2024) revealed increasing LVEF, though akinesis of all apical segments, midline akinesis of anterior, antero- and inferoseptal segments, midline hypokinesis of other segments was reported (Table 1, Fig. 2). Contrast-enhanced computed tomography of the chest was arranged to exclude pulmonary embolism: no signs suggestive of the neoplasm, acute inflammatory and disseminated pulmonary diseases were detected, although bilateral hydrothorax was visualized.

During the magnetic resonance imaging (MRI) of the brain on January 15 (signs of single focal lesions of vascular origin in the brain matter, left-sided exudative sinusitis), hypotension (60/40 mm Hg) and relapsing AFib (VR 140-160 bpm) emerged due to forced

halt of inotropic and vasopressor support. Diuresis also decreased, and this led to the replacement of epinephrine with dobutamine (treatment continued as follows: dobutamine — 14 µg/kg/min, norepinephrine — 0.9 µg/kg/min); albumin, gelofusin infusions were administered. HECGM demonstrated AFib-AFl with irregular and regular 2:1 conduction, transient tachycardia-dependent complete right bundle branch block with an average VR of 151/min, QT of 378-511 ms; no significant pauses were recorded.

The history of recent viral infection (December 2023) and high anti-coronavirus antibody titers (IgG — 25,291 U/mL) provided a possible diagnosis of active myocarditis with cardiac conduction disorders. This was an indication to gadolinium-enhanced cardiac MRI, which demonstrated no signs of myocarditis or scarring lesions.

The follow-up EchoCG revealed increasing EF with the normalization of functional parameters and restoration of motion in all LV walls (Table 1, Fig. 3, Fig. 4).

Table 1. Echocardiography data during dynamic observation

	29.12.23 r.	12.01.24 r.	13.01.24 r. (Fig. 2)	17.01.24 r. (Fig. 3)	19.01.24 r. (Fig. 4)	22.01.24 r.
Left ventricular ejection fraction, %	60 %	25 %	31 %	40 %	50 %	70 %
Local contractility of the left ventricle	No local contractility disorders were detected	The assessment of local kinetics against the background of a marked decrease in global contractility is not correct	Akinesia of all apical segments, anterior, anterior and inferior septal segments at the median level, hypokinesia of the remaining segments at the median level	Diffuse myocardial hypokinesia, no local contractility disorders detected	Mild hypokinesia of the anterior septal and anterior walls of the left ventricle	No local contractility disorders were detected

Table 2. Dynamic troponin level data

	12.01.24 r.	13.01.24 r.	14.01.24 r.	15.01.24 r.	20.01.24 r.
Troponin, ng/l	24,6	370	456,8	414,4	97,1

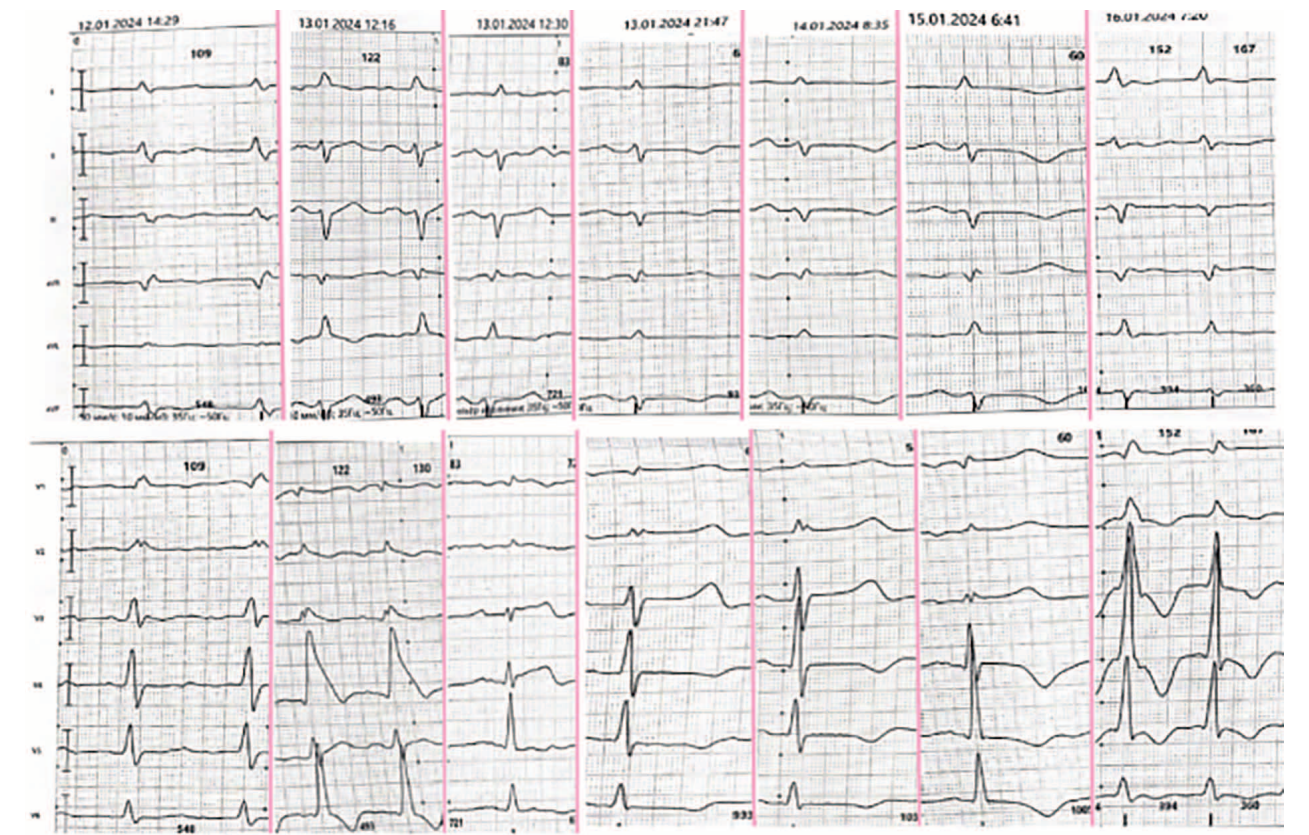


Figure 1. ECG data in dynamics

By January 22, hemodynamics stabilized with restoring LV contractility and VR decreasing to 90-100 bpm — after that, inotropic and vasopressor support was discontinued. The patient was transferred to the cardiology department for further follow-up and deciding on the management tactics.

When analyzing the time course of clinical and laboratory-instrumental data, secondary TS that had developed in response to ECV was suspected.

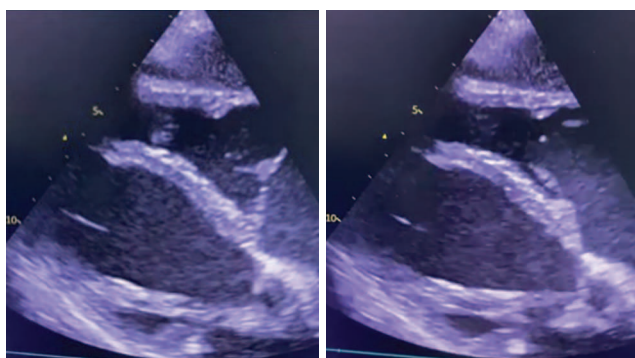


Figure 2. Echocardiography from 13.01: akinesia of all apical segments, anterior, anterior and inferior septal segments at the median level, hypokinesis of the remaining segments at the median level

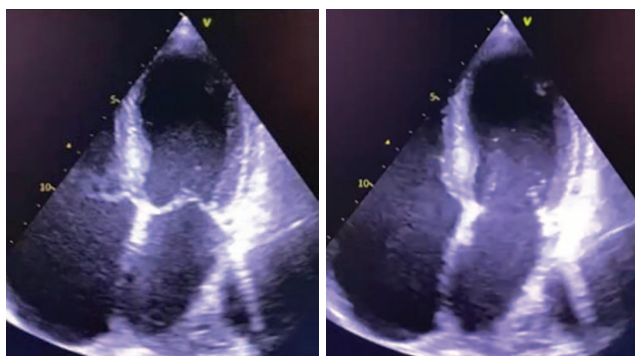


Figure 3. Echocardiography from 17.01: diffuse myocardial hypokinesia, no local contractility disorders detected

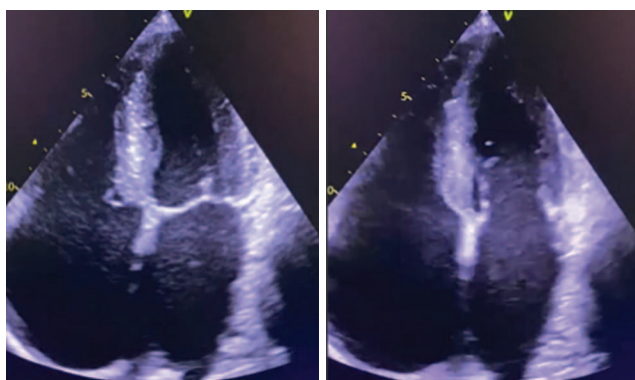


Figure 4. Echocardiography from 19.01: mild hypokinesia of the anterior septal and anterior walls of the left ventricle

During the hospital stay, the active patient regimen was extended, HF signs did not progress, while HECGM revealed persisting AFib with an average VR of 80-90 bpm. The following rate control and supportive treatment was administered: metoprolol succinate 150 mg/day, apixaban 5 mg twice daily, losartan 25 mg/day, spironolactone 25 mg/day, torasemide 5 mg/day, atorvastatin 20 mg/day, omeprazol 20 mg/day. The patient was discharged with the recommendation of elective hospitalization in 3 months to discuss the necessity of cardioversion.

Diagnosis upon discharge: Grade 3 essential hypertension, controlled hypertension, very high risk of cardiovascular complications (4). Persistent atrial fibrillation, paroxysm dated December 2023. Cardioversion with ECV on January 13, 2024. Takotsubo syndrome (January 13, 2024). Cardiogenic shock (January 13, 2024). Circulatory arrest (January 13, 2024). New-onset tachycardia-dependent complete right bundle branch block. Paroxysmal AFib (January 15, 2024), not controlled. EHRA 2b. CHA2DS2-VASc 4 points. HAS-BLED: 2 points.

Discussion

The presented case study demonstrates that ECV may become a trigger for TS with severe hemodynamic complications.

The pathogenesis of this ECV-associated condition has not been studied well, though it is presumed that electrical myocardial damage may activate the classic reaction cascade with hypercatecholaminemia, hyper-sympathetic tone, mitochondrial dysfunction, thus initiating the transient LV dysfunction [7].

The severity of the patient's condition after the ECV was probably related to a prolonged history of AFib. This point of view correlates with the literature data analyzing this issue — it is stated that the emergence of TS in patients with the history of this arrhythmia is an AHF predictor, as AFib directly correlates with worsening hemodynamics. Based on the results presented by I. El-Battrawy et al., it was also demonstrated that compared to patients without AFib, patients with TS and AFib developed cardiogenic shock (requiring urgent treatment, including the intraaortic balloon pump) more frequently, with a more significant hospital mortality [2].

Conclusion

The case study presented emphasizes the importance of the physician awareness about the possible risk of TS after ECV. Such approach enables the timely diagnosis with the initiation of the corresponding treatment.

Probably, a more thorough and prolonged follow-up is required after cardioversion for patients with various TS risk factors, including the history of psychiatric or neurological diseases, asthma, chronic obstructive pulmonary disease, diffuse nodular goiter, hypo-/hyperthyroidism [8, 9, 10]. Such tactics will possibly enable the timely diagnosis of this complication to prevent serious complications; however, this issue has to be analyzed more thoroughly.

Вклад авторов:

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

Евдокимов Д.С.: сбор данных, написание текста статьи, работа с литературными источниками

Быкова Е.Г.: написание текста статьи, редактирование текста статьи, утверждение финального варианта рукописи

Болдуева С.А.: редактирование текста статьи, утверждение финального варианта рукописи

Реснянская Е.Д.: сбор данных, написание текста статьи, работа с литературными источниками

Author Contribution:

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

Evdokimov D.S.: data collection, writing the text of the article, working with literary sources

Bykova E.G.: writing the text of the article, editing the text of the article, approval of the final version of the manuscript

Boldueva S.A.: editing the text of the article, approval of the final version of the manuscript

Resnyanskaya E.D.: data collection, writing the text of the article, working with literary sources

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Информация об авторах

Евдокимов Дмитрий Сергеевич — ассистент кафедры факультетской терапии ФГБОУ ВО СЗГМУ им. И.И. Мечникова, Санкт-Петербург, e-mail: kasabian244@gmail.com, ORCID ID: <https://orcid.org/0000-0002-3107-1691>

Быкова Елена Григорьевна — к.м.н., доцент кафедры факультетской терапии ФГБОУ ВО СЗГМУ им. И.И. Мечникова, Санкт-Петербург, e-mail: bykovaelenag@mail.ru, ORCID ID: <https://orcid.org/0000-0001-9902-2338>

Болдуева Светлана Афанасьевна — д.м.н., профессор, заведующая кафедрой факультетской терапии ФГБОУ ВО СЗГМУ им. И.И. Мечникова, Санкт-Петербург, e-mail: svetlanaboldueva@mail.ru, ORCID ID: <https://orcid.org/0000-0002-1898-084X>

Реснянская Екатерина Денисовна — студентка 6 курса лечебного факультета ФГБОУ ВО СЗГМУ им. И.И. Мечникова, Санкт-Петербург, e-mail: katerina.resn_7@mail.ru, ORCID ID: <https://orcid.org/0000-0001-7889-3679>

Information about the authors

Dmitrii S. Evdokimov — Assistant of the Department of Faculty Therapy of I.I. Mechnikov NWSMU, St. Petersburg, e-mail: kasabian244@gmail.com, ORCID ID: <https://orcid.org/0000-0002-3107-1691>

Elena G. Bykova — Candidate of Medical Sciences, Associate Professor of the Department of Faculty Therapy of I.I. Mechnikov NWSMU, St. Petersburg, e-mail: bykovaelenag@mail.ru, ORCID ID: <https://orcid.org/0000-0003-4161-35>.

Svetlana A. Boldueva — Doctor of Medical Sciences, Professor, Head of the Department of Faculty Therapy of I.I. Mechnikov NWSMU, St. Petersburg, e-mail: svetlanaboldueva@mail.ru, ORCID ID: <https://orcid.org/0000-0002-1898-084X>.

Ekaterina D. Resnyanskaya — 6th year student of the Medical Faculty of the I.I. Mechnikov NWSMU, St. Petersburg, e-mail: katerina.resn_7@mail.ru, ORCID ID: <https://orcid.org/0000-0001-7889-3679>

 Автор, ответственный за переписку / Corresponding author