https://doi.org/10.26442/20751753.2024.1.202665

CASE REPORT

Differential diagnosis of amiodarone-induced thyrotoxicosis in a patient with atrial fibrillation and agranulocytosis on the background of thyrostatic therapy. Case report

Kristina Yu. Zherebchikova✉[1], Dmitry S. Bubnov[2], Alexandra S. Ermolaeva[1], Madina O. Soltakhanova[1], Yulia P. Sych[1], Marat V. Ezhov[2], Valentin V. Fadeev[1]

[1]Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia;
[2]Myasnikov Research Institute of Clinical Cardiology, Chazov National Medical Research Center of Cardiology, Moscow, Russia

Abstract
Atrial fibrillation is the most common heart rhythm disorder in patients with chronic heart failure. One of the most effective antiarrhythmic drugs for the treatment and prevention of a wide range of supraventricular and ventricular tachyarrhythmia is amiodarone. In the group of patients with paroxysmal atrial fibrillation and low left ventricular ejection fraction, it is the drug of choice when conducting a „rhythm control" strategy. Patients receiving amiodarone often develop an adverse event – amiodarone-induced thyrotoxicosis, which exacerbates the course of cardiovascular pathology. In this article, we consider a clinical case of amiodarone-induced thyrotoxicosis in a 30-year-old patient with dilated cardiomyopathy and agranulocytosis that developed against the background of thyrostatic therapy.

Keywords: amiodarone, amiodarone-induced thyrotoxicosis, glucocorticoids, thyroid gland, thyrostatics, agranulocytosis, atrial fibrillation, chronic heart failure
For citation: Zherebchikova KYu, Bubnov DS, Ermolaeva AS, Soltakhanova MO, Sych YuP, Ezhov MV, Fadeev VV. Differential diagnosis of amiodarone-induced thyrotoxicosis in a patient with atrial fibrillation and agranulocytosis on the background of thyrostatic therapy. Case report. Consilium Medicum. 2024;26(1):7–11. DOI: 10.26442/20751753.2024.1.202665

КЛИНИЧЕСКИЙ СЛУЧАЙ

Дифференциальная диагностика амиодарон-индуцированного тиреотоксикоза у пациентки с фибрилляцией предсердий и агранулоцитозом на фоне тиреостатической терапии

К.Ю. Жеребчикова✉[1], Д.С. Бубнов[2], А.С. Ермолаева[1], М.О. Солтаханова[1], Ю.П. Сыч[1], М.В. Ежов[2], В.В. Фадеев[1]

[1]ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Минздрава России (Сеченовский Университет), Москва, Россия;
[2]Научно-исследовательский институт клинической кардиологии им. А.Л. Мясникова ФГБУ «Национальный медицинский исследовательский центр кардиологии им. акад. Е.И. Чазова» Минздрава России, Москва, Россия

Аннотация
Фибрилляция предсердий является самым частым нарушением ритма сердца у пациентов с хронической сердечной недостаточностью. Одним из наиболее эффективных антиаритмических препаратов для лечения и профилактики широкого спектра наджелудочковых и желудочковых тахиаритмий является амиодарон. В группе пациентов с пароксизмальной формой фибрилляции предсердий и низкой фракцией выброса левого желудочка он является препаратом выбора при проведении стратегии «контроля ритма». У пациентов, получающих амиодарон, нередко развивается экстракардиальное нежелательное явление – амиодарон-индуцированный тиреотоксикоз, усугубляющий течение сердечно-сосудистой патологии. В статье представляем клинический случай амиодарон-индуцированного тиреотоксикоза у пациентки 30 лет с дилатационной кардиомиопатией и агранулоцитозом, развившимися на фоне тиреостатической терапии.

Ключевые слова: амиодарон, амиодарон-индуцированный тиреотоксикоз, глюкокортикоиды, щитовидная железа, тиреостатики, агранулоцитоз, фибрилляция предсердий, хроническая сердечная недостаточность
Для цитирования: Жеребчикова К.Ю., Бубнов Д.С., Ермолаева А.С., Солтаханова М.О., Сыч Ю.П., Ежов М.В., Фадеев В.В. Дифференциальная диагностика амиодарон-индуцированного тиреотоксикоза у пациентки с фибрилляцией предсердий и агранулоцитозом на фоне тиреостатической терапии. Consilium Medicum. 2024;26(1):7–11. DOI: 10.26442/20751753.2024.1.202665

Information about the authors / Информация об авторах

✉**Kristina Yu. Zherebchikova** – Assistant, Sechenov First Moscow State Medical University (Sechenov University).
E-mail: k.y.zherebchikova@gmail.com; ORCID: 0000-0003-0292-5907

Dmitry S. Bubnov – Res. Assist., Myasnikov Research Institute of Clinical Cardiology, Chazov National Medical Research Center of Cardiology. ORCID: 0000-0003-1538-2761

Alexandra S. Ermolaeva – endocrinologist, Sechenov First Moscow State Medical University (Sechenov University).
ORCID: 0000-0002-6471-8252

✉**Жеребчикова Кристина Юрьевна** – ассистент каф. эндокринологии №1 Института клинической медицины им. Н.В. Склифосовского ФГАОУ ВО «Первый МГМУ им. И.М. Сеченова» (Сеченовский Университет). E-mail: k.y.zherebchikova@gmail.com

Бубнов Дмитрий Сергеевич – мл. науч. сотр. лаб. рентген-эндоваскулярных методов диагностики и лечения в амбулаторных условиях НИИ клинической кардиологии им. А.Л. Мясникова ФГБУ «НМИЦК им. акад. Е.И. Чазова»

Ермолаева Александра Сергеевна – врач-эндокринолог эндокринологического терапевтического отд-ния №2 Университетской клинической больницы №2 ФГАОУ ВО «Первый МГМУ им. И.М. Сеченова» (Сеченовский Университет)

https://doi.org/10.26442/20751753.2024.1.202665

Introduction

Atrial fibrillation (AF) is the most common heart rhythm disorder in patients with chronic heart failure (CHF). Combined, these two conditions worsen the prognosis by increasing the risk of overall mortality and exacerbating the disease symptoms, including increasing the risk of hospitalizations due to CHF decompensation [1]. The prevalence of AF increases with an increase in the functional class of CHF, reaching 50% or more in patients with functional class IV [2]. In addition, the occur-rence of AF in patients who had a sinus rhythm at the time of implantation of the cardiac resynchro-nizer is a not uncommon cause of loss of treatment effectiveness and the CHF decompensation, which requires compliance with the "rhythm control" strategy. Patients with low left ventricular (LV) ejection fraction (EF) have significant limitations in antiarrhythmic therapy options. Also, de-spite the availability of clinical guidelines, approaches to treating such patients remain debatable and thus individualized. Comorbidities in such clinical situations will determine the severity of the condition and require multidisciplinary involvement to choose a treatment approach. Amiodarone, a class III antiarrhythmic agent, is one of the most commonly used agents for treating and preventing a wide range of supraventricular and ventricular tachyarrhythmias [3–7]. In the group of patients with paroxysmal AF and low LVEF, amiodarone is the drug of choice for the "rhythm control" strategy [8]. The drug is also used to treat patients with implanted resynchronizers and cardioverter defibril-lators. The positive effect of amiodarone has also been demonstrated in the treatment of progressive heart failure due to a minimal negative inotropic effect and a relatively insignificant arrhythmogenic effect [9]. However, it is necessary to consider the possibility of non-cardiac side effects, the proba-bility of which increases with the therapy duration [10].

Patients receiving amiodarone often develop adverse events of amiodarone-induced thyrotoxicosis (AmIT). Its prevalence can be as high as 10–20.5%. AmIT is often indistinguishable from spontane-ous thyrotoxicosis; however, clinical signs do not appear in all patients and can be offset by manifes-tations of heart disease. It can occur both in the early period and several months after the start of therapy and even several months after discontinuation of therapy since amiodarone and its metabo-lites have a large volume of distribution and a prolonged half-life due to accumulation in some tis-sues, particularly in adipose tissue. There are two types of AmIT, and making a clear differential di-agnosis between them is important because different treatments are required. Often, verifying the AmIT type is extremely difficult for the clinician. Type 1 AmIT (AmIT1) is due to the increased production of thyroid hormones, while AmIT2 is associated with the release of synthesized and ac-cumulated hormones from thyrocytes due to their destruction. AmIT1 is often associated with pre-existing thyroid disorders (multinodular goiter, latent Graves' disease, etc.), whereas AmIT2 is more common among patients with intact thyroid. The median time of AmIT1 onset is 3.5 months from the start of therapy, while AmIT2 can occur much later, with a median time of onset of 30 months [11].

Clinical manifestations of AmIT may include fatigue, weight loss, increased sweating, and hand tremor. However, due to the anti-adrenergic activity of amiodarone and its inhibitory effect on the conversion of thyroxine (T4) to triiodothyronine (T3), the described symptoms may be alleviated or completely absent. Cardiovascular manifestations become dominant: deterioration in pre-existing cardiac arrhythmias, increased angina attacks, and the onset or worsening of CHF signs.

The diagnosis of AmIT is based on the determination of thyroid hormone levels; there is a de-crease in thyroid-stimulating hormone (TSH) and an increase in free T3 and free T4, antibodies to the TSH receptor (anti-TSHR-Ab) are usually elevated in patients with AmIT1. Ultrasound (US) ex-amination of the thyroid gland in patients with AmIT1 shows increased blood flow, an increase in the thyroid gland volume, and nodules may be observed, while in patients with AmIT2, the vascular-ization of the gland is reduced.

Scanning using sodium pertechnetate 99mTc to assess the absorption capacity of the thyroid gland and determine its functional activity is not always informative since the gland is completely saturat-ed with iodine. Scanning with 99mTc-sestaMIBI is preferable for the differential diagnosis of AmIT types [12].

Since AmIT can lead to increased hospitalization and mortality of patients, achieving euthyroid-ism is the primary goal of the clinician. In AmIT1, thyroid hormone synthesis blockers (thiamazole or propylthiouracil) are indicated. However, the use of these drugs is associated with adverse events such as leukopenia and agranulocytosis. As the first line of AmIT2 therapy, glucocorticoids are used, in particular prednisolone, as it has a membrane-stabilizing and anti-inflammatory effect, inhibits the cytolytic effects of amiodarone, and also reduces the conversion of T4 to the more active form, T3, by inhibiting type 1 deiodinase [13].

A clinical case

Patient N., 30 years old, was admitted to the Department of Infectious Disease with suspected COVID-19 in May 2020 with dyspnea, shortness of breath, palpitations, dizziness, and fever up to 38.5°C for 4 days. She had a history (Fig. 1) of hypertrophic cardiomyopathy diagnosed in 2004 at the age of 14 years. In 2015, she had pneumonia complicated by myocarditis. Subsequently, she not-ed periodic edema of the lower extremities and dyspnea during physical exertion. Echocardiography showed a decrease in LVEF up to 30%, and dilated cardiomyopathy was diagnosed. Since 2018, a complete left bundle branch block (LBBB) has been recorded, for which a three-chamber cardiore-synchronizer-defibrillator was implanted. In 2019, a paroxysm of tachysystolic AF occurred, medi-cally corrected with amiodarone. The drug was administered continuously to maintain sinus rhythm. Over the past year, repeated paroxysms of AF occurred, controlled by electric cardioversion.

Madina O. Soltakhanova – Student, Sechenov First Moscow State Medical University (Sechenov University). ORCID: 0009-0004-2816-0943

Yulia P. Sych – Cand. Sci. (Med.), Sechenov First Moscow State Medical University (Sechenov University). ORCID: 0000-0002-7000-0095

Marat V. Ezhov – D. Sci. (Med.), Department Head, Myasnikov Research Institute of Clinical Cardiology, Chazov National Medical Research Center of Cardiology. ORCID: 0000-0002-1518-6552

Valentin V. Fadeev – D. Sci. (Med.), Prof., Corr. Memb. RAS, Department Head, Sechenov First Moscow State Medical University (Sechenov University). ORCID: 0000-0002-2504-7468

Солтаханова Мадина Омаровна – студентка VI курса Клинического института детского здоровья им. Н.Ф. Филатова ФГАОУ ВО «Первый МГМУ им. И.М. Сеченова» (Сеченовский Университет)

Сыч Юлия Петровна – канд. мед. наук, доц. каф. эндокринологии №1 Института клинической медицины им. Н.В. Склифосовского ФГАОУ ВО «Первый МГМУ им. И.М. Сеченова» (Сеченовский Университет)

Ежов Марат Владиславович – д-р мед. наук, гл. науч. сотр., рук. лаб. нарушений липидного обмена НИИ клинической кардиологии им. А.Л. Мясникова ФГБУ «НМИЦК им. акад. Е.И. Чазова»

Фадеев Валентин Викторович – чл.-кор. РАН, д-р мед. наук, проф., зав. каф. эндокринологии № 1 Института клинической медицины им. Н.В. Склифосовского ФГАОУ ВО «Первый МГМУ им. И.М. Сеченова» (Сеченовский Университет)

Fig. 1. Medical history.

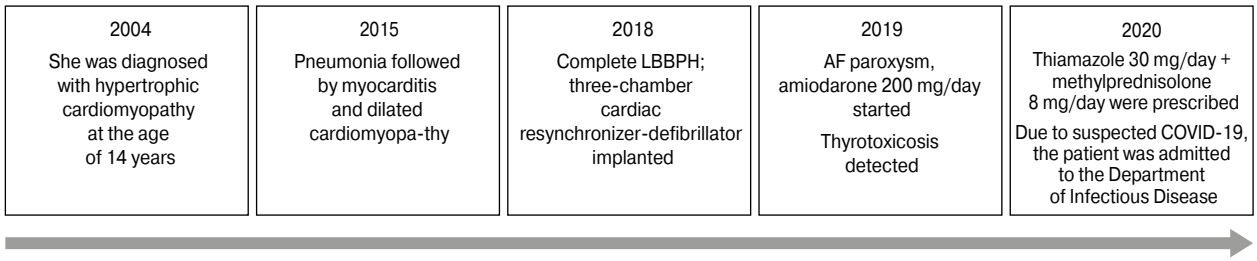
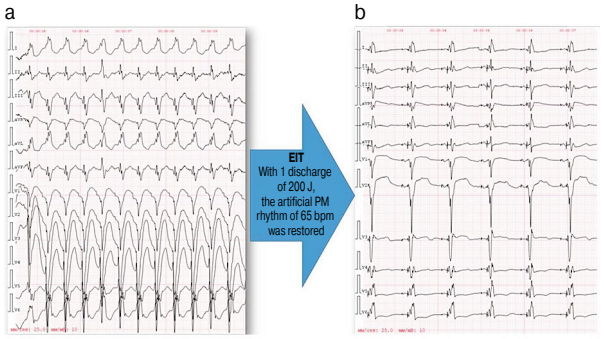


Fig. 2. Ultrasound of the thyroid gland. The thyroid gland volume was not increased, and a reduced echogenicity of the parenchyma was visualized.

Fig. 3. Electrocardiograms before (a) and after (b) the EIT: *a* – AFL with an atrial contraction rate of 271 per minute and a ventricular contraction rate of 2:1; a ventricular contraction rate of 144 per minute; complete LBBPH; *b* – an artificial rhythm of atrial ventricular stimulation, with a heart rate of 65 beats per minute.



Note. PM — pacemaker.

In December 2019, thyrotoxicosis developed during amiodarone therapy. In March 2020, thiama-zole 30 mg/day and methylprednisolone 8 mg/day were administered, followed by a dose reduction to 2 mg/day, which the patient continued to take at the time of admission.

Electrocardiography showed atrial flutter (AFL) with a 140–150 bpm heart rate and a complete LBBPH.

Echocardiography showed dilatation and global systolic dysfunction of all heart chambers and a dif-fuse decrease in the global contractility of the LV myocardium: end-diastolic LV size 64 mm, LV EF ~20–25% (using Simpson method), left atrium 80 mL, TAPSE 9 mm, right ventricular fractional area change 25%, eccentric hypertrophy of the LV myocardium, and signs of high central venous pressure. Complete blood count showed decreased leukocyte levels to 2.3*109 (4.8–10.8) and neutrophils to 0.3*109 (1.9–8.0). Thyrotoxicosis was laboratory-confirmed: TSH <0.01 mIU/L (0.27-4.2), free T4 73.35 pmol/L (12–22), free T3 6.22 pmol/L (3.1-6.8), anti-TSHR-Ab 6.3 mU/L (0.3–1.75). The thyroid gland ultrasound showed the total volume within normal limits with decreased parenchyma echogenicity and normal vascularization (Fig. 2).

Upon admission, no signs of pneumonia were detected according to chest computed tomography. Negative results of the polymerase chain reaction test for SARS-CoV-2 (COVID-19) were obtained twice. During the first day of hospital stay, tachysystolic AFL and a pronounced decrease in the global contractile function of the LV, a progressive deterioration of the condition was observed with the rapid decompensation of heart failure. Electro-impulse therapy (EIT) was used with the restora-tion of the artificial rhythm with atrioventricular stimulation of 68 beats per minute, which contrib-uted to the rapid stabilization of the patient's condition (Fig. 3). Subsequently, during antiarrhythmic therapy with sotalol 320 mg/day, arrhythmia did not recur. According to current clinical guidelines, basic therapy was used for patients with CHF, including angiotensin-converting enzyme inhibitors, mineralocorticoid receptor antagonists, and loop diuretics [14]. During the therapy, heart failure compensation was achieved.

A patient with suspected viral pneumonia was diagnosed with agranulocytosis, and therefore, thi-amazole therapy was discontinued for the treatment of life-threatening conditions, and prednisolone was started at a dose of 30 mg/day. A serial complete blood count after 3 days showed an improve-ment; leukocytes and neutrophils increased; therefore, it was decided against using hematopoietic stimulants.

Thyroid hormone levels subsequently normalized. In 2020, at the Shumakov Federal Research Center of Transplantology and Artificial Organs, orthotopic heart transplantation and a pacemaker explantation were performed using the bicaval technique and cardiopulmonary bypass. After the surgery, the patient received monitoring, standard cardiovascular therapy, and immunosuppression. By December 2023, the patient's condition was stable; the graft function was satisfactory, and the expected clinical effect was achieved: tolerance to physical activity increased, the general condition improved significantly, and euthyroidism persisted.

Discussion

When diagnosing AmIT, it is crucial to determine its type, which is a decisive factor in the choice of therapy. For this purpose, radioisotope scanning (thyroid scintigraphy) is usually used. However, it is not always possible to reliably determine the

nature of the process in the thyroid tissue. Consid-ering the low effectiveness of thyrostatic therapy (the continuing increased level of free T4 during thiamazole therapy at 30 mg for 2 months, the reduced echogenicity of the thyroid parenchyma, and the absence of increased vascularization according to ultrasound, despite the increased level of anti-TSHR-Ab), the patient's condition was regarded as AmIT2 (destructive thyroiditis). The lack of im-provement during glucocorticosteroid therapy at the beginning of treatment is most likely due to the insufficient dose of the drug and its early withdrawal.

Possible causes of agranulocytosis include the myelotoxic effect of thiamazole since its use in pa-tients with Graves' disease leads to agranulocytosis in 0.2–0.5% of cases [15] and concomitant viral infection. The detection of anti-TSHR-Ab in patients with AmIT does not necessarily indicate hy-perfunction of the thyroid gland and can be observed in patients with a destructive type of AmIT [16].

The absence of signs of pneumonia according to computed tomography, as well as the twice-negative result of the polymerase chain reaction test for SARS-CoV-2 (COVID-19), further con-firmed the concept that the clinical symptoms developed in the patient were due to progression of heart failure, and not viral pneumonia. After successful cardioversion, the patient was able to effec-tively maintain sinus rhythm during sotalol therapy. The "rhythm control" strategy stabilized hemo-dynamic parameters and achieved compensation for CHF. Given the critical decrease in the systolic function of the heart during the most tolerated drug therapy and the extremely high risk of fatal events, it was decided to perform a heart transplant. This approach is preferred since, despite the modern possibilities of drug therapy, the only radical method of treating the terminal stage of CHF is orthotopic heart transplantation, i.e., transplantation of a donor's heart into the recipient's chest after removal of the affected heart.

The bicaval technique was chosen for our patient due to several advantages that affect overall sur-vival. In particular, this method provides better preservation of the morphology and function of the right atrium, thereby reducing the frequency of atrial dilation, the need for electrical stimulation, and the occurrence of supraventricular arrhythmias and tricuspid regurgitation [17].

Conclusion

In real-world clinical practice, the combination of AF and CHF is always extremely unfavorable, especially in comorbid patients. AmIT is a challenging diagnostic and therapeutic task, especially in patients with comorbidities. Combination therapy with glucocorticoids and thyrostatics is often used to treat undiagnosed forms of AmIT, which can be associated with complications such as agranulo-cytosis. This clinical case demonstrates the challenges of verifying the thyrotoxicosis type and choosing an optimal approach for managing patients with severe comorbidities.

The involvement of an interdisciplinary team helped choose the optimal treatment promptly and achieve the desired clinical effect.

The authors acknowledge the contribution of Leyla Omarovna Soltakhanova, a neurologist of the Neurological Department No. 9 of the Botkin Hospital.

Disclosure of interest. The authors declare that they have no competing interests.

Раскрытие интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Authors' contribution. The authors declare the compliance of their authorship according to the international ICMJE criteria. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

Вклад авторов. Авторы декларируют соответствие своего авторства международным критериям ICMJE. Все авторы в равной степени участвовали в подготовке публикации: разработка концепции статьи, получение и анализ фактических данных, написание и редактирование текста статьи, проверка и утверждение текста статьи.

Funding source. The authors declare that there is no external funding for the exploration and analys

Источник финансирования. Авторы декларируют отсутствие внешнего финансирования для проведения исследования и публикации статьи.

is work.

Consent for publication. Written consent was obtained from the patient for publication of relevant medical information and all of accompanying images within the manuscript.

Информированное согласие на публикацию. Пациент подписал форму добровольного информированного согласия на публикацию медицинской информации.

References/Литература

1. Verma A, Kalman JM, Callans DJ. Treatment of Patients With Atrial Fibrillation and Heart Failure With Reduced Ejection Fraction. *Circulation.* 2017;135(16):1547-63. DOI:10.1161/CIRCULATIONAHA.116.026054
2. Осмоловская Ю.Ф., Романова Н.В., Жиров И.В., Терещенко С.Н. Эпидемиология и особенности терапии хронической сердечной недостаточности в сочетании с фибрилляцией предсердий. *Медицинский Совет.* 2016;(10):93-7 [Osmolovskaya YF, Romanova NV, Zhirov IV, Tereschenko SN. Epidemiology and management of heart failure patients with atrial fibrillation. *Medical Council.* 2016;(10):93-7 (in Russian)]. DOI:10.21518/2079-701X-2016-10-93-97
3. Hindricks G, Potpara T, Dagres N, et al; ESC Scientific Document Group. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC. *Eur Heart J.* 2021;42(5):373-498. DOI:10.1093/eurheartj/ehaa612
4. Аракелян М.Г., Бокерия Л.А., Васильева Е.Ю., и др. Фибрилляция и трепетание предсердий. Клинические рекомендации 2020. *Российский кардиологический журнал.* 2021;26(7):4594 [Arakelyan MG, Bockeria LA, Vasilieva EYu, et al. 2020 Clinical guidelines for Atrial fibrillation and atrial flutter. *Russian Journal of Cardiology.* 2021;26(7):4594 (in Russian)]. DOI:10.15829/1560-4071-2021-4594
5. Puzanov I, Diab A, Abdallah K, et al.; Society for Immunotherapy of Cancer Toxicity Management Working Group. Managing toxicities associated with immune checkpoint inhibitors: consensus recommendations from the Society for Immunotherapy of Cancer (SITC) Toxicity Management Working Group. *J Immunother Cancer.* 2017;5(1):95. DOI:10.1186/s40425-017-0300-z
6. Connolly SJ. Evidence-based analysis of amiodarone efficacy and safety. *Circulation.* 1999;100(19):2025-34. DOI:10.1161/01.cir.100.19.2025
7. Dan GA, Martinez-Rubio A, Agewall S, et al.; ESC Scientific Document Group. Antiarrhythmic drugs-clinical use and clinical decision making: a consensus document from the European Heart Rhythm Association (EHRA) and European Society of Cardiology (ESC) Working Group on Cardiovascular Pharmacology, endorsed by the Heart Rhythm Society (HRS), Asia-Pacific Heart Rhythm Society (APHRS) and International Society of Cardiovascular Pharmacotherapy (ISCP). *Europace.* 2018;20(5):731-2an. DOI:10.1093/europace/eux373
8. Deedwania PC, Singh BN, Ellenbogen K, et al. Spontaneous conversion and maintenance of sinus rhythm by amiodarone in patients with heart failure and atrial fibrillation: observations from the veterans affairs congestive heart failure survival trial of antiarrhythmic therapy (CHF-STAT). The Department of Veterans Affairs CHF-STAT Investigators. *Circulation.* 1998;98(23):2574-9. DOI:10.1161/01.cir.98.23.2574.
9. Martino E, Bartalena L, Bogazzi F, Braverman LE. The effects of amiodarone on the thyroid. *Endocr Rev.* 2001;22(2):240-54. DOI:10.1210/edrv.22.2.0427
10. Ruzieh M, Moroi MK, Aboujamous NM, et al. Meta-Analysis Comparing the Relative Risk of Adverse Events for Amiodarone Versus Placebo. *Am J Cardiol.* 2019;124(12):1889-93. DOI:10.1016/j.amjcard.2019.09.008
11. Tomisti L, Rossi G, Bartalena L, et al. The onset time of amiodarone-induced thyrotoxicosis (AIT) depends on AIT type. *Eur J Endocrinol.* 2014;171(3):363-8. DOI:10.1530/EJE-14-0267
12. Pattison DA, Westcott J, Lichtenstein M, et al. Quantitative assessment of thyroid-to-background ratio improves the interobserver reliability of technetium-99m sestamibi thyroid scintigraphy for investigation of amiodarone-induced thyrotoxicosis. *Nucl Med Commun.* 2015;36(4):356-62. DOI:10.1097/MNM.0000000000000260

13. Tauveron I, Batisse-Lignier M, Maqdasy S. Enjeux liés à l'hyperthyroïdie induite par l'amiodarone [Challenges in the management of amiodarone-induced thyrotoxicosis]. *Presse Med.* 2018;47(9):746-56 [Article in French]. DOI:10.1016/j.lpm.2018.09.001.
14. Мареев В.Ю., Фомин И.В., Агеев Ф.Т., и др. Клинические рекомендации ОССН – РКО – РНМОТ. Сердечная недостаточность: хроническая (ХСН) и острая декомпенсированная (ОДСН). Диагностика, профилактика и лечение. *Кардиология.* 2018;58(Suppl. 6):8-158 [Mareev VYu, Fomin IV, Ageev FT, et al. Russian Heart Failure Society, Russian Society of Cardiology. Russian Scientific Medical Society of Internal Medicine Guidelines for Heart failure: chronic (CHF) and acute decompensated (ADHF). Diagnosis, prevention and treatment. *Kardiologiia.* 2018;58(Suppl. 6):8-158 (in Russian)]. DOI:10.18087/cardio.2475
15. Vicente N, Cardoso L, Barros L, Carrilho F. Antithyroid Drug-Induced Agranulocytosis: State of the Art on Diagnosis and Management. *Drugs R D.* 2017;17(1):91-6. DOI:10.1007/s40268-017-0172-1
16. Cappellani D, De Marco G, Ferrarini E, et al. Identification of Two Different Phenotypes of Patients with Amiodarone-Induced Thyrotoxicosis and Positive Thyrotropin Receptor Antibody Tests. *Thyroid.* 2021;31(10):1463-71. DOI:10.1089/thy.2021.0118
17. Toscano G, Bottio T, Gambino A, et al. Orthotopic heart transplantation: the bicaval technique. *Multimed Man Cardiothorac Surg.* 2015;2015:mmv035. DOI:10.1093/mmcts/mmv035

The article received / Статья поступила в редакцию: 04.02.2024
The article approved for publication / Статья принята к печати: 18.03.2024