



A case report of surgical treatment of amiodarone-induced thyrotoxicosis in a patient with multiple organ failure

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Received: June 2, 2022. Received in revised form: July 7, 2022. Accepted: July 18, 2022.

Abstract

Amiodarone is a drug used in the treatment of life-threatening arrhythmias, which can lead to the development of amiodarone-induced thyrotoxicosis. In most cases this pathology can be treated by conservative methods; surgical treatment is resorted to in cases of thyrotoxicosis refractory to medical treatment.

This case report describes surgical treatment of a patient with amiodarone-induced thyrotoxicosis, progressive heart failure, neurological pathology, bilateral pneumonia, functioning tracheostomy, systemic infectious process, multiple organ dysfunction syndrome, who was treated in the intensive care unit. Due to the lack of response to therapy with antithyroid drugs (thiamazole, lithium preparations and pulse therapy with prednisolone) and a progressive deterioration of the condition in a short period of time, according to vital indicators, the patient underwent thyroidectomy. In the postoperative period, there was a decrease in the occurrence of chronic heart failure symptoms. Medical control of cardiac arrhythmias was achieved. Surgical stage proceeded without complications in the period of 30-days. The patient was discharged for outpatient rehabilitation treatment.

Keywords: amiodarone induced thyrotoxicosis, amiodarone, thyroidectomy, heart failure, tracheostomy

Cite this article as: Salov M.A., Shulyakovskaya A.S., Danilov I.N., Glebovskaya T.D., Lapshin K.B., Dalmatova A.B., Tsvetkova E.V., Nasedkin D.B., Kovalev A.A., Lapshina S.E., Neymark A.E. A case report of surgical treatment of amiodarone-induced thyrotoxicosis in a patient with multiple organ failure. *Innovative Medicine of Kuban.* 2022;(3):58–63. <https://doi.org/10.35401/2541-9897-2022-25-3-58-63>

Клинический случай хирургического лечения амиодарон-индуцированного тиреотоксикоза у пациента с полиорганной недостаточностью

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Поступила в редакцию 2 июня 2022 г. Исправлена 7 июля 2022 г. Принята к печати 18 июля 2022 г.

Резюме

Амиодарон – это препарат, применяемый в лечении жизнеугрожающих аритмий, прием которого может приводить к развитию амиодарон-индуцированного тиреотоксикоза. Чаще всего лечение данной патологии осуществляется консервативными методами, к хирургическому лечению прибегают в случае рефрактерного к медикаментозному лечению тиреотоксикоза.

В данном клиническом примере описан случай хирургического лечения пациентки с амиодарон-индуцированным тиреотоксикозом, прогрессирующей сердечной недостаточностью, неврологической патологией, двухсторонней пневмонией, функционирующей трахеостомой, системным инфекционным процессом, синдромом полиорганной дисфункции, которая находилась на лечении в отделении интенсивной терапии. В связи с отсутствием ответа на терапию антигипертензивными препаратами (тиамазолом, препаратами лития и пульс-терапией преднизолоном) и прогрессирующим ухудшением состояния в короткие сроки по жизненным показаниям больной выполнена тиреоидэктомия. В послеоперационном периоде отмечено уменьшение проявлений хронической сердечной недостаточности. Достигнут медикаментозный контроль нарушений ритма сердца. Хирургический этап протекал без осложнений в 30-дневный период. Пациентка выписана на амбулаторное восстановительное лечение.

Ключевые слова: амиодарон-индуцированный тиреотоксикоз, амиодарон, тиреоидэктомия, сердечная недостаточность, трахеостомия



Цитировать: Салов М.А., Шуляковская А.С., Данилов И.Н., Глебовская Т.Д., Лапшин К.Б., Далматова А.Б., Цветкова Е.В., Наседкин Д.Б., Ковалев А.А., Лапшина С.Е., Неймарк А.Е. Клинический случай хирургического лечения амиодарон-индуцированного тиреотоксикоза у пациента с полиорганной недостаточностью. *Инновационная медицина Кубани*. 2022;(3): 58–63. <https://doi.org/10.35401/2541-9897-2022-25-3-58-63>

Introduction

Amiodarone is one of the most effective and commonly prescribed antiarrhythmic drugs. Due to the high content of iodine, this drug affects the thyroid hormone metabolism [1].

According to the literature data, the use of amiodarone leads to dysfunction of the thyroid gland in 15–20% of cases, while the incidence of amiodarone-induced thyrotoxicosis (AIT) ranges from 3 to 15% [2–6]. Commonly the amiodarone-induced thyrotoxicosis is classified into types 1, 2, and a mixed form. Thyroidectomy is an effective treatment for drug-refractory AIT, but its use is considered to be limited due to higher perioperative morbidity and mortality compared to thyroidectomy performed for Graves' disease or toxic multinodular goiter [7]. According to a cohort study that has been conducted at the Mayo Clinic (USA) for 14 years, out of 370 cases of AIT, thyroidectomy was performed only in 17 patients (4.6%) [4].

However, in a number of studies, the authors note a low incidence of perioperative complications of thyroidectomy in patients with AIT refractory to drug therapy, which may be due to the presence of a multidisciplinary team in these clinics and the peculiarities of the anesthetic service [8–10]. Thus, in a retrospective study performed at the St Vincent's Hospital Heart Lung Transplant Unit (Australia), thyroidectomy was performed in 24 patients with AIT. According to echocardiography, in 38% of patients at the time of surgery a decrease in the ejection fraction (EF) of the left ventricle was less than 30%. No cardiorespiratory complications or lethal outcomes were recorded in the postoperative period [11].

According to a retrospective study by D. Cappellani et al., covering a 20-year period, the level of overall mortality and mortality due to cardiovascular diseases after 5 and 10 years in patients undergoing surgical treatment for AIT was statistically significantly lower than in patients who received drug therapy. This difference was most pronounced in patients with intermediate and low left ventricular EF, while no differences were obtained in patients with normal EF [12].

According to 2018 European Thyroid Association Guidelines for the Management of Amiodarone-Associated Thyroid Dysfunction thyroidectomy as a treatment for AIT can be considered in the following cases: no effect of drug therapy; the need to continue therapy with amiodarone; the development of unwanted side effects of drug therapy; the presence of vital indications in case of worsening of the course of the cardiovascular system disease [3].

This publication demonstrates a case of complex treatment of a patient who was in the intensive care unit with amiodarone-induced thyrotoxicosis and severe comorbid status.

Material and methods

Female patient X., 51 years old, since 2018 has been suffering from atrial fibrillation (AF), manifested by heart work interruptions and general weakness. She was prescribed amiodarone at a dose of 200 mg per day for oral administration and received the drug from October 2018 to July 2019. On July 3rd, 2019, the patient was hospitalized with a clinical picture of paroxysmal AF, it was stopped, recommendations were given to continue amiodarone therapy at the same dose. However, according to the patient, in July 2019 she stopped the intake of amiodarone. The reason for the drug cancellation – whether it was patient's or doctor's decision – has not been clarified. In addition to that, the patient did not control the thyroid status neither before the period of amiodarone intake nor during it. In May 2021, thyrotoxicosis was first detected. According to laboratory tests: thyroid-stimulating hormone (TSH) – 0.005 μ U/ml (0.350–4.940), free thyroxine (FT4) – 90.75 pmol/l (9.0–19.0). According to the thyroid gland ultrasound examination dated June 1st, 2021: the echostructure is heterogeneous due to the alternation of hypo- and hyperechoic areas. The volume of the right lobe is 34.0 ml³, along the posterior surface there is a hyperechoic nodule (pseudonodule?) sized 0.7×0.6×0.55 cm, homogeneous, avascular in the color Doppler imaging. The volume of the left lobe is 25.3 ml³, in the middle part along the anterior surface there is a hyperechoic nodule 1.1×0.7×0.85 cm, with fuzzy, smooth contours, heterogeneous in structure due to anechoic inclusions, in the mode of color Doppler mapping (CDM) – a single perinodular blood flow, TIRADS 3. The total volume of the gland is 59.3 ml³ (the norm is less than 18 ml³). Regional lymph nodes are not located. Vascularization of the thyroid gland in CDM is moderately increased, peak systolic blood flow velocities in the thyroid arteries are changed to 38 cm/sec.

On June 1, 2021, the patient was consulted by an endocrinologist at the Almazov National Medical Research Centre on the topic of amiodarone-induced thyrotoxicosis type 1 or Graves' disease. Treatment with thiamazole at a dose of 30 mg per day was recommended, therapy was initiated on June 2, 2021. The patient stopped taking the drug on her own initiative 5 days later.

On June 21, 2021, the patient arrived at the Almazov National Medical Research Centre for hospitalization in

the endocrinology department. During the examination among her complaints were shortness of breath both at rest and during minimal physical activity, palpitations, occasional body tremor, lower extremities swelling, an increase in the abdomen volume.

Atrial fibrillation, tachysystole with a heart rate of up to 180 beats per minute, decompensated chronic heart failure up to functional class IV (shortness of breath at rest, ascites, anasarca, lower extremities edema) were diagnosed. The patient was admitted to the intensive care unit.

According to echocardiography data, the EF upon admission according to Simpson's method equaled 31%, the global left and right ventricles contractility is severely reduced due to diffuse hypokinesia, the estimated pulmonary artery systolic pressure ranges from 40 to 45 mm Hg, the inferior vena cava is dilated, the inspiratory collapse is less than 20%. According to the examination data, dated June 21st, 2021, free triiodothyronine (T3) – 5.15 pmol/l (2.63–5.69), FT4 – 93.07 pmol/l, TSH – 0.001 mIU/l. Antibodies to TSH receptors – 13.94 IU/l (< 1.75), which confirmed the presence of Graves' disease. Therapy with thiamazole at a dose of 30 mg per day was resumed.

After the condition stabilization and some decrease in the manifestations of heart failure on June 25, 2021, the patient was transferred to the cardiology department of the Center, where she was under the supervision of an endocrinologist. However, despite the ongoing therapy with thiamazole, there was an increase in FT4 and T3 levels, the dosage of the drug was increased to 40 mg per day, oral therapy with prednisolone at a dose of 30 mg per day was started. Against the background of combination therapy, the levels of T3 and FT4 decreased.

On July 4, 2021, the patient developed dysarthria and left-sided hemiparesis. According to computed tomography (CT) of the brain: CT signs of a formed zone of an acute cerebrovascular accident (CVA) in the right temporal-parietal-occipital area and the insular region on the right (middle cerebral artery) sized 6.2×3.5×5.7 cm. The patient was transferred to the intensive care unit for treatment. As of July 5, 2021, thyrotoxicosis persisted without significant changes in the level of thyroid hormones: TSH – 0.001 mIU/l, FT4 – 62.9 pmol/l, T3 – 12.08 pmol/l. According to the data of 24-hour cardiac monitoring dated July 6, 2021, AF persisted with a frequency of 161 to 190 (average 176) beats/min during the entire observation period.

Considering the acute period of extensive cerebrovascular accident, it was decided to reduce the dose of prednisolone to 20 mg/day. For uncontrolled thyrotoxicosis in a patient with severe tachysystole, lithium carbonate at a dose of 900 mg/day was added to therapy.

Since July 9, 2021, the presence of febrile fever has been noted, at the same time an increase in markers of a systemic inflammatory reaction was registered, gram-positive cocci were detected in the blood culture.

Bilateral polysegmental pneumonia was diagnosed according to the chest organs CT. The patient was prescribed antibiotic therapy according to the identified sensitivity of microorganisms. Two days later, a tracheostomy was performed.

Against the background of an ongoing multicomponent therapy (thiamazole, prednisolone, lithium carbonate), during the control on July 12, 2021, positive dynamics was noted: T3 – 7.56 pmol/l, FT4 – 28.3 pmol/l. Decrease in heart rate to 110–120 beats/min was clinically noted, as well as regression of heart failure phenomena. According to the results of echocardiography, the EF increase according to Simpson's method was up to 45%. Taking into account the generalized infectious process, it was decided to reduce the dose of oral prednisolone to 10 mg/day. On the fifth day from the moment of dose reduction, a significant increase in the thyroid hormones levels was noted, mainly due to FT4 from 28.3 pmol/l dated July 12, 2021, to 128.7 pmol/l dated July 16, 2021; T3 from 7.56 pmol/l dated July 7, 2021, to 30.6 pmol/l dated July 16, 2021. The ratio of FT4/T3 was 4.18. There was an idea about the current destructive process in the thyroid gland and the progression of "leak" thyrotoxicosis.

Considering the severity of the patient's condition, the presence of a tracheostomy, and swallowing disorders, it was decided to administer prednisolone intravenously at a dose of 180 mg/day. Lithium carbonate therapy was canceled, thiamazole therapy was continued at the same dose. Against the background of thyrotoxicosis relief and heart rate control, regression of heart failure was noted: according to echocardiography, the EF increase according to Simpson's method was up to 45%. However, with a decrease of the dose of prednisolone, a relapse of thyrotoxicosis occurred again.

On July 29, 2021, a multidisciplinary consultation was held. A patient with clinical symptoms of sepsis, an ongoing infectious process in the form of mucopurulent endobronchitis with a significant titer of multiresistant flora in bronchoalveolar lavage cultures and a high risk of systemic infectious process progression, received confirmed indications for urgent thyroidectomy. These indications were determined due to the need to stop thyrotoxicosis, which is adequately resistant to drug treatment, as well as due to the need of expanding the possibilities of antiarrhythmic therapy and reducing the duration of immunosuppressive therapy with glucocorticosteroids.

It was decided to refrain from preoperative fine-needle aspiration biopsy of the left lobe node of the thyroid gland due to the presence of a tracheostomy and, therefore, the technical complexity of the manipulation. It should be noted that despite the listed above diagnoses and a pronounced neurological deficit and intoxication, at the time of the examination the patient was conscious, sane and oriented in time, which made it possible to obtain consent to the intervention.

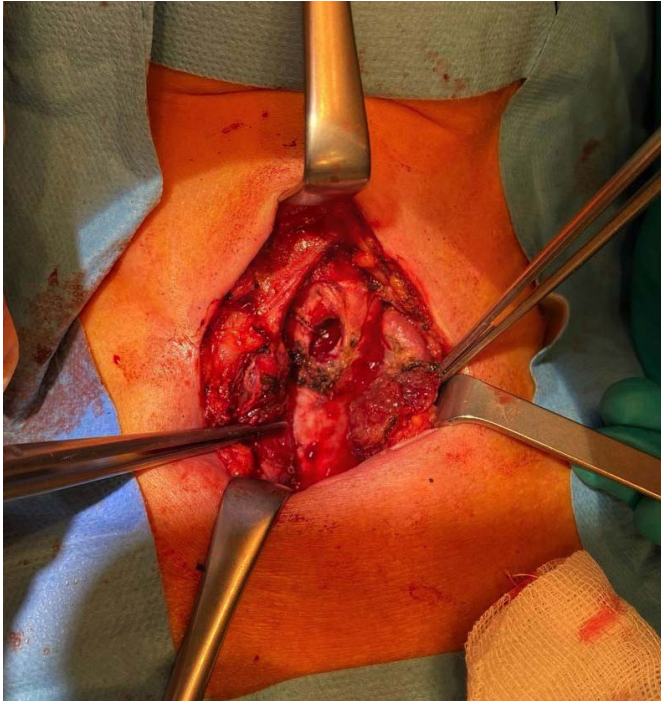


Figure 1. Surgical wound. The tracheostomy defect, transected isthmus of the thyroid gland

Рисунок 1. Операционная рана – трахеостомический дефект, пересеченный перешеек щитовидной железы

Results

On August 4, 2021, a thyroidectomy was performed. An ENT doctor was present during the operation. After anesthesia induction, bronchoscopy was performed through a tracheostomy cannula. The visible part of the trachea is freely passable, the bronchi lumen can be traced, contain a moderate amount of serous-mucous liquid sputum – diffuse, catarrhal endobronchitis. During tracheal intubation, the tracheostomy cannula was removed. Access to the thyroid gland was performed according to Kocher's method with complete excision of the edges of the tracheostomy canal up to the trachea. The defect in the trachea was located above the thyroid gland isthmus (figure 1). During the examination the right lobe of the thyroid gland was 7.0×4.0×3.0 cm, the left lobe was 6.0×3.0×2.5 cm, the isthmus was also enlarged. The thyroid gland was mobilized extrafascially using a Ligasure bipolar coagulator. The isthmus of the gland was crossed for the purpose of the staged removal of its lobes. The recurrent laryngeal nerves and most of the parathyroid glands are visualized in typical places. The superior and inferior thyroid vessels are ligated. A thyroidectomy was performed, a drain was installed in the pretracheal space, brought out through the counter-opening just above the jugular notch, and connected to a vacuum.

The patient was discussed intraoperatively with the participants of the council. Considering the risk of developing acute respiratory failure in the early postoperative period, that is associated with a previous stroke and

a complicated course of the disease, it was decided to keep the tracheostomy. Layered wound suturing was performed with the formation of a new tracheostomy canal with the separation of the latter from the paratracheal and subcutaneous spaces. During extubation of the trachea, it was cannulated with a tracheostomy tube of the same diameter.

The postoperative period proceeded without surgical complications (figure 2). Laboratory monitoring on the first day: parathyroid hormone – 47.7 pg/ml (15–65 pg/ml), ionized calcium – 1.23 mmol/l (1.16–1.32). Hormone replacement therapy with levothyroxine started at a dose of 50 mcg, therapy with prednisolone continued at a dose of 120 mg per day. On the second day after the surgery, the dose of prednisolone was reduced to 60 mg per day, the dose of levothyroxine was increased to 75 mcg. On the third day after the surgery, prednisolone was discontinued, however, due to occurring symptoms of adrenal insufficiency, on the fifth day after the operation, the drug was resumed at a dose of 60 mg per day. During the further treatment the dose of prednisolone was reduced within 2 weeks.

On the 8th day after the surgery, direct laryngoscopy was performed: the vocal cords are located symmetrically, closing during phonation. After control bronchoscopy, the trachea was decannulated. On the 10th day after the surgery, the patient was transferred to the cardiology department.

According to the results of control echocardiography, the EF increase according to Simpson's method was up to 55%. Against the background of hormone replacement therapy with levothyroxine at a dose of 75 mcg on the twelfth day after the surgery, the level of FT4 was 15.3 pmol/l.

The patient was discharged on the 30th day after surgery in a hemodynamically and clinically stable condition, on therapy with antiarrhythmic drugs and prednisolone at



Figure 2. Postoperative wound on the 13th day after surgical treatment

Рисунок 2. Вид послеоперационной раны на 13-е сутки после оперативного вмешательства

a dose of 2.5 mg per day. The tracheostomy defect closed on the twenty-seventh day after the surgery (figure 3). According to the results of histological examination of the surgical material – a diffuse toxic nodular goiter that occurred during the therapy.

During telephone contact with the patient 5 months after discharge, data were obtained on the patient's satisfactory condition, the successful increase of physical activity (walking). Against the background of levothyroxine replacement therapy euthyroidism occurred (TSH – 2.5 mIU/l).

Discussion

This clinical case report presents a successful case of surgical treatment of an ongoing amiodarone-induced thyrotoxicosis in a tracheostomy resuscitation patient with occurring symptoms of multiple organ dysfunction syndrome of varying compensation degrees. In foreign and Russian literature, we did not find publications about such experience, although, undoubtedly, such interventions were carried out.

Conclusion

Despite the fact that in most cases AIT is treated conservatively, surgical treatment may be the only method for correcting this pathology.

Treatment of this category of patients, in our opinion, is possible only in a multidisciplinary hospital with the possibility of holding a multidisciplinary consultation.

Performing thyroidectomy in the conditions of the ongoing systemic inflammatory process against the background of a functioning tracheostomy is possible if adequate surgical principles and rationally selected antibiotic therapy are followed.

Thyroidectomy for AIT, performed according to indications, has proven efficacy in the treatment of thyrotoxic

cardiomyopathy and its manifestations. In this particular case, a permanent form of atrial fibrillation and chronic heart failure.

References/Литература

1. Trohman RG, Sharma PS, McAninch EA, et al. Amiodarone and thyroid physiology, pathophysiology, diagnosis and management. *Trends in cardiovascular medicine*. 2019;29(5):285–295. <https://doi.org/10.1016/j.tcm.2018.09.005>
2. Grineva EN, Tsoy UA, Karonova TL, et al. Draft of the Federal Clinical Recommendations for diagnosis and treatment of amiodarone-induced thyroid dysfunction. *Clinical and experimental thyroidology*. 2020;16(2):12–24. (In Russ.). <https://doi.org/10.14341/ket12693>
- Гринева Е.Н., Цой У.А., Каронова Т.Л. и др. Проект федеральных клинических рекомендаций по диагностике и лечению амиодарон-индуцированной дисфункции щитовидной железы. *Клиническая и экспериментальная тиреология*. 2020;16(2):12–24. <https://doi.org/10.14341/ket12693>
3. Bartalena L, Bogazzi F, Chiovato L, et al. 2018 European Thyroid Association (ETA) guidelines for the management of amiodarone-associated thyroid dysfunction. *Eur Thyroid J*. 2018;7(2):55–66. <https://doi.org/10.1159/000486957>
4. Kotwal A, Clark J, Lyden M, et al. Thyroidectomy for amiodarone-induced thyrotoxicosis: Mayo Clinic Experience. *J Endocr Soc*. 2018;2(11):1226–1235. <https://doi.org/10.1210/js.2018-00259>
5. Maqdasy S, Benichou T, Dallel S, et al. Issues in amiodarone-induced thyrotoxicosis: update and review of literature. *Ann Endocrinol (Paris)*. 2019;80(1):54–60. <https://doi.org/10.1016/j.ando.2018.05.001>
6. Kinoshita S, Hayashi T, Wada K, et al. Risk factors for amiodarone-induced thyroid dysfunction in Japan. *Journal of Arrhythmia*. 2016;32(6):474–480. <https://doi.org/10.1016/j.joa.2016.03.008>
7. Houghton SG, Farley DR, Brennan MD, et al. Surgical management of amiodarone-associated thyrotoxicosis: Mayo Clinic experience. *World J Surg*. 2004;28:1083–1087. <https://doi.org/10.1007/s00268-004-7599-6>
8. Tomisti L, Materazzi G, Bartalena L, et al. Total thyroidectomy in patients with amiodarone-induced thyrotoxicosis and severe left ventricular systolic dysfunction. *J Clin Endocrinol Metab*. 2012;97:3515–3521. <https://doi.org/10.1210/jc.2012-1797>
9. Patel N, Inder WJ, Sullivan C, et al. An audit of amiodarone-induced thyrotoxicosis – do anti-thyroid drugs alone provide adequate treatment? *Heart Lung Circ*. 2014;23:549–554. <https://doi.org/10.1016/j.hlc.2014.01.013>
10. Gough J, Gough IR. Total thyroidectomy for amiodarone-associated thyrotoxicosis in patients with severe cardiac disease. *World J Surg*. 2006;30:1957–1961. <https://doi.org/10.1007/s00268-005-0673-x>
11. Isaacs M, Costin M, Bova R, et al. Management of amiodarone-induced thyrotoxicosis at a cardiac transplantation centre. *Frontiers in Endocrinology*. 2018;9:1–8. <https://doi.org/10.3389/fendo.2018.00482>
12. Cappellani D, Papini P, Pingitore A, et al. Comparison Between Total Thyroidectomy and Medical Therapy for Amiodarone-Induced Thyrotoxicosis. *J Clin Endocrinol Metab*. 2020;105(1):242–251. <https://doi.org/10.1210/clinem/dgz041>

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Figure 3. Postoperative wound on the day of discharge from the hospital

Рисунок 3. Вид послеоперационной раны в день выписки из больницы

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Conflict of interest: none declared.

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Конфликт интересов

Авторы заявляют об отсутствии конфликта интересов.