




Amiodarone – thyroid – arrhythmia. Difficult cooperation between endocrinologist and cardiologist

A patient with amiodarone-induced thyroid disturbances

Amiodaron – tarczycza – arytmia. Taniec na linie endokrynologa i kardiologa.
Pacjent z poamiodaronowymi zaburzeniami funkcji tarczycy

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Należy cytować wersję pierwotną

Abstract

We present the case of a patient with atrial flutter (treated with cryoablation) and amiodarone-induced hypothyroidism (AIH). Administration of L-thyroxine supplementation triggered a recurrence of arrhythmia. This report shows how difficult it can be to treat a patient with AIH and how important it is to ensure cooperation between cardiologist and endocrinologist.

Key words: amiodarone, atrial flutter, hypothyroidism, AIH

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Introduction

Amiodarone is a drug used in the treatment of supraventricular arrhythmias, and in ventricular arrhythmias as a supplement to electrotherapy. Thyroid dysfunction is a known side effect. In a patient with such complications, treatment becomes a challenge requiring close cooperation between a cardiologist and an endocrinologist, for whom the goals of treatment are not always convergent.

Case study

We present the case of a patient with atrial flutter treated with cryoablation, with thyroid dysfunction occurring after the inception of amiodarone therapy. The patient was an 85 year-old man with coronary disease, pharmacologically controlled hypertension, post-infarction heart failure, diet-controlled type 2 diabetes, and hypercholesterolemia. The patient did not smoke and had a negative family history for atherosclerosis.

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At the age of 77, he suffered a ST-elevation myocardial infarction (STEMI): in urgent coronary angiography:

- left coronary artery distally narrowed to 50–70%;
- left anterior descending artery with marginal changes narrowing the light to 70% on the entire course;
- ostium of the circumflex artery of the coronary artery narrowed to 90%;
- recessive right coronary artery, amputated ostia, periphery fills with its own collaterals.

Primary balloon angioplasty of the circumflex branch was performed. After three days, cardiogenic shock occurred as a result of early restenosis. A stent was placed during re-angioplasty. Left ventricular ejection fraction (LVEF) assessed before discharge was 38%, and in the electrocardiogram (ECG) there were no deviations, except for the features of previous infarction.

A year later, a second myocardial infarction, non-ST-elevation (NSTEMI) occurred. In view of the sub-optimal effect of percutaneous treatment from the previous year, the patient was qualified for surgical revascularisation. An arterial bypass was implanted into the anterior descending branch and venous bypass to the circumflex branch. After the operation, the patient required an intraaortic balloon contrapulsation. At discharge, LVEF was 40%. The patient was prescribed:

- bisoprolol 2.5 mg;
- clopidogrel 75 mg;
- atorvastatin 20 mg;
- pantoprazole 20 mg;
- acetylsalicylic acid 150 mg;
- eplerenone 25 mg.

At the age of 79, paroxysmal atrial tachycardia followed by atrial flutter appeared for the first time. Due to the short-term effectiveness of pharmacotherapy, the patient was

qualified for interventional treatment. The electrophysiological examination revealed an atrial flutter of 270/min. Cryoablation of the cavotricuspid isthmus was performed. After surgery, sinus rhythm was obtained with right branch bundle block and left anterior fascicular block. The thyroid hormone profile was assessed for the first time – all were normal [thyroid-stimulating hormone [TSH] 2.26 mIU/l, free triiodothyronine [fT3] 4.54 pmol/L, free thyroxine [fT4] 21.14 pmol/L). Acenocoumarol was started.

At the age of 84, Holter ECG monitoring performed during the rehabilitation programme recorded elongation of the PQ interval, one episode of ventricular tachycardia (nine evolutions at a frequency of 144/min); and one episode of supraventricular tachycardia (three evolutions at a frequency of 120/min). Asymptomatic arrhythmias did not cause any changes in pharmacological treatment.

At the age of 85, Holter monitoring was repeated, which revealed two episodes of ventricular tachycardia: 27 and six evolutions > 100/min, and seven episodes of supraventricular tachycardia (from three to six evolutions > 100/min). In view of the severity of the arrhythmia, it was decided to permanently use amiodarone 200 mg, while monitoring the thyroid function by an endocrinologist.

Six months after the amiodarone introduction, elongation of the PQ interval to 286 ms and QT interval to 526 ms were recorded (Figure 1). Therefore, the dose of bisoprolol was reduced to 2.5 mg, and amiodarone to 100 mg. In a follow-up study performed two months later, the reduced beta-blocker and amiodarone doses had shortened the PQ interval to 228 ms and the QT interval to 464 ms (Figure 2).

In a control ECG after six months, an extended PQ interval (304 ms), and an extended QT interval (514 ms) were observed. For this reason, the beta-blocker and amiodarone doses were reduced again. After three months of



Figure 1. Electrocardiogram (ECG) of the patient in April 2016: PQ interval 286 ms, QT interval 526 ms; bisoprolol 5 mg, amiodarone 200 mg. ECG recordings and measurements of QTc intervals were made using the ECG Cardiovit MS-12 blue from Schiller (Switzerland)



Figure 2. Electrocardiogram of the patient in June 2016 after reduction of drug doses: PQ interval 228 ms, QT interval 464; bisoprolol 2.5 mg, amiodarone 100 mg



Figure 3. Electrocardiogram of the patient after the start of 50 µg of L-thyroxine

treatment with 1.25 mg bisoprolol and 50 mg amiodarone per day, in ECG the PQ interval was 274 ms and the QT interval was 488 ms.

At the next cardiology follow-up visit, the patient informed a cardiologist that he was taking a new medicine from an endocrinologist and that he often felt fast palpitations. ECG revealed atrial tachycardia with 2:1 conduction. It turned out that L-thyroxine 25 µg/day was started due to the high TSH value of 28 mIU/l. Because TSH value was 18 mIU/l, after the first month of therapy, the dose of L-thyroxine was escalated to 50 µg, which caused re-episodes of paroxysmal atrial tachycardia (Figure 3).

In the case described, the following therapeutic options were considered:

- another ablation – the patient did not agree to re-treatment;
- discontinuation of L-thyroxine – this carried the risks arising from hypothyroidism, which may be one of the causes of QT prolongation;
- discontinuation of amiodarone – this was associated with a risk of worsening the patient’s arrhythmia.

Over the following months, the patient did not appear at the Cardiology Clinic due to a difficult family situation. It turned out that after six months he had discontinued

amiodarone on his own, and L-thyroxine was taken in a dose of 37.5 µg/day. Despite the discontinuation of amiodarone six months earlier, in the ECG the QTc interval was still extended to 554 ms, and sinus rhythm of 76/min was interrupted by episodes of tachycardia 114 /min. However, the patient did not feel any discomfort and refused hospitalisation to optimise his treatment because of the personal situation.

Discussion

In patients with cardiovascular diseases, post-amiodarone thyroid disorders are particularly dangerous because severe hypothyroidism is one of the causes of prolonged QT interval which predisposes to severe ventricular arrhythmias, whereas hyperactivity can exacerbate heart disease and increase mortality [1]. Disorders of thyroid function during the use of amiodarone may occur as a result of

toxic effects of the drug on the thyroid gland or due to the presence of a large amount of iodine, which in some patients leads to the occurrence of hypothyroidism (AIH). Hyperthyroidism may occur as a result of iodine-induced uncontrolled hormone production. This occurs most often in patients with nodular goitre or a silent Graves-Basedow disease [type 1 amiodarone-induced thyroid toxicity (AIT)]. Type 2 hyperthyroidism is the result of destructive thyroiditis in the previously healthy thyroid gland [2]. The prognostic factors for the occurrence of thyroid intestinal disorders are unknown. Retrospective studies indicate that the predictive factor may be the serum ratio of desethylenedioquinone (DEA) – a drug metabolite – to amiodarone (AMD) [3]. In AIT cases, a higher DEA/AMD ratio was found than in AIH patients. Amiodarone is metabolised in the liver via CYP 3A4 [4]. Therefore, the polymorphisms of this enzyme gene and its inducers may influence the probability of thyroid complications.

Streszczenie

W pracy przedstawiono leczonego krioablacją pacjenta z trzepotaniem przedsionków, u którego po włączeniu amiodaronu wystąpiła niedoczynność tarczycy. Włączenie suplementacji L-tyroksyną wywołało nawrót arytmii. Przypadek ukazuje, jak trudne może być leczenie w takiej sytuacji oraz jak ważna jest ścisła współpraca kardiologa i endokrynologa podczas terapii poamiodaronowych zaburzeń czynności tarczycy.

Słowa kluczowe: amiodaron, trzepotanie przedsionków, niedoczynność tarczycy

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