Archives of the Balkan Medical Union
vol. 58, no. 1, pp. 74-77
March 2023
Copyright © 2023 Balkan Medical Union

CASE REPORT

TYPE 2 ACUTE ANTERIOR ST-ELEVATION MYOCARDIAL INFARCTION SECONDARY TO HYPERTHYROIDISM: A CASE REPORT

Nevena G. IVANOVA[1,2]✉

[1] Department of Urology and General Medicine, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria
[2] St Karidad MBAL, Karidad Medical Health Center, Cardiology, Plovdiv, Bulgaria

Received 03rd Febr 2023, Accepted 28th Febr 2023
https://doi.org/10.31688/ABMU.2023.58.1.09

Abstract

Introduction. Hyperthyroidism is a condition associated with excessive production of thyroid hormones, thyroxine and triiodothyronine. The adverse effects on the cardiovascular system are myocardial ischemia, rhythm disturbances, cardiomyopathy, heart failure and sudden death. The presented clinical case describes the development of type 2 acute anterior myocardial infarction with ST- elevation complicated by heart failure in a patient with hyperthyroidism.
Case presentation. A 76-year-old patient with late-diagnosed hyperthyroidism complicated by dilated cardiomyopathy was urgently admitted to the cardiology department because of typical angina and shortness of breath. The electrocardiogram demonstrated an acute anterior ST-elevation myocardial infarction. Echocardiography revealed increased left ventricular volumes, severely depressed systolic function with ejection fraction 15% by Simpson's method, severe mitral regurgitation and wall-motion abnormalities. Percutaneous coronary intervention revealed underlying atherosclerotic disease without acute thrombosis.
Conclusions. The clinical case illustrates the rarer possibility of developing an acute coronary syndrome

Résumé

Infarctus aigu du myocarde antérieur de type 2 avec élévation ST secondaire à l'hyperthyroïdie : rapport du cas

Introduction. L'hyperthyroïdie est une maladie associée à une production excessive d'hormones thyroïdiennes, la thyroxine et la triiodothyronine, qui pénètrent dans la circulation systémique. Les effets indésirables sur le système cardiovasculaire sont l'ischémie myocardique, les troubles du rythme, la cardiomyopathie, l'insuffisance cardiaque et la mort subite. Le cas clinique présenté décrit le développement d'un infarctus du myocarde antérieur aigu de type 2 avec élévation du segment ST compliqué par une insuffisance cardiaque chez un patient souffrant d'hyperthyroïdie.
Présentation du cas. Un patient de 76 ans souffrant d'hyperthyroïdie tardivement diagnostiquée compliquée d'une cardiomyopathie dilatée a été admis en urgence au service de cardiologie en raison de symptômes typiques d'angor et d'essoufflement. L'électrocardiogramme montre un infarctus aigu du myocarde antérieur avec élévation du segment ST.

✉ Address for correspondence: Nevena G. IVANOVA
St Karidad MBAL, Karidad Medical Health Center, Cardiology, Plovdiv, Bulgaria.
Address: 23A Nikola Vapcarov St., Plovdiv 4004, Bulgaria
Email: nevenai@yahoo.com, Phone +35 9889130416

because of an imbalance between oxygen demand and available coronary flow in another underlying disease, as opposed to type 1 myocardial infarction, which is the most common. There is a need for screening of cardiovascular complications of thyroid disorders.

Keywords: thyrotoxicosis, myocardial ischemia, dilated cardiomyopathy, heart failure, atrial fibrillation.

List of abbreviations:
T4 – thyroxine
T3 – triiodothyronine
PCI – percutaneous coronary intervention
LCXm – circumflex artery medial segment
Diag I – first diagonal branch
LADm –left descending artery medial segment
RCAm – right coronary artery medial segment
DES – drug-eluting stent
TIMI – thrombolysis in myocardial infarction flow grade score system

L'échocardiographie a révélé une augmentation des volumes ventriculaires gauches, une fonction systolique sévèrement déprimée, avec fraction d'éjection de 15% par la méthode de Simpson, une régurgitation mitrale sévère et des anomalies du mouvement des parois. L'intervention coronarienne percutanée a révélé une maladie artérioscléreuse sous-jacente sans thrombose aiguë.
Conclusions. Le cas clinique illustre la possibilité plus rare de développer un syndrome coronarien aigu à la suite d'un déséquilibre entre la demande en oxygène et le débit coronaire disponible dans une autre maladie sous-jacente, par opposition à l'infarctus du myocarde de type 1, qui est le plus fréquent. Il existe un besoin de dépistage des complications cardiovasculaires des troubles thyroïdiens.

Mots-clés: thyréotoxicose, ischémie myocardique, cardiomyopathie dilatée, insuffisance cardiaque, fibrillation auriculaire.

Introduction

Hyperthyroidism is a condition associated with excessive production of thyroid hormones, thyroxine (T4) and triiodothyronine (T3). The etiology is diverse and includes autoimmune processes in Graves' disease, autonomous in adenoma and goiter (diffuse or nodular), malignant tumours, inflammatory diseases of the thyroid gland or drug effects. The clinical presentation is determined by the effects of thyroid hormones on the metabolism and various systems – increase in basic metabolism, stimulation of calcium-phosphorus, over activation of the nervous system, increased catecholamine stimulation of the heart. The relationship between hyperthyroidism and heart disease was first described in the middle of the last century[1]. The negative effects on the cardiovascular system are diverse and can lead to myocardial ischemia, rhythm disorders, development of cardiomyopathy, heart failure and sudden death[2,3]. We present a patient with hyperthyroidism who developed a type 2 acute anterior ST-elevation myocardial infarction complicated by heart failure and systolic left ventricular dysfunction.

Case presentation

A 76-year-old woman patient living in a small village in the Rhodope mountains was diagnosed with thyrotoxicosis and was treated in the endocrinology department. Two months before, the patient visited an outpatient cardiologist for palpitations, swelling of the lower limbs and shortness of breath during physical exertion, including at rest during night. During the cardiology consultation, the diagnosis of atrial fibrillation and dilated cardiomyopathy was established, raising the suspicion of thyrotoxic heart disease. The laboratory tests of thyroid hormones confirmed the diagnosis of hyperthyroidism, and the patient was referred for further treatment to the endocrinologist, being hospitalized in the endocrinology department. During the hospital stay, therapy with thyrostatic (thiamazole 5 mg, 2 tablets bid), corticosteroids (prednisolone 5 mg, 4 tablets in the morning and 2 tablets in the afternoon), diuretics (spironolactone 25 mg, 1 tablet in the morning, and torasemide 10 mg, 1 tablet in the morning), non-vitamin K oral anticoagulant (edoxaban 30 mg daily), gastric protector (famotidine 40 mg, 1 tablet daily), antiarrhythmic (cordarone 200 mg, 1 tablet daily) drugs was initiated.

After a few days, intermittent complaints of chest discomfort of great intensity, shortness of breath and severe headache began. The electrocardiogram revealed an acute ST-elevation anterior myocardial infarction, and the patient was urgently admitted to the cardiology department. The physical examination found a woman with a poor general condition, asthenic habitus, and reduced body mass index. Skin and visible mucous membranes were pale, perioral cyanosis and exophthalmos were present. On palpation of the thyroid gland, it was enlarged, with a soft consistency. Auscultation of the lung revealed a weakened vesicular breathing bilaterally, with prolonged expiration, bilateral crackles. The cardiovascular physical examination revealed tachycardia (101 beats per minute), gallop rhythm, a grade 3/6 apical

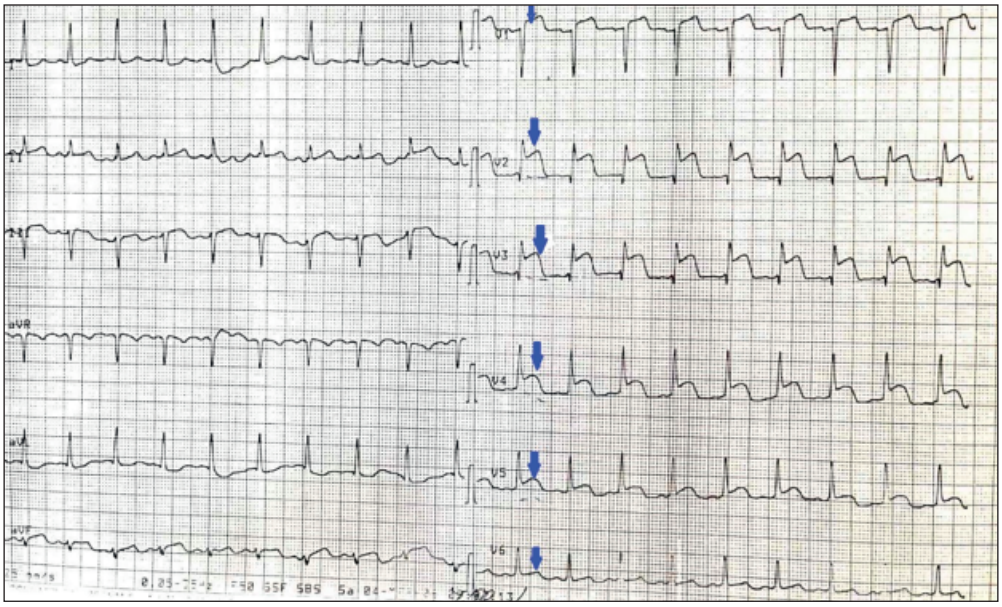


Fig. 1. Electrocardiogam. Acute anterior myocardial infarction – ST elevation in V1-V6

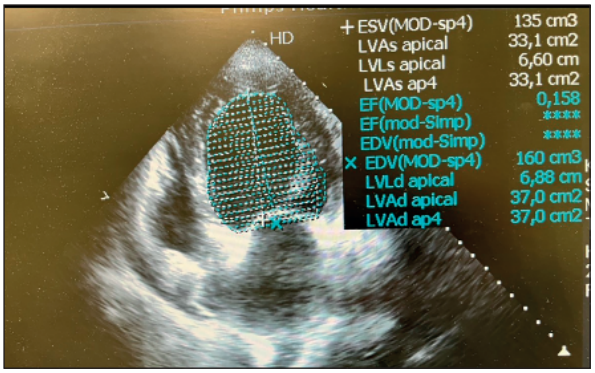


Fig. 2. Transthoracic echocardiography – 4 chambers view. Left ventricle measurements revealed enlarged volumes.

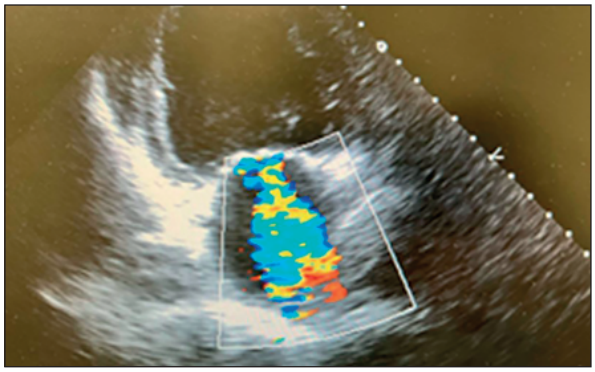


Fig. 3. Color-Doppler transthoracic echocardiography, 2 chambers view. Severe mitral regurgitation

systolic murmur with axillary propagation, and arterial hypotension (blood pressure 90/60 mmHg). The electrocardiogram proved an acute anterior myocardial infarction with ST-elevations in the precordial leads V1-V6 and ST-depression in the standard peripheral leads I and aVL (Fig.1). Echocardiography revealed an increased left ventricular end-systolic volume of 135 cm^3 (normal value in women 65 cm^3) and end-diastolic volume of 160 cm^3 (normal value in women 138 cm^3), severely decreased systolic left ventricular ejection fraction assessed by the Simpson method, 15% (normal > 55%) (Fig. 2) and severe mitral regurgitation IIIrd degree (Fig. 3), left ventricular wall motion abnormalities – apical dyskinesia, mid septal, mid lateral, mid anterior akinesia, hyperkinetic basal segments. The laboratory tests confirmed myocardial damage, with elevated troponin values 0.5 ng/mL (normal value 0.01ng/mL), as well as thyroid overfunction, with free-T3 8.29 pmol/L (normal range 3.20 – 6.80 pmol/L), free-T4 57.28 pmol/L (normal range 12.00 – 22.00 pmol/L). According to the current guidelines for the management of acute coronary syndrome, the therapeutic strategy was invasive. Percutaneous coronary intervention (PCI) was performed through right radial access, finding a critical stenosis of the circumflex artery in the medial segment (LCXm), severe stenosis of the first diagonal branch (Diag. I), borderline stenosis of the left descending artery in the medial segment (LADm) and right coronary artery in medial segment (RCAm). After a balloon dilatation with a 2.0mm balloon, a drug-eluting stent (DES) 2.5/25m was implanted in the LCXm. The procedure continued with balloon dilatation with a 2.0mm balloon in Diag. I with implantation of one DES. The restored distal blood flow in the treated arteries according to the Thrombolysis in Myocardial Infarction flow grade score system (TIMI) was III. TIMI is widely used to assess coronary

blood flow in acute coronary syndromes during percutaneous angioplasty. The TIMI grades range from 0 to 3 (0 no perfusion, 3 complete perfusion). The drug therapy included unfractionated heparin, antiplatelet agents (clopidogrel and acetylsalicylic acid), statin (rosuvastatin), beta-blocker (bisoprolol), metabolic agent (trimetazidine dihydrochloride). Nitrate (molsidomin 2 mg, 2 tablets daily) and diuretic (torasemide 5 mg, 1 tablet in the morning) were added before discharge. The next echocardiographic assessment noted a positive dynamic change in the systolic left ventricular function reaching an ejection fraction of 35% assessed by Simpson method and a reduction of mitral regurgitation to mild (grade I). After hospital discharge, the patient was referred for further treatment in the endocrinology department.

Discussion

The relation between hyperthyroidism and heart disease was first described in the middle of the last century. Data indicate that high levels of free triiodothyronine lead to the occurrence of coronary events 2.6 times more often compared to normal[4]. The underlying atherosclerotic coronary artery disease, embolization, direct vascular endothelial damage, mismatch between oxygen demand and blood supply, and vasospasm arising from an overactivated sympathetic nervous system are involved[2,5]. This overactivation leads to tachycardia, increased stroke volume, and increased myocardial oxygen consumption. Vasospastic angina may occur in up to 20% of patients with hyperthyroidism[6]. Coronary spasm probably results from increased reactivity of arteries to vasospastic agents at the expense of their decreased sensitivity to dilators[2]. Furthermore, coronary spasm in underlying atherosclerotic disease can lead to plaque rupture and subsequent thrombosis. Thyroid hormones also have direct and indirect effects on myocardial contractility, excitability, and peripheral vascular resistance. In patients with autoimmune hyperthyroidism, direct myocardial damage is a result of the synthesis of organ-specific antibodies with cytotoxic effect that, in combination with the altered activity of cellular deiodinase-1, involved in the peripheral conversion of thyroxine into T3, leads to the development of thyrotoxic cardiomyopathy.

The presented clinical case is a demonstration of the development of anterior ST-elevation myocardial infarction, complicated by acute left heart failure in a patient with a delayed diagnosis of underlying cardiomyopathy and untimely treated thyrotoxicosis. An underlying atherosclerotic disease was detected by coronary angiography, without evidence of acute thrombosis with occlusion of a coronary vessel. In view of the angiographic finding, it was assumed to be a type 2 acute myocardial infarction, based on the imbalance between oxygen demand and available coronary flow, with underlying atherosclerotic disease and vasospasm.

Conclusions

This case highlights the rarer possibility (compared to the more common type 1 myocardial infarction) of developing an acute anterior ST-elevation myocardial infarction in the context of late-diagnosed hyperthyroidism. The insufficient number of healthcare professionals in some rural areas of Bulgaria may lead to delayed diagnosis and treatment.

Author contributions:

The author was responsible for cardiology consultation, concept, design and manuscript writing.

Compliance with Ethics Requirements:

"The author declares no conflict of interest regarding this article"

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from the patient included in the study"

"No funding for this study"

Acknowledgements:

None

References

1. Jeffers WA, Littman DS, Rose E. The infrequency of myocardial infarction in patients with thyrotoxicosis. *Am J Med Sci.* 1957;233(1):10-5.
2. Kuang XH, Zhang SY. Hyperthyroidism-associated coronary spasm: A case of non-ST segment elevation myocardial infarction with thyrotoxicosis. *J Geriatr Cardiol.* 2011;8(4):258–259.
3. Common K, Milburn KS, Cawood TS, et al. Coronary artery spasm due to thyrotoxicosis. *N Z Med J.* 2012;126(1368):75-8.
4. Zhou D, Qu Z, Wang H, et al. Severe hyperthyroidism presenting with acute st segment elevation myocardial infarction. *Case Rep Cardiol.* 2015;2015:1-4.
5. Canpolat U, Sunman H, Gurses KM, et al. Vasospastic angina in a patient with hyperthyroidism. *Herz.* 2012, 37(5):570-2.
6. Dedov II, Kalashnikov Vlu, Terekhin SA, et al. Fatal coronary artery spasm in a patient with thyrotoxicosis. *Kardiologiia.* 2012;52(11):92-6.