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Case Report

Myocardial infarction as a first clinical manifestation of hyperthyroidism

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ABSTRACT

Introduction: Hyperthyroidism is a condition in which there is an increased secretion of thyroid hormones that exceeds the current needs of the body. Increased concentrations of hormones affect proper functioning of many organs and systems, including the cardiovascular system. Untreated hyperthyroidism may result in myocardial infarction (MI), which very rarely is its first clinical manifestation.

Aim: The aim of this work is to present the case of a male patient with no previous symptoms, in whom ST segment elevation myocardial infarction (STEMI) occurred as the first clinical manifestation of hyperthyroidism.

Case study: Presentation and analysis of the case of a 41-year-old male admitted with clinical and electrocardiographic features of anterior wall STEMI in the course of hyperthyroidism.

Results and discussion: The cause of ischemia and MI in patients with no previous coronary artery disease is not fully known. Probable predisposing factors may include increased serum thyroid hormone concentrations. It enhances the vasoconstrictor effect of catecholamines and hyperkinetic circulation, which may be the potential causes of ischemia. In addition, it predisposes to a hypercoagulable state, which is the cause of coronary thrombosis.

Conclusions: It is not only high cholesterol levels and anemia that are the possible causes of MI. Endocrine disorders, such as hyperthyroidism should always be taken into account.

1. Introduction

Thyroid hormones have a significant effect on the maintenance of body homeostasis and proper functioning of many systems, including the cardiovascular system. Thus, in the majority of patients hyperthyroidism presents with a variety of cardiovascular symptoms. Increased levels of thyroid hormones may be the direct or indirect cause of arrhythmias, symptoms of heart failure and ischemic heart disease.[1–4]

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2. Aim

The aim of this work was to present a rare case of ST segment elevation myocardial infarction (STEMI) in the course of clinically silent hyperthyroidism in a male patient with no previous cardiovascular or endocrine diseases.

3. Case study

This article was written based on the history and physical examination, laboratory and imaging study results of a patient admitted to the Cardiac Intensive Care Unit. Patient, a 41-year-old male, with no history of cardiovascular signs and symptoms of thyroid disease, was transported to the hospital by Medical Air Rescue with clinical and electrocardiographic features of anterior wall STEMI. During transport the patient experienced five events of sudden cardiac arrest in ventricular fibrillation mechanism, which were effectively treated by defibrillation.

In the week preceding hospital admission patient experienced chest pains three times which lasted for approximately 5 min. On admission, the patient presented with chest pain stronger than previously and did not report any complaints from other organs or systems. He smoked about 30 cigarettes per day for 20 years. He consumed alcohol occasionally. Family history was negative for genetic, cardiovascular and social diseases.

On admission, physical examination showed a regular heart rate of 100 bmp, normal heart tones, with no pathological murmurs. No abnormal findings from other organs and systems were found, body composition was normal. Body temperature was 38.4°C. Blood pressure: 110/80 mmHg.

Laboratory results abnormalities included: increased levels of high-sensitivity troponin T (0.988 ng/mL, norm <0.010 ng/mL), increased levels of creatinine kinase (187 U/L, normal range 0–24 U/L), leukocytosis (27.31 × 10^3/μL, range from −4.1 to 10.9 10^3/μL), granulocytosis (24.13 × 10^3/μL, normal range 1.5–7.0 × 10^3/μL), as well as elevated D-dimer levels (61.8 μg/mL, norm <0.5 μg/mL), aspartate aminotransferase (235 U/L, normal range 10–50 U/L), C-reactive protein (1.71 mg/dL, norm <0.5 mg/dL), prolonged activated partial thromboplastin time (62.2 s, normal range 24–37 s) and significantly decreased concentrations of thyreotropic hormone (<0.005 μIU/mL, normal range 0.27–4.20 μIU/mL).

The admission electrocardiogram (ECG) showed intermediate axis (Fig. 1). Sinus rhythm, regular rate of 100 bpm, with

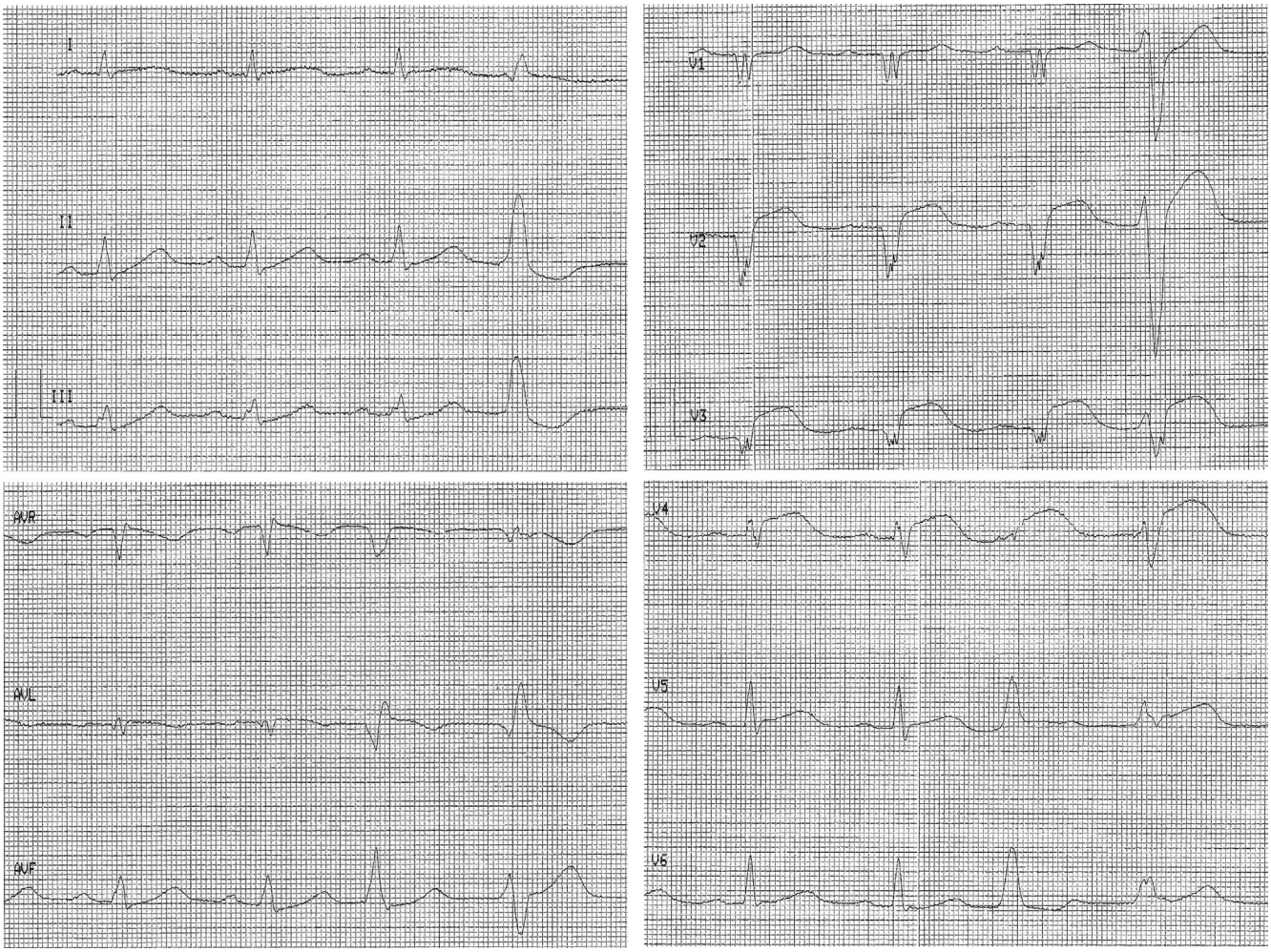

Fig. 1 – ECG on admission.

single ventricular beats. ST segment elevation in leads V2–V5, downsloping ST depression in leads II, III, aVF.

After diagnostic procedures the patient was immediately transferred to cardiac catherization laboratory for coronary angiography and potential simultaneous percutaneous coronary intervention. The examination showed proximally amputated anterior descending coronary artery with no atherosclerotic component. The remaining arteries had no significant stenosis. Balloon pre-dilation to restore patency of proximally amputated anterior descending coronary artery was performed. Then, sirolimus-eluting stent was implanted in this branch with complete perfusion and post-procedural TIMI 3 flow.

Due to the significantly decreased level of serum thyreotropic hormone, an endocrinologist was consulted and further laboratory examinations were performed. Abnormalities found: increased free thyroid hormones concentrations, triiodothyronine (16.0 pmol/L, normal range 3.1–6.8 pmol/L) and thyroxine (62.4 pmol/L, normal range 12–22 pmol/L) as well as thyroid peroxidase antibodies (322 IU/mL, norm <34 IU/mL) and TSH receptor antibodies (8.04 IU/L, normal range 0.0–1.75 IU/L).

Upon recommendation of endocrinologist thyroid ultrasound was performed, which evidenced a two-lobe thyroid gland in a normal position, enlarged (right lobe – 6.0 × 2.4 × 2.4 cm; left lobe 6.0 × 2.0 × 2.0 cm; isthmus 5 mm, volume 28 mL), reduced echogenicity of the glandular tissue, heterogeneous; the surrounding lymph nodes were normal size. Ultrasound images confirmed Graves' disease.

During hospital stay typical pharmacological treatment of MI and hyperthyroidism was implemented, in accordance with guidelines.

Echocardiogram, performed at the fourth day of hospitalization, showed akinesia of the apex and anterior septum, significant hypokinesia of posterior septum, dilated left atrium. Left ventricular wall thickness was normal. There were no significant changes in valve morphology. Minor mitral and tricuspid regurgitation. No fluid in the pericardium. Ejection fraction 42%–45%.

A Holter study showed underlying sinus rhythm, 91 bpm, with single ventricular beats and 4 episodes of non-sustained ventricular tachycardia.

The patient was discharged home after a two-week hospital stay in a good general condition with recommended pharmacotherapy typical for post-MI state and hyperthyroidism, with follow-up in cardiology and endocrinology clinic.

4. Results and discussion

Numerous factors influence the incidence of cardiovascular diseases. One of the pathomechanisms is vasoconstriction of a frequently unknown origin.[5] An important predisposing factor is hyperthyroidism.

Hyperthyroidism may be the cause of cardiovascular symptoms by inducing the incidence of arrhythmias, symptoms of heart failure, cardiomyopathy and coronary heart disease.[2,4,6]

Angina occurs in approximately 20% of patients with hyperthyroidism. Usually, it is caused by coronary artery atherosclerosis and increased sympathetic activity. Origin of ischemia and MI in patients with no coronary pathology is not fully understood. Previously undetected coronary heart disease, direct effect of thyroid hormones on the heart muscle and secondary supraventricular tachycardia or atrial fibrillation are listed as a determinant of these conditions.[4]

Hyperthyroidism significantly increases metabolism by increasing myocardial oxygen demand.[1] Hypoxia in cardiomyocytes in patients with no coronary artery atherosclerosis is caused by difficulties in the delivery, transport and utilization of oxygen.[7,8]

Moreover, thyroid hormones sensitize adrenergic receptors which enhances the vasoconstrictor effect of catecholamines on coronary arteries and hyperkinetic circulation. Prolonged adrenergic stimulation impairs myocardial contractility and contributes to cardiac arrhythmias. Paroxysmal atrial fibrillation is the frequent complication and subclinical hyperthyroidism is a significant risk factor of its incidence.[2,4]

Thyroxine and triiodothyronine also induce cell proliferation changes in the vascular wall which may lead to myocardial ischemia,[9] and predispose to a hypercoagulable state, which is the cause of coronary thrombosis.[10]

Another causal factor in vasoconstriction is cigarette smoking. Active smokers account for 75% of patients with symptoms of angina. They are exposed to many toxic substances, such as nicotine, carbon monoxide and pro-inflammatory substances. They damage endothelium, impair vascular smooth muscle relaxation and in consequence contribute to vasoconstriction.[5] Smoking is believed to be one of the major risk factors of MI, sudden cardiac death and angina, thus, it seems highly probable that through coronary vasospasm it may cause myocardial cell hypoxia.[11,12]

5. Conclusions

The presented patient, with no previous symptoms, normal body composition, was not treated for arterial hypertension, diabetes mellitus or hypercholesterolemia. He had no history of cardiovascular symptoms, such as palpitations, which is one of the most common signs of hyperthyroidism that occurs in 85% of patients, or exertional dyspnea and decreased exercise tolerance, which occur in 50% of patients.[10,13]

Laboratory findings did not confirm any other potential cause of MI, such as high cholesterol levels indicating coronary atherosclerosis or anemia which may be the cause of hyperkinetic circulation.[14]

Prolonged smoking may have been the concomitant factor in the cause of myocardial infarction. However, considering very high serum concentrations of thyroid hormones, it seems more likely that hyperthyroidism was the primary cause of MI.[5,11,12]

To sum up, hyperthyroidism may be the cause of MI, particularly in patients with few risk factors for coronary artery atherosclerosis.[15]

Conflict of interest

None declared.

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