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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.

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#### 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.

### 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  $38.4^{\circ}$ 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  $\times$   $10^3/\mu L$ , range from -4.1 to  $10.9~10^3/\mu L$ ), granulocytosis (24.13  $\times$   $10^3/\mu L$ , normal range 1.5–7.0  $\times$   $10^3/\mu L$ ), as well as elevated D-dimer levels (61.8  $\mu g/mL$ , norm  $<\!0.5~\mu g/mL$ ), 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 ( $<\!0.005~\mu IU/mL$ , normal range 0.27–4.20  $\mu IU/mL$ ).

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

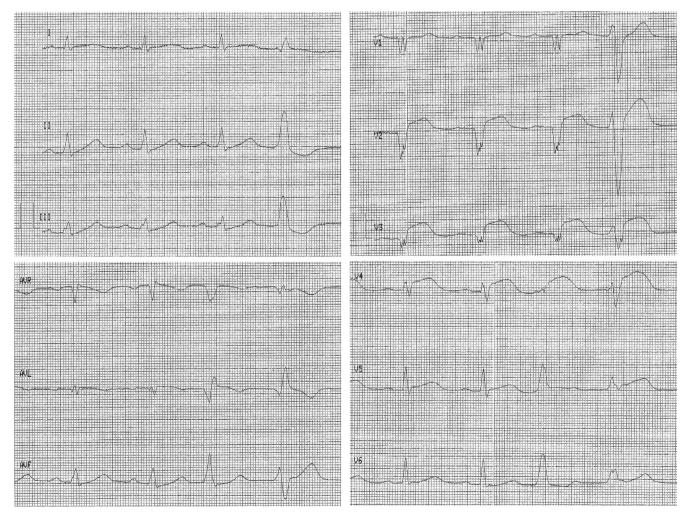


Fig. 1 - ECG on admission.

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