# Acute coronary syndrome, the first manifestation of hyperthyroidism?

*By* Eleonora Dragan

WORD COUNT 952
TIME SUBMITTED 18-JUN-2024 02:06PM
PAPER ID 109705850

AMALTEA MEDICAL PUBLISHING HOUSE

https://medscience.center/journals/

Word count: 949

**TYPE OF ARTICLE:** Original Article

# Acute coronary syndrome, the first manifestation of hyperthyroidism?

Eleonora Dragan [1,2], Irina Cuciureanu [1,2], Catalina Liliana Andrei [1,2]

[1]Cardiology, Bagdasar-Arseni Emergency Hospital, Bucharest, Romania

[2]Bagdasar-Arseni Emergency Clinical Hospital, Bucharest, Romania

*Corresponding author:*

Eleonora Dragan

E-mail: norisor13@yahoo.com

## ABSTRACT

Thyroid hormones exert important effects on the cardiovascular system, through indirect and direct mechanisms. The involvement of the cardiovascular system in hyperthyroidism has been recognized for a long time. In some patients, signs and symptoms of heart failure and atrial fibrillation may dominate the clinical picture. In other patients, acute coronary syndrome is a rare but severe and potentially life-threatening manifestation of hyperthyroidism. The relationship between coronary artery, thyroid function and angina is complex and poorly understood. Because of the rarity of this association, its pathogenicity and its therapeutic interest, we present a case so that clinicians will keep in mind in the diagnosis of similar cases.

**Keywords:** acute coronary syndrome, hyperthyroidism, coronary angiography

**Abbreviations:**

ACS – acute coronary syndrome

VT – ventricular tachycardia

PVC – premature ventricular contraction

LVEF – left ventricular ejection fraction

LAD – left anterior descending artery

RCA – right coronary artery

NV – normal value

TSH – thyroid stimulating hormone

## INTRODUCTION

The cardiovascular disease, including acute myocardial infarction, remains a leading cause of morbidity and mortality all over the world [1]. Acute coronary syndrome (ACS) encompasses unstable angina, acute myocardial infarction with or without elevation of ST segment - a spectrum of cardiovascular emergencies arising from the obstruction of coronary artery blood flow. Coronary atherosclerosis is one of the most important pathogenic factors leading to acute coronary syndrome [2]. It is well known that the cardiovascular system is sensitive to the action of thyroid hormone, and that abnormal metabolism of this hormone influences the onset and progression of coronary disease. In vivo and in vitro studies have shown that TSH, independent of thyroid hormones, increases liver cholesterol via hepatic 3-hydroxy-3-methyl-glutaryl coenzyme A reductase [3], or it predisposes to a hypercoagulable state, which is the cause of coronary thrombosis [4].

## MATERIALS AND METHODS

We present the case of a 76-year-old female patient, non-smoker, hospitalized for chest pain on exertion. She arrived at the hospital with sustained TV episode with hemodynamic deterioration requiring cardioversion. Clinical: regular heart sounds, 3/6 aortic holosystolic murmur, 2/6 mitral systolic murmur, without systemic or pulmonar congestion. ECG showed sinus rhythm with PVC (Figure 1) with diffuse negative T with pseudonormalization in pain. Biology was negative for myocardial infarction. Chest X-ray showed dilated aorta with calcifications, normal cardiac silhouette. Chest CT: excludes aortic syndrome, reveals plunging goiter. Hyperthyroidism was detected (TSH=0.113$\mu$UI/ml; NV=0.35-4.95$\mu$UI/ml). Echocardiographic: dilated left atrium,

degenerative mitral regurgitation, aortic sclerosis, normal LVEF. Coronary angiography revealed bivascular lesion (LAD infiltrated with stenosis 60% segment II, RCA occlusion segment I) (Figure 2).

## RESULTS

The peculiarity of the case is the presence of hyperthyroidism - not obvious, as it usually happens. Another peculiarity is the discrepancy between the minimal ECG changes and the angiographic appearance of the coronary lesions. The patient was referred to cardiovascular surgery and to an endocrinologist for antithyroid therapy.

## DISCUSSION

In the light of the research carried out to date, there is accumulating data associating thyroid dysfunction with atherosclerotic cardiovascular disease, due to its effect on blood pressure and its role in the regulation of lipid metabolism and coronary atherosclerosis [5]. The metabolic process is generally accelerated by thyroid hormone and therefore each, or in combination with each other, may be an important independent risk factor for atherosclerotic heart disease [6, 7]. In our patient, the main manifestation was chest pain and she was diagnosed with hyperthyroidism because she had a goiter. The electrocardiogram was not diagnostic but coronary angiography revealed atherosclerotic bivascular lesions. It is not only high cholesterol levels and anemia that are the possible causes of ACS; endocrine disorders, such as hyperthyroidism should always must be considered.

In order to increase the current knowledge on the contribution of dysthyroidism to the onset and aggravation of cardiovascular disease, respectively, to accurately confirm the effects of the thyroid-heart inter-relationship, both in the short and long term, specifically designed clinical trials are needed.

## CONCLUSION

Hyperthyroidism should be considered a precipitating or causative factor of acute coronary syndrome, especially in patients without risk factors for atherosclerotic disease. More studies are needed to answer the question of whether restoring euthyroid status influences morbidity and mortality.

**Conflict of interest:**

I sign and guarantee that I have no financial or personal obligations that could affect the content of this work.

**Author's contributions:**

All authors have read and agreed to the published version of the manuscript.

**REFERENCES**

1. OMS(https://www.who.int/home/search?indexCatalogue=genericsearchindex1&searchQuery=cardiovascular%20disease%20mortality&wordsMode=AllWords&healthtopic=undefined&country=undefined)
2. Boudoulas KD, Triposciadis F, Geleris P, Boudoulas H. Coronary Atherosclerosis: Pathophysiologic Basis for Diagnosis and Management. Prog Cardiovasc Dis. 2016;58:676–692
3. Tian L, Song Y, Zing M et al: A novel role for thyroid-stimulating hormone: up-regulation of hepatic 3-hydroxi-e-methyl-glutaryl-coenzime A reductase expression through the cyclic adenosine monophosphate/protein kinase A/cyclic adenosine monophosphate-responsive element binding protein pathway. Hepatology 2010, 52:1401-1409
4. Forfar JC, Caldwell GC. Hyperthyroid heart disease. Clin Endocrinol and Metab 1985; 14: 491-508
5. Ochs N, Auer R, bauer DC, Nanchen D, Gussekloo J, Cornuz J, Rodondi N: Meta-analalysis: subclinical thyroid dysfunction and risk for coronary heart disease and mortality. Am Intern med 2008, 148; 832-845
6. Bai MF, Gao CY, Yang CK, Wang XP, Liu J, Qi DT, Zhang Y, Hao PY, Li MW. Effects of thyroid dysfunction on the severity of coronary artery lesions and its prognosis. J Cardiol. 2014;64:496–500
7. Somerville W, Levine SA. Angina pectoris and thyrotoxicosis. Br Heart J 1950; 12: 245-7

**FIGURES**

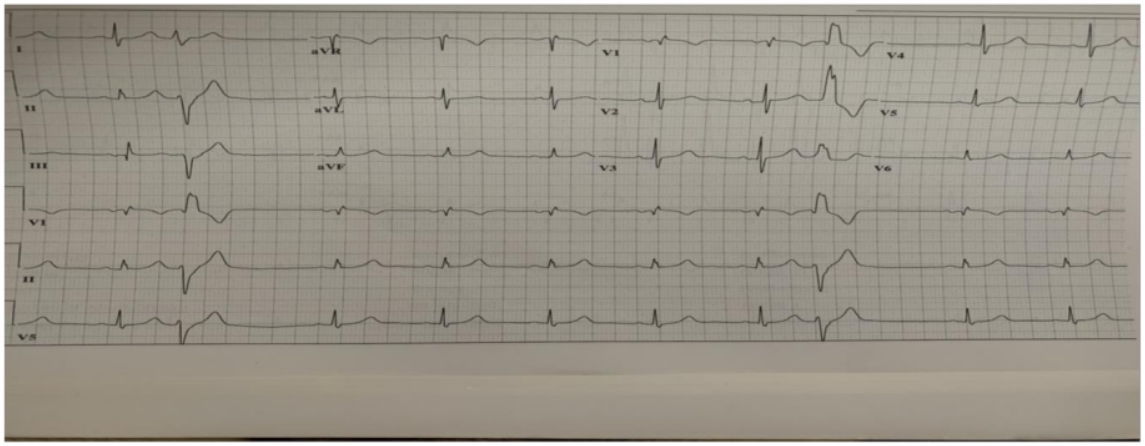


**Figure 1.** ECG – sinus rhythm, 60bpm, with PVC, AQRS=60˚, QTc=420msec, no ST-T changes

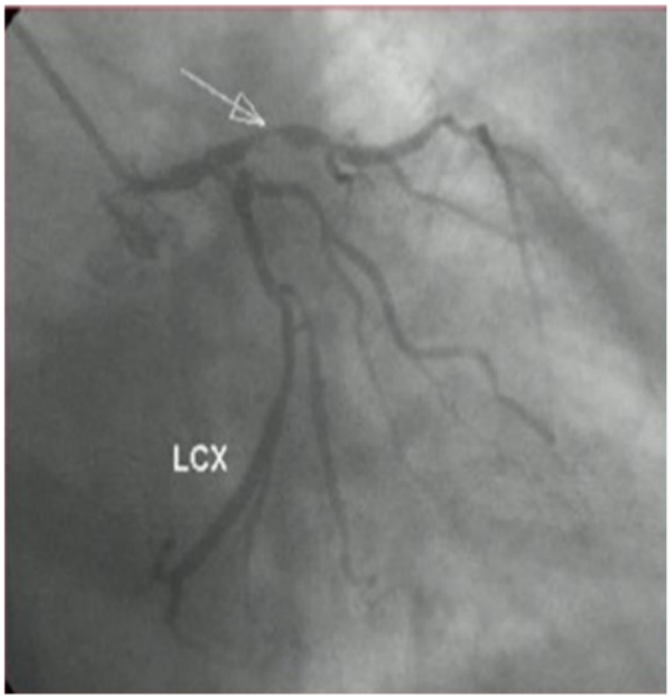


**Figure 2.** Coronary angiography - bivascular lesion (LAD infiltrated with stenosis 60% segment II, RCA occlusion segment I)