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CASE REPORT

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ACUTE MYOCARDIAL INFARCTION SECONDARY TO GRAVE'S DISEASE IN A 35-YEAR-OLD AFRICAN MALE : A CASE REPORT

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A R T I C L E I N F O

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A B S T R A C T

Background: Grave's disease is characterized by the triad of hyperthyroidism, ophtalmopathy, and dermopathy, wich may occur singly or in combination. Hyperthyroidism and thyrotoxicosis were reported to be associated with myocardial infarction. To the best of our knowledge, few cases of myocardial infarction secondary to hyperthyroidism was reported in sub-saharan.

Case presentation: We present a case of myocardial infarction (MI) secondary to Grave's disease in a 35-year old african male with no risk factors for atherosclerosis who complained of epigastric pain. The diagnosis of MI was confirmed by the ectrocardiogram (ECG) and myocardial enzymology. He was treated with anti-thyrotoxicosis and anti-anginal medication.

Conclusion: We recommend to add thyroid function test in young patients with MI, particulary in the absence of classical risk factors.

INTRODUCTION

Background

Grave's disase is the most common cause of hyperthyroidism, accounting for 60-80% of cases. The prevalence is around 1% in women aged 35-60 years, with a 5-10 lower frequency in men [1]. Hyperthyroidism is accompanied by important changes in hemodynamics and cardiac functions. These include clinical manifestation such as an increased cardiac contractility and heart rate as well as decreased peripheral vascular resistance [2].Various causes of nonatherosclerotic myocardial infarction were reported in the medical litterature including few cases of hyperthyroidism presumably secondary to thyroid-induced coronary artery spasm [3,4,5,6,7]. In Africa, neither hyperthyroidism nor myocardial infarction can be considered a rare disorder, yet there have been a few cases reported of the two conditions occuring simultaneously. We herein report a case of myocardial infarction secondary to Grave's disease in a 35-year old african male.

Case presentation

A 35-year-old male was admitted in the emergency department (ED) with a 14 hours history of sudden onset of localised epigastric pain and vomiting.

Physical examination showed a weight loss. His temperature was 37,1°C, blood pressure of 105/60mmHg, heart rate was about 80 beats per minute. The lungs were clear to auscultation. The heart sounds were normal and there was no abdominal tenderness. The thyroid gland was enlarged. There was no exophtalmos, but he had a slight tremor, damp palm and complained of a gritty sensation in the eyes with excessive lacrimation. The admission electrocardigram (ECG) showed, 3 to 4mm ST segment elevations in II, III and aVF. (Fig1)

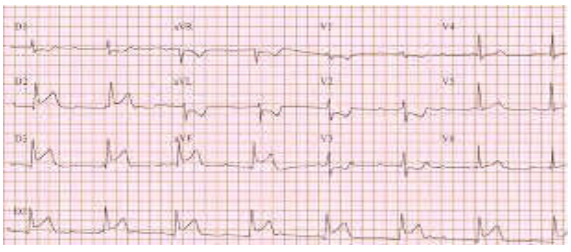

Fig1 Admission ECG at the ED showing ST segment elevations in leads II,III and aVF).

Table1 Results of thyroid function tests

	Values	Reference range
Free T3	38.71 pmol/L	2.8–7.1
Free T4	89 pmol/L	12–22
TSH	<0.005 μIU/mL	0.27–4.2

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Laboratory workup revealed an increased level of Troponin I (7.15μg/L) in addition to creatine kinase and MB levels. Inflammatory markers such as C-reactive proteine (CRP) and erythrocyte sedimentation rate (ESR), glucose, liver and renal function were in normal ranges. The lipid parameters at admission were within normal levels. No classical risk factors of acute coronary syndrome were reported but his medical history revealed that he was diagnosed of hyperthyroidism 6 months ago and stopped medication except propanolol (a non selective beta-blocker) by himself 2 months ago. Hormone analyses (Table1) revealed increased levels of free T3 and T4 with a decreased level of Thyroid Stimulating Hormone (TSH).

The patient was treated with aspirin, low molecular weight heparin (LMWH), atorvastatin, bisoprolol, clopidogrel and propylthiouracile (PTU). The transthoracic echocardiography performed 5 days after admission was normal with left ventricle ejection fraction of 69%.

A thyroid ultrasound scan showed a hypervascularsed mild enlargement gland reffered as "thyroid inferno" (Fig2).Patient's financial status causes coronary angiogram was not arranged.

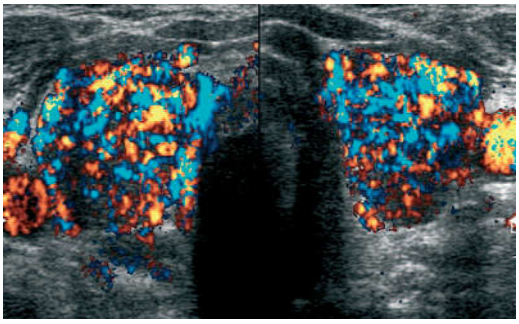

Fig 2 Echography showing hypervascularisation of the thyroid gland

Clinical course was good, the patient was successfully discharged after 10 days of treatment. His ECG before discharge showed no obvious abnormality (Fig 3).

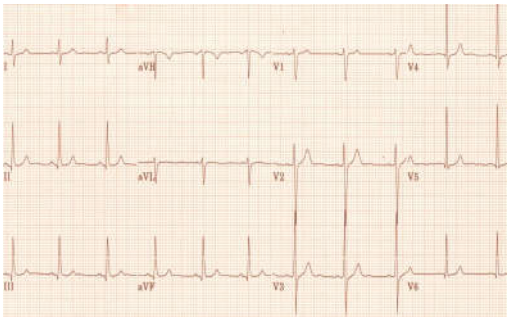

Fig 3 Normal ECG before discharge).

A follow-up ECG showed no pathological Q waves, and patient denied any anginal symptoms. The patient remained euthyroid on PTU, which was gradually discontinued.

DISCUSSION

Grave's disase is the most common cause of hyperthyroidism, accounting for 60-80% of cases. It is characterized by the triad of hyperthyroidism, ophtalmopathy, and dermopathy, wich may occur singly or in combination. Although Grave's disease occurs most often in young females, it canoccur at any age and in either sex. Thyroid hormones affect cardiovascular system by increasing stroke volume and heart rate [8]. Excessive thyroid hormones are linked to many heart diseases such as angina, heart failure, arrhythmia and sudden death [9]. Thyrotoxicosis was reported to be associated with angina in 0,5% to 20% of the patients[10,11] . Although rare, both hyperthyroidism and thyrotoxicosis were reported to be associated with myocardial infarction with normal coronary arteries [4,5,12]. In sub-sahara, despite many studies on the occurence of hyperthyroisim, few cases of myocardial infarction in hyperthyroid patient was reported.

A variety of explanations for the occurence of angina in hyperthyroidism was reported including the metabolic effect of the hormone on the myocardium, tachyarythmias induced by disease, in situ coronary thrombosis and vasospasm [4,11]. Coronary vasospasm was reported to be the most plausible explanation [6,7,12]. According to the typical ST segment elevation in the inferior leads on the ECG, the epigastric pain, and the elevated myocardial enzymology, there was no doubt about the diagnosis of acute myocardial infarction in our patient. Although, coronarography was not performed in this patient because of his financial status, we believe that vasospasm was probably the main mechanism. The absence of classical cardiovascular disease risk factors, and normalisation of the ECG within 10 days, reinforce this idea.

CONCLUSION

Hyperthyroisim should be considered as a possible cause of myocardial infarction, particularly in patients without classical risk factors for atherosclerotic disease. Since the diagnosis of hyperthyroidim may be overlooked or delayed in patients without typical symptoms, thyroid function test should be mandatory in such patients.

List of abbreviations

MI: myocardial infarction.
ECG: ectrocardiogram .
ED: emergency department .
CRP: C-reactive protein.
ESR: erythrocyte sedimentation rate.
PTU: propylthiouracile.
LMWH: low molecular weight heparin.

Declarations

***Consent for publication*:** a signed consent to publish was obtained from the patient
***Competing of interest*:** the authors declare that they have no competing interests

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