

Hyperthyroidism presenting as ST Elevation Myocardial Infarction with Normal Coronaries – A Case Report

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Abstract

A 31-year-old male, apparently well, presented with typical chest pain. His ECG showed ST-elevation from V1-V4 and echocardiogram revealed anteroseptal wall hypokinesia with ejection fraction of 45%. Normal coronary arteries were seen on coronary angiogram. A thyroid function test showed elevated free T4 levels with suppressed thyroid stimulating hormone (TSH). Treatment with thionamides and beta-blockers improved symptoms. Upon review 4 months later he was well. Repeat echocardiogram showed good ejection fraction with no hypokinetic area.

Key words: thyrotoxicosis, Acute Myocardial Infarction (AMI), angina, Graves' disease, Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA)

INTRODUCTION

An acute myocardial infarction (AMI) can be triggered by multiple factors like increased oxygen demand, hyperlipidemia, hypercoagulable states, coronary vasospasm and cocaine abuse.¹ Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is an increasingly recognised entity.² It is well known that hyperthyroidism is associated with heart disease, and rarely, can cause AMI.³ The mechanism for this is not fully understood, although there have been many postulations.¹ We present an interesting case of a young man with newly diagnosed thyrotoxicosis secondary to Graves' disease presenting with AMI and normal coronaries.

CASE

A 31-year-old male with no prior medical illness presented to the Emergency Department with sudden onset of severe left-sided chest pain at rest, associated with palpitations, diaphoresis and dyspnea. Over the preceding month, he had palpitations, heat intolerance and lost 3 kg of weight. He denied use of illicit drugs. He is a smoker and works as a rubber tapper. Family history was non-contributory. On presentation, blood pressure was 133/86 mmHg, with heart rate of 137 beats per minute, regular with good volume. Cardiovascular and respiratory system examination were otherwise unremarkable. He had exophthalmos and a diffuse goitre measuring 9x6 cm, with no bruit. Electrocardiogram (ECG) showed sinus tachycardia with ST elevation in leads V1 to V4 (Figure 1). His complete blood count and renal profile were normal. Creatinine kinase-MB (CK-MB) done within 6 hours of presentation was normal; however, a Troponin level was not measured. Echocardiogram showed hypokinetic anteroseptal wall with ejection fraction 45%, normal

valves, chamber sizes and ventricular dimensions with no evidence of left ventricular hypertrophy. He was treated for ST elevation myocardial infarction and thrombolysed with intravenous (IV) Streptokinase. He was subsequently started on aspirin, clopidogrel, fondaparinux, metoprolol and atorvastatin. Despite resolution of pain, there was minimal reduction of ST elevation with poor R wave progression from V2-V5. Coronary angiography done 3 days later showed completely normal coronary vessels with no stenosis or spasm. Thyroid function test (TFT) revealed TSH- <0.005 mIU/L (0.4-4.0) and FT4- 66 pmol/L (7.86-14.4 pmol/L). Neck ultrasound showed a diffusely enlarged thyroid gland with increased vascularity. Burch-Wartofsky score on presentation was 30. A clinical diagnosis of Graves' disease with thyrotoxicosis was made. TSH receptor antibodies were not done as it was not supported by the local lab. He was started on carbimazole 30 mg daily, gradually tapered over 4 months, and propranolol 40 mg twice daily while all other medications were discontinued. Upon review 4 months later, he was well with good effort tolerance. He was clinically and biochemically euthyroid. A repeat electrocardiogram (ECG) showed persistent T-wave inversions in leads V2 to V5 with poor R-wave progression (Figure 2). Repeat echocardiogram showed normal chambers with ejection fraction 53% and no hypokinetic area.

DISCUSSION

Myocardial infarction induced by thyrotoxicosis is rare with an incidence of 1.8% but is showing an upward trend⁴ A subset of thyrotoxic patients can experience angina-like chest pain. Thyrotoxic angina is described as the following: (i) the presence of angina at rest, (ii) rapidly progressive angina, (iii) cessation of angina with treatment of hyperthyroidism and (iv) the lack of typical clinical

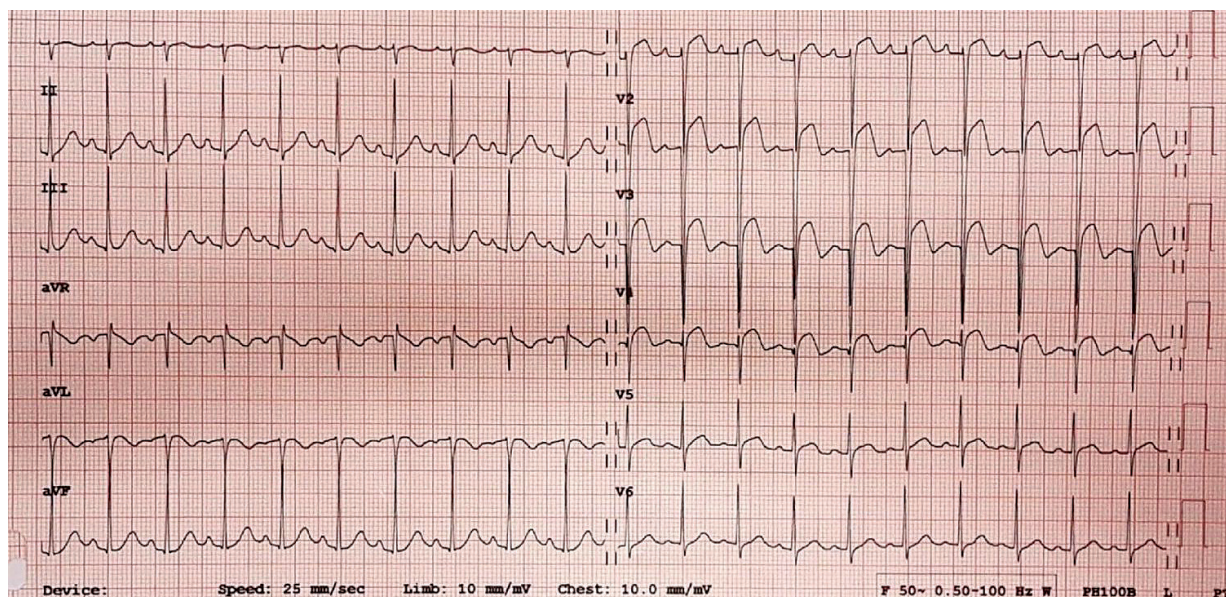


Figure 1. ECG upon arrival (March 2018).



Figure 2. ECG upon review 4 months later (July 2018).

manifestations of hyperthyroidism upon presentation.⁵ Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is being increasingly recognised and has multiple potential underlying mechanisms.² The diagnosis of MINOCA requires: (i) clinical documentation of a myocardial infarct, (ii) exclusion of obstructive coronary arteries and (iii) no overt cause for the AMI presentation, such as cardiac trauma.²

The exact cause of acute myocardial infarction (AMI) in thyrotoxic patients with normal coronary arteries is unclear. There are several proposed mechanisms such as temporary major coronary artery occlusion, small vessel disease and increased myocardial oxygen demand.¹ There is evidence that thyrotoxicosis is directly associated with the presence of a prothrombotic state.⁶ Higher levels of prothrombotic factors and lower levels of anticoagulative factors have been demonstrated among patients with a history of thyroid cancer receiving TSH-suppressive L-thyroxine therapy in comparison the same subjects in hypothyroid phase prior to radioiodine whole-body scanning procedure.⁶ Homoncik et al., reported raised concentrations of von-Willibrand factor (vWF) and increased baseline platelet plug formation in patients with thyrotoxicosis which were corrected by treatment of thyrotoxicosis with thionamides.⁷ Vasospastic angina secondary to transient coronary vasospasm occurs in up to 20% of hyperthyroid patients, yet is difficult to confirm.⁸ Diagnosis is suggested by finding a reversible

coronary artery stenosis on coronary angiography. However, the use of coronary angiography as a first diagnostic test in confirming this is not supported as iodine containing contrast agents used have the potential to induce thyrotoxicosis.^{3,5} Possible mechanisms of thyrotoxicosis-induced vasospasm include enhanced coronary sensitivity to vasoconstrictors and reduced sensitivity to vasodilators.³ Coronary vasospasm can also promote atherosclerosis by accelerating the formation of a thrombus and delaying fibrinolysis.³ In a subgroup of females under 50 years old with documented coronary artery spasm, the incidence of hyperthyroidism was 29% and these subjects, like in our reported case, presented with severe myocardial ischemia.⁹ Angina associated with coronary artery spasm among hyperthyroid patients resolve upon being rendered euthyroid.⁹

The documented aetiology of MINOCA include coronary artery spasm, microvascular dysfunction and thrombophilic states.² Although thyrotoxicosis has not been documented as a cause for MINOCA, the postulated mechanisms leading to thyrotoxicosis associated AMI is similar to some causes for MINOCA.

Our patient's coronary angiography did not demonstrate coronary vasospasm (Figure 3), possibly because it was done relatively late, after administration of nitrates and resolution of symptoms. Another postulate is that he had a clot occluding his coronary vessel which

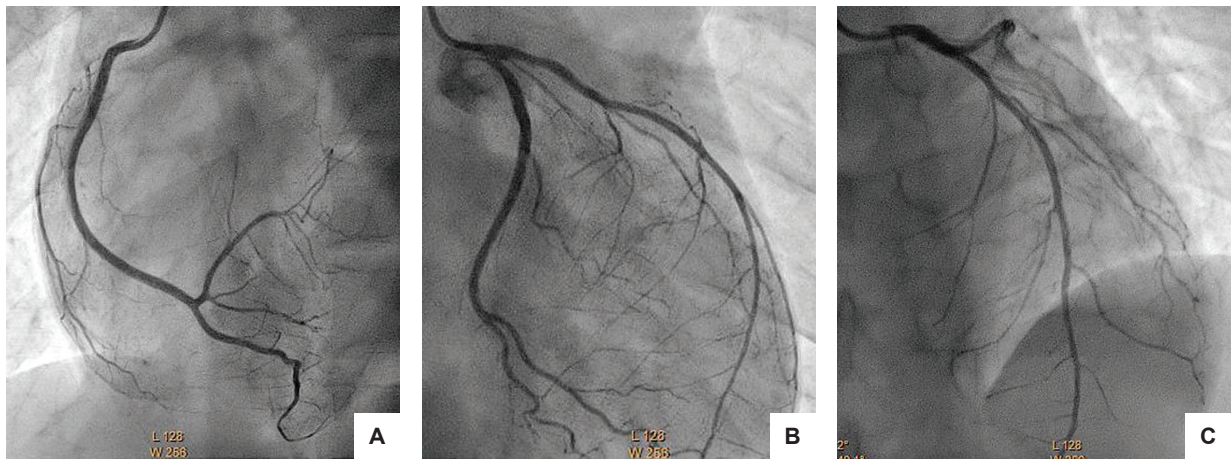


Figure 3. Coronary angiogram (March 2018) showing: (A) Left- Normal right coronary artery (RCA); (B) middle- Normal left circumflex artery (LCx); (C) Right- Normal left anterior descending artery (LAD).

was successfully thrombolysed with IV Streptokinase. Limitations of this case report includes unavailability of a Troponin test that is a more sensitive marker confirming myocardial damage. This could have had added value as his CK-MB value was normal. A cardiac MRI if done early may be able to aid diagnosis in confirming the cause of initial cardiomyopathy. However, this was not done as the service was not readily available at our centre.

CONCLUSION

Our case highlights that thyrotoxicosis can present as ST elevation myocardial infarction and may be a potential cause for MINOCA. A diagnosis of hyperthyroidism should be considered in a patient with little or no risk factors presenting with AMI.

Ethical Consideration

Authors certified that all efforts to secure patient consent have been exhausted to no avail. All information in the case report has been provided without mention of name and every effort has been taken to ensure anonymity. They have sought permission from the Head of the Department of Medicine of Hospital Raja Perempuan Zainab II (HRPZ II) to publish the case.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

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