CASE REPORT

A stormy chase of coronary artery spasm: Thyroid storm in acute myocardial infarction

Yik Hon Ho, MBBS[1], Caryn Tsujean Lim, MB BCh BAO[1], Han Bing Chow, MRCP[1,2], Pei Lin Chan, MRCP[3], Chen Ting Tan, MRCP[1], Asri Said, MMed[1,2]

[1]Department of Cardiology, Sarawak Heart Center, Kota Samarahan, Sarawak, Malaysia, [2]Department of Medicine, Faculty of Medicine and Health Sciences, University of Malaysia Sarawak, Kota Samarahan, Sarawak, Malaysia, [3]Endocrinology Unit, Department of Medicine, Sarawak General Hospital, Kuching, Sarawak, Malaysia

SUMMARY

Cardiovascular disease represents the largest cause of death globally, with multifactorial causes. This is a case study of a thyroid storm with hyperactive coronary arteries resulting in acute myocardial infarction (AMI). A 56-year-old gentleman presented with left-sided chest pain radiating to the neck. Initial ECG showed ST elevation over anterior leads, which dynamically changed to Lambda-wave ("shark fin" pattern) over inferior leads 60 minutes later, along with raised cardiac enzymes. Urgent invasive coronary angiogram was performed, in which we repeatedly did ballooning of the left anterior descending coronary artery due to recurrent spasms in the artery, with the patient's haemodynamics being labile with systolic blood pressure of as low as 60 mmHg. He was intubated for acute pulmonary oedema and admitted to cardiac intensive care unit post-procedure and had persistent tachycardia with multiple episodes of tachyarrhythmia. Laboratory test revealed hyperthyroidism, and he was treated for thyroid storm with Burch-Wartofsky Point Scale of 50 points. However, his clinical condition deteriorated rapidly with the development of acute kidney injury and severe metabolic acidosis. He eventually succumbed after 4 days of intensive care despite maximum multidisciplinary resuscitation effort. This study calls for routine screening of thyroid function test in patients with persistent tachycardia during AMI.

INTRODUCTION

Cardiovascular disease represents the largest cause of death globally, claiming about 17.9 million lives each year, contributing to an estimation of 32% of all deaths worldwide.[1] Despite various works on primary and secondary prevention of myocardial infarction (MI), the climbing trend of the disease suggests a multifactorial cause of disease processes. This case study reported thyroid storm in the setting of AMI and highlighted the possibility that hyperthyroidism may be an overlooked cause of MI which has been increasingly recognised as a potential contribution to the disease occurrence.

CASE REPORT

A 56-year-old gentleman who was an active smoker with underlying type 2 diabetes mellitus was admitted to the Cardiology Department for ST elevation myocardial infarction (STEMI). He initially presented with left-sided chest pain radiating to the neck; associated with headache, nausea and reduced effort tolerance. His symptoms lasted for 2 weeks but worsened on the day of presentation. He did not complain of tachycardia, tremors or loss of weight.

Physical examination noted initial heart rate to be within the higher end of normal range, but later became increasingly tachycardic. Initial 12-lead electrocardiogram (ECG) showed ST elevation over leads aVR and V1 with reciprocal ST depression over leads I, II, III, aVF and V3-V6 and tall tented T wave over leads V2-V4, suggestive of extensive anterior STEMI (Figure 1A). Subsequent ECG 60 minutes later revealed significant dynamic changes of Lambda-wave ("shark fin" pattern) over inferior leads and reciprocal deep ST depression over leads I, aVR, aVL and V1-V2, suggestive of inferior STEMI (Figure 1B). Laboratory test showed elevated cardiac enzymes (CK 682 IU/L, CK-MB 43 IU/L).

An urgent invasive coronary angiogram revealed intermediate (50% stenosis) in the left main lesion and the most significant lesion (80% stenosis) was at the proximal left anterior descending artery (LAD). Balloon dilation was done for the LAD with a semi-compliant 2.5 mm x 12 mm balloon. The proximal LAD showed improvement but there was a new spasm at the mid and distal LAD with a drop in systolic blood pressure to 80 mmHg. Balloon dilation was repeated at sub-nominal pressure with improvement to the flow. The proximal to mid-segment of the LAD was then stented with a 3.0 mm x 34 mm drug eluting stent. Soon after, the left main showed spasms, and there was also no reflow to the left circumflex artery (LCX) with a further drop in systolic blood pressure to 60 mmHg (Figure 2A-L). Fluid resuscitation was done and a bolus of Glycoprotein IIb/IIIa inhibitor (tirofiban) was given. A repeated angiogram showed re-established flow to LCX and TIMI 3 flow to both LAD and LCX and an improvement of BP to 140/100 mmHg.

After the procedure, the patient was transferred to coronary care unit, and he was given Glycoprotein IIb/IIIa inhibitor (tirofiban) over 24 hours. However, he developed acute pulmonary oedema post-primary cutaneous intervention and was intubated due to impending respiratory collapse. Physical examination showed persistent tachycardia with heart rate ranged from 150 to 170 beats per minute. Cardiac monitor showed multiple episodes of transient

This article was accepted: 22 November 2022
Corresponding Author: Yik Hon Ho
Email: richardho920825@gmail.com

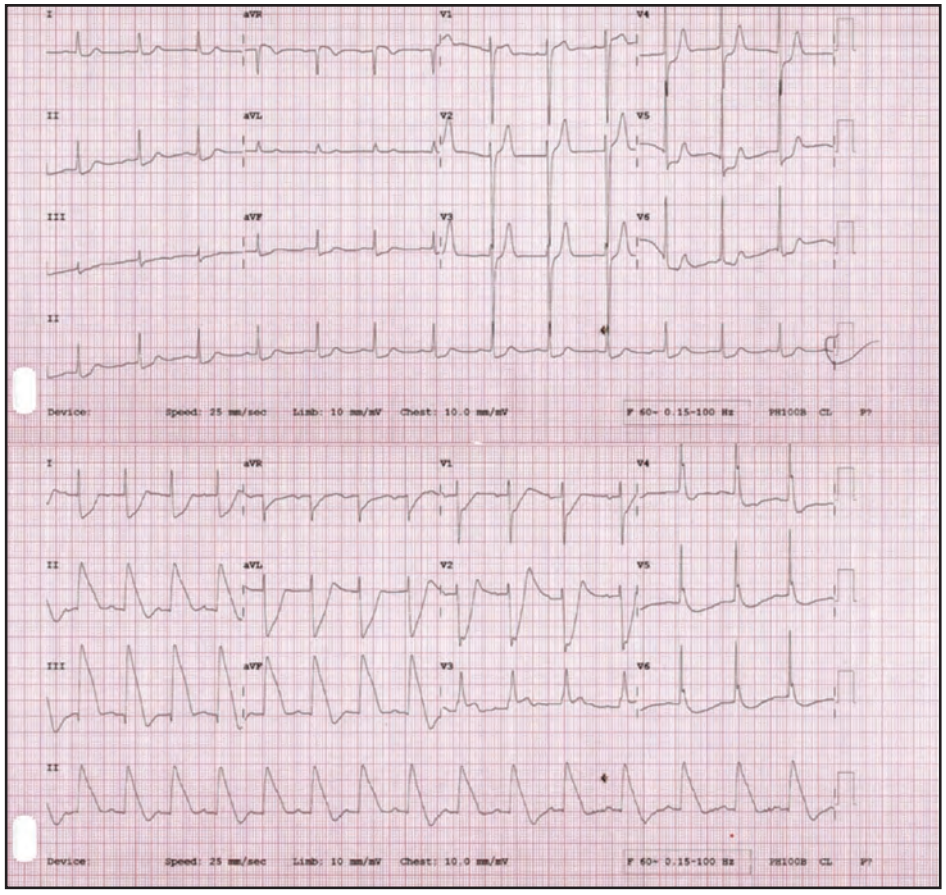

Fig. 1: 12-lead electrocardiogram. (A) Presence of ST elevation over ECG leads aVR and V1 with reciprocal ST depression over leads I, II, III, aVF and V3-V6 and tall tented T wave over leads V2-V4, suggestive of extensive anterior STEMI. (B) Subsequent 12-lead electrocardiography 60 minutes later showed significant dynamic changes; Lambda wave of shark-fin pattern over inferior leads and reciprocal deep ST depression over leads I, aVR, aVL and V1-V2, suggestive of inferior STEMI.

supraventricular tachycardia, which were spontaneously aborted. Thyroid function test (TFT) was sent, which revealed a low concentration of thyroid-stimulating hormone (TSH <0.005 uIU/mL) and a significantly elevated free thyroid hormone level (fT4 50.08 pmol/L; reference range 9.01–19.05 pmol/L). His Burch-Wartofsky Point Scale was 50 points which was highly suggestive of thyroid storm.

The patient had no history of thyroid disease until current hospitalisation. He was treated for thyroid storm and was given Lugol's iodine, hydrocortisone and carbimazole. Later, he developed non-sustained ventricular tachycardia in which he was chemically cardioverted with amiodarone. Despite the intensive treatment, patient's condition continued to deteriorate with increasing requirement of inotropic support. Transthoracic echocardiography showed a poor ejection fraction of 17.8% using Simpson's biplane method. NT pro-Brain natriuretic peptide (NT-proBNP) was raised at 6660 pg/mL. Patient also developed acute kidney injury with severe metabolic acidosis but renal replacement therapy was not commenced as he was clinically unstable. Despite multidisciplinary effort of maximum resuscitation, patient succumbed after 4 days of hospitalisation.

DISCUSSION

Thyrotoxicosis in the setting of AMI is not commonly been thought of and may be more commonly present than it was clinically diagnosed. The presence of thyroid hormone (TH) receptors in myocardium and vascular endothelial tissues allows changes in circulating TH concentration to modulate end-organ activity.[2] ThyrAMI 1 study done on the population of North of England showed that thyroid dysfunction is common in acute myocardial infarction (AMI) patients on admission to hospital.[3] This patient was diagnosed with hyperthyroidism upon hospitalisation for cardiac event after being noted to have persistent tachycardia. He did not report any symptom of hyperthyroidism prior to current admission, which may be due to the lack of recognition of symptoms among Asian patients, and the only precipitating factor to the occurrence of thyroid storm was AMI. One might also wonder the possibility of diabetes mellitus in masking the symptoms of hyperthyroidism, hence he might have long-standing hyperthyroidism but never been symptomatic to seek medical attention. However, there is no literature so far to suggest such phenomenon. There is also no possible way to establish the temporal relationship of either hyperthyroidism had caused the AMI first or AMI had precipitated the occurrence of thyroid storm.

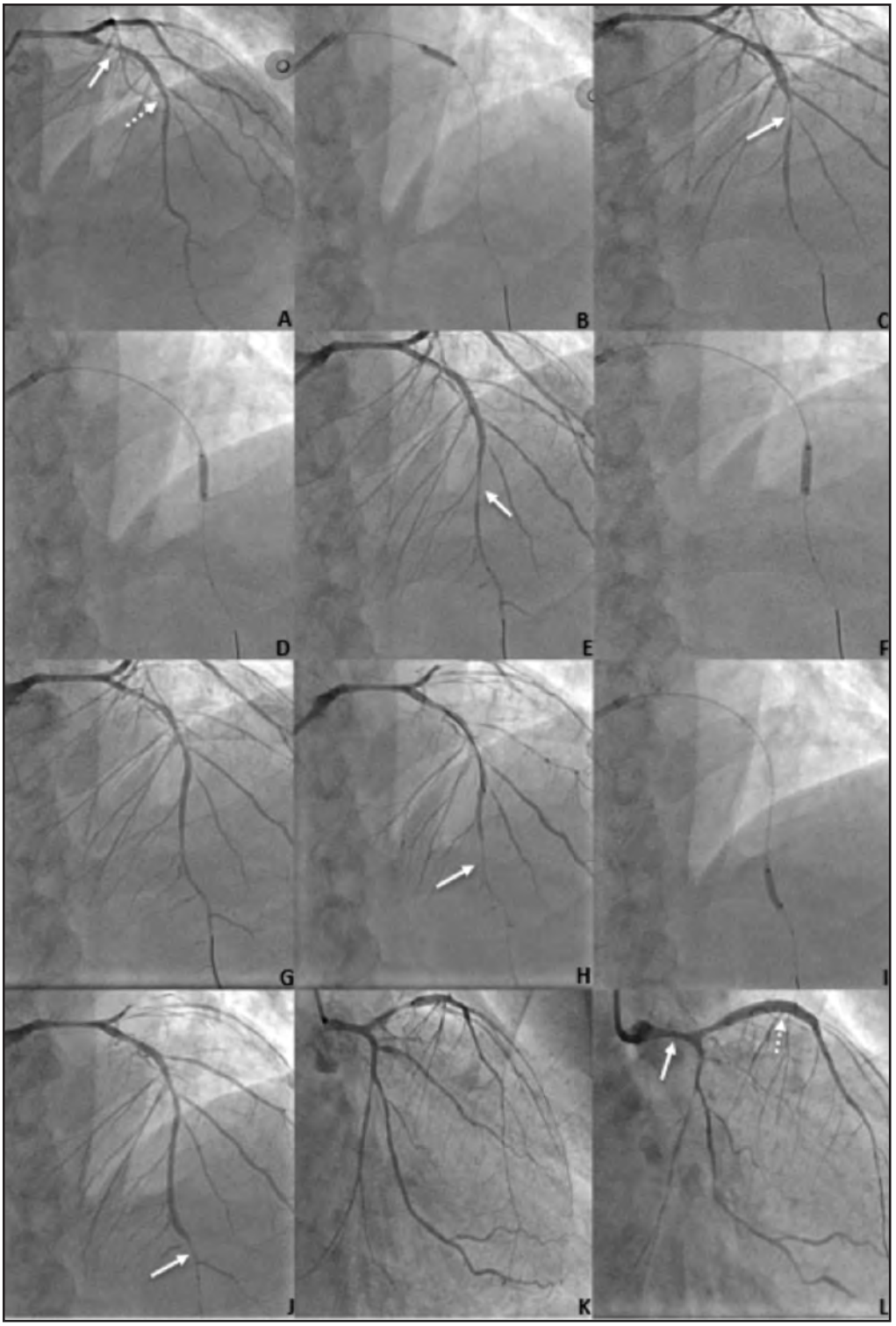

Fig. 2: Series of images from invasive coronary angiogram. (A) AP cranial view during invasive coronary angiography showing 80% stenosis at the proximal (white arrow) and 50% stenosis at the mid-left anterior descending artery (LAD) (dotted arrow) before PCI. (B) Pre-dilatation of proximal LAD with balloon. (C) AP cranial view showing a new tight stenosis between second and third diagonals post dilatation with balloon (white arrow). (D) Further pre-dilatation of the new lesion with balloon. (E) AP cranial view showing a new tight stenosis distal to the third diagonal post dilatation with balloon (white arrow), suggestive of possible coronary artery spasm. (F) Further pre-dilatation of the new lesion with balloon. (G) AP cranial view showing no new coronary artery spasm post dilatation with balloon. (H) AP cranial view showing new coronary artery spasm at the distal LAD (white arrow) during the removal of intracoronary wire. (I) Further pre-dilatation of the new lesion with a balloon. (J) AP cranial view showing residual coronary artery spasm at the far distal LAD (white arrow) post dilatation with a balloon. (K) RAO caudal view showing normal left main stem before PCI. (L) RAO caudal view showing new left main spasm (white arrow) after PCI of the proximal LAD with drug-eluting stent (dotted arrow).

Although the effect of hyperthyroidism on various organs is usually considered reversible, our patient did not experience any significant improvement after delivery of hyperthyroidism treatment. On the contrary, his clinical condition steadily went downhill, and he quickly succumbed. His thyroid dysfunction may have contributed to the adverse outcome of the cardiovascular event. Studies have shown that patients who have thyroid disease had higher risk of death from cardiovascular disease.[4] Compared to cases of AMI with hyperthyroidism who had relatively normal coronary arteries,[5,6] our patient had multi-vessels disease which might contribute further to his poor outcome.

The exact mechanism of MI in thyrotoxicosis is unclear. However, there are several postulations on the possible pathophysiology. There are evidences showing hyperthyroidism as a pro-thrombotic state due to an increase in fibrinogen and factor X levels.[7] Higher von Willebrand antigen levels in thyrotoxicosis lead to enhanced platelet plug formation.[8] These may result in in-situ coronary thrombosis. In addition, direct metabolic effect of thyroid hormone on myocardium or as a result of arrhythmia are potential contributory factors.[9] While the development of tachyarrhythmias in our patient might be due to a direct effect of coronary artery occlusion, thyroid dysfunction might have contributed to the event.

There were subtle clues from the ECG that this case was not a straightforward AMI in that the ST elevation changed from inferior leads to anterior leads suggesting that the patient has had coronary spasms rather than an acute thrombosis. The presence of recurrent coronary artery spasm in our patient during invasive coronary angiogram was consistent with the finding of other cases of MI in young patients with thyrotoxicosis.[6,9] This observation might be due to the enhanced vasoconstriction effect from catecholamines and sympathetic α-adrenergic receptor stimulation from increased sensitivity and numbers of adrenergic receptors in thyrotoxicosis state.[6] The awareness of a different pathophysiology in patients with MI may influence the management in which it might differ from typical MI secondary to atherosclerotic plague formation. In MI with thyrotoxicosis due to severe coronary artery spasm, the coronary artery spasm may be completely relieved by intracoronary administration of nitrates without the need for stenting.[6] Non-dihydropyridine calcium channel blockers and long-acting nitroglycerin can also be used in the treatment of vasospastic angina.[10] However, the severe hypotension in our patient precludes the administration of intracoronary nitrates or other anti-vasospastic agents.

There is growing evidence to suggest thyrotoxicosis as a potential cause of MI.[3,5,6,9] Screening of TFT in patients with MI especially with the observation of persistent tachyarrhythmia should be considered. Urgent TFT testing is of paramount importance in confirming the diagnosis of thyroid storm upon clinical suspicion for timely administration of treatment. It is important to achieve euthyroid state in patients with thyrotoxicosis to improve mortality outcomes.

CONCLUSION

Thyroid storm as an atypical cause of AMI is rarely diagnosed in clinical practice. Coronary artery spasm is a common finding in many cases of AMI with thyrotoxicosis. While intracoronary administration of nitrate and other vasospastic treatment were proven to be effective in relieving coronary artery spasm, its role might be limited by clinical condition of hypotension. Patients who have cardiovascular disease profile with thyrotoxicosis might have poorer outcomes compared to patients who did not have typical cardiovascular disease profile presenting as AMI. Our study resonates with other studies in which thyroid dysfunction can be a cause of AMI. This has called for a routine screening of thyroid function test in patients with myocardial infarction to allow for early delivery of thyrotoxicosis treatment to improve mortality outcomes.

ACKNOWLEDGEMENT

The authors would like to thank the Director General of Health Malaysia, Clinic Research Centre (CRC) Sarawak General Hospital and Sarawak Heart Center for the permission to publish this article. We would also like to thank University Malaysia Sarawak for the assistance in preparing this manuscript.

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

1. World Health Organization. 2011. Cardiovascular diseases [cited February 2022]. Available from: https://www.who.int/health-topics/cardiovascular-diseases#tab=tab_1.
2. Razvi S, Jabbar A, Pingitore A, Danzi S, Biondi B, Klein I, et al. Thyroid hormones and cardiovascular function and diseases. J Am Coll Cardiol 2018; 71(16): 1781-96.
3. Jabbar A, Ingoe L, Thomas H, Carey P, Junejo S, Addison C, et al. Prevalence, predictors and outcomes of thyroid dysfunction in patients with acute myocardial infarction: the ThyrAMI-1 study. J Endocrinol Invest 2021; 44(6): 1209-18.
4. Franklyn JA, Maisonneuve P, Sheppard MC, Betteridge J, Boyle P. Mortality after the treatment of hyperthyroidism with radioactive iodine. New Engld J Med 1998; 338(11): 712-8.
5. Zhou D, Qu Z, Wang H, Wang Z, Xu Q. Severe hyperthyroidism presenting with acute ST segment elevation myocardial infarction. Case Reports Cardiol. 2015; 901214.
6. Kuch M, Rostek M, Chmielewski M, Bargieł M. Myocardial infarction caused by Graves' disease. Endokrynol Pol 2021; 72(2): 185-6.
7. Dörr M, Robinson DM, Wallaschofski H, et al. Low serum thyrotropin is associated with high plasma fibrinogen. J Clin Endocrinol Metab 2006; 91: 530-4.
8. Homoncik M, Gessl A, Ferlitsch A, Jilma B, Vierhapper H. Altered platelet plug formation in hyperthyroidism and hypothyroidism. J Clin Endocrinol Metab 2007; 92: 3006-12.
9. Masani ND, Northridge DB, Hall RJC. Severe coronary vasospasm associated with hyperthyroidism causing myocardial infarction. British Heart J 1995; 74(6): 700-1.
10. European Society of Cardiology. 2020. Acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation (management of) guidelines [cited February 2022]. Available from: https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Acute-Coronary-Syndromes-ACS-in-patients-presenting-without-persistent-ST-segm.