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Case Report

Electrocardiographic changes mimicking Acute coronary syndrome in a patient of hyperthyroidism

Vikas Singh [a,*], Varun Shankar Narain [b], Aniket Puri [c], Rishi Sethi [c]

[a] Consultant – Cardiology, Paras HMRI Hospital, Patna, India
[b] Professor, Department of Cardiology, King George's Medical University, Lucknow, India
[c] Associate Professor, Department of Cardiology, King George's Medical University, Lucknow, India

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ABSTRACT

We present a case of hyperthyroidism presenting as Acute Coronary Syndrome (ACS) with marked ST-T changes, classical ischemic chest pain and raised cardiac enzymes, epicardial coronary arteries being completely normal on angiography.

1. Introduction

Cardiovascular manifestations are a prominent presenting feature of hyperthyroidism. Tachycardia is very common. Symptoms and signs of heart failure and atrial fibrillation may dominate the clinical picture particularly in elderly.

In rare cases, patients with hyperthyroidism can present with chest pain or ECG changes suggestive of cardiac ischemia. In older patients with known or suspected underlying coronary artery disease, this reflects the increase in myocardial oxygen demand in response to the increase in cardiac contractility and workload associated with thyrotoxicosis. Rarely, however, young patients with no known cardiac disease can manifest similar findings. In such patients, coronary angiography often demonstrates normal coronary anatomy, and the cause for these findings has been related to coronary vasospasm, microvascular disease or thromboembolism with recanalization of the arterial lumen.

2. Case discussion

A 40 yr lady, thin built, postmenopausal, was admitted to the ER with severe chest pain radiating to left arm at rest, on and off for 3 days. She had no conventional risk factors for coronary artery disease and gave a history of breathlessness for a week prior to admission. On examination, pulse rate was 110/min, regular; blood pressure 90/60 mm Hg; chest examination showed bilateral basal crepts, while the cardiovascular examination was unremarkable. The patient had a large nodular goitre but did not have any ophthalmic or dermatologic signs of hyperthyroidism. She admitted to have known of some

* *Corresponding author*. RCA-30, Near-TV Tower, Bahadurpur Housing Colony, Kankarbagh, Patna-800026, India. Tel.: +91 9661803767.
E-mail address: dr.vikas.s@gmail.com (V. Singh).

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thyroid disorder, and was on irregular medications for the same.

The ECG on admission showed generalized T wave inversion, and ST coving in leads V2-5, in addition to sinus tachycardia. (Fig. 1)

She was admitted to the cardiac care unit with a provisional diagnosis of Acute Coronary Syndrome (ACS). The quantitative Troponin T done at admission was 0.193 ng/ml (ref: <0.01 ng/ml). Repeat ECG done showed a greater ST elevation in the precordial leads (Fig. 2). The repeat Troponin T after 6 h showed a rising trend, value being 0.205 ng/ml.

X-Ray Chest showed a normal CT ratio, with evidence of pulmonary venous congestion. 2-D echocardiography done showed global LV hypokinesia (normal LV dimensions) with an ejection fraction of 41% and mild mitral regurgitation. No regional wall abnormalities were demonstrated. No evidence of pericardial fluid was found. The ECG of the patient continued to show gradually increasing ST elevations in all leads except V1, aVR and aVL. (Fig. 3) However, no signs of pericarditis could be found clinically or on repeat 2-D Echo. The patient was taken in for a coronary angiogram which revealed normal epicardial arteries (Fig. 4) with normal TIMI flows, TIMI counts and TIMI perfusion grades.

Thyroid function tests available the next day showed elevated T3, T4 and a markedly depressed TSH. T3 = 8.38 (ref 1.49–2.96), T4 = >320 (ref 71.20–171)), TSH = <0.015 (ref 0.47–4.52). Serum sodium and potassium levels were normal. The patient was started on anti-thyroid medications in addition to frusemide aldactone combination, ramipril and metoprolol.

She showed good clinical response to the drugs with respect to heart failure and chest pain, and the ST elevation on ECG showed settlement while the T wave inversions continued much the same as seen on the presenting ECG (Fig. 5).

The patient was seen at 2 weeks and 6 weeks post discharge in the OPD. She continued to do well on drugs. A repeat 2D-Echo at 6 weeks showed normal EF without any evidence of regional wall motion abnormality (RWMA). Thyroid function tests were within range at 6 weeks (T4-150 nmol/L; TSH-0.5 mIU/ml). The ECG was quite like that at discharge showing T inversions but the ST elevations had settled.

3. Discussion

In a patient with chest pain and ST-T segment changes on the ECG, myocardial ischemia due to coronary atherosclerosis is the most likely diagnosis. ST-T segment changes in the ECG are seen in many conditions other than coronary atherosclerosis e.g, pericardial disease, hypothyroidism, electrolyte abnormalities, digoxin therapy, cardiomyopathies. Some ST-T segment changes have been described in otherwise normal individuals, 'normal variants'.

A Bhattacharya[1] reported a case of thyrotoxicosis without any evidence of peripheral neuropathy, with ST segment elevation in whom an initial diagnosis of acute coronary myocardial syndrome was made.Myocardial stunning in hyperthyroidism has been described by Perera et al.[2] Andrew Ying-Siu Lee et al[3] reported two patients with hyperthyroidism presenting as Acute coronary syndrome. Successful treatment of hyperthyroidism resulted in subsidence of angina.

Cardiovascular manifestations of thyroid disorders are well described in literature.[4,5,6] Yokoyama[7] reported a young Japanese man with thyrotoxic periodic paralysis who had ST segment depression during such an episode precipitated by hypokalemia (serum potassium 2.7 mmol/L). Abnormal LV function in hyperthyroidism has been described by Forfar et al[8] and possible reversible cardiomyopathy has been cited as the likely mechanism. Pronounced ST-T segment changes were also reported in two young female patients with Grave's disease and systemic myopathy; they had no overt cardiac disease, and the changes disappeared with treatment.[10] T wave changes possibly due to myo-pericardial involvement were reported in 22% of the 123 hyperthyroid patients studied by Hoffman and Lowrey[11] but neither depression nor elevation of the ST segment was reported.

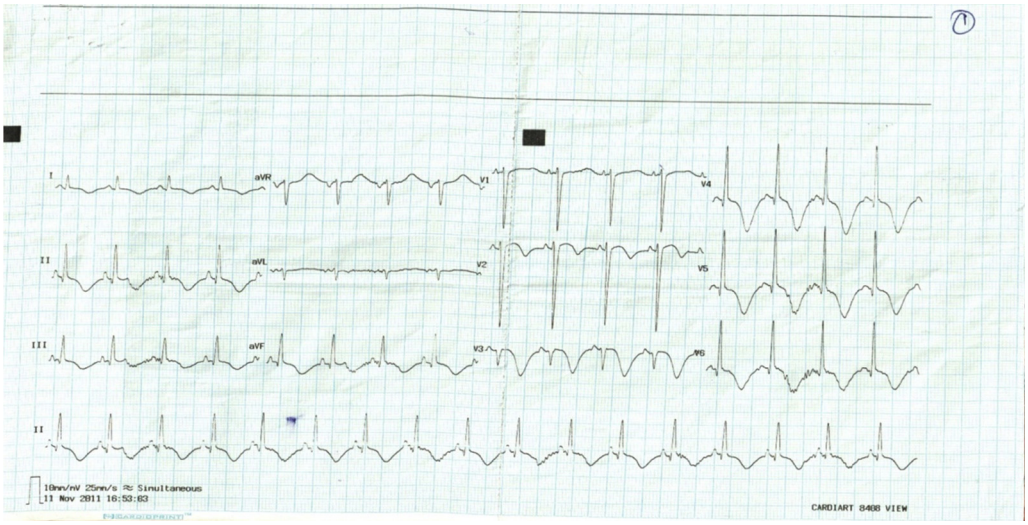

Fig. 1 – Admission ECG showing generalized T wave inversions.