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HYPOTHYROIDISM- AN UNDERRATED DIFFERENTIAL TO CHEST PAIN?

General Medicine

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ABSTRACT

While treating a patient with left sided chest discomfort associated with anxiety, palpitation, dyspnea or diaphoresis, our vision sometimes narrows down to primarily the heart, lungs and esophagus. We report 3 cases where patient reported with symptoms mimicking acute coronary syndrome but detailed evaluation and investigations ruled out cardiac abnormalities, rather hypothyroidism was attributed to for the symptoms.In the field of medical science where rarities are no exception, a physician should try to exclude hypothyroidism in cases where he finds the conventional evaluations for a case of chest pain inconclusive.

KEYWORDS

Creatine kinase-muscle/brain (CKMB), Troponin I, Thyroid Stimulating Hormone (TSH), Woltman's Sign.

INTRODUCTION

It has become a protocol that when a patient reports with left sided chest discomfort associated with anxiety, palpitation, dyspnoea or diaphoresis the first investigation that a clinician asks for is ECG. If the ECG is suggestive of any ST-T changes then the saga of tests from cardiac biomarkers to 2D-echocardiography follow. But we found that in some cases these investigations lead to no cardiac abnormality but certain other symptomatology encouraged us to go for assessing the thyroid function of the patients. We present 3 such cases where hypothyroidism was found to mimic acute coronary syndrome.

CASE 1

43-year-old morbidly obese female reported to the OPD with complaints of headache, breathlessness, nausea, dyspnoea and chest discomfort. During evaluation, the patient had syncope in the out-patient department. Patient was immediately rushed to ICU where her vitals were found to be normal (inclusive of random blood sugar). Patient's ECG(electrocardiogram) was suggestive of T inversion from V_{2-6} with normal sinus rhythm (figure 1). Cardiac biomarkers were sent where patient was found to have CKMB of 99 U/L with normal troponin I levels. Follow up CKMB were also found to be elevated. Patient was sent for 2D-echocardiography which turned out to be normal. On further evaluation we considered her obesity and increased somnolence in the ward and went for thyroid function test where TSH was found to be 41uIU/ml. Patient was started on levothyroxine and discharged. On her 3-monthly follow-up, patient was found to have significant symptomatic improvement and reduction in weight.

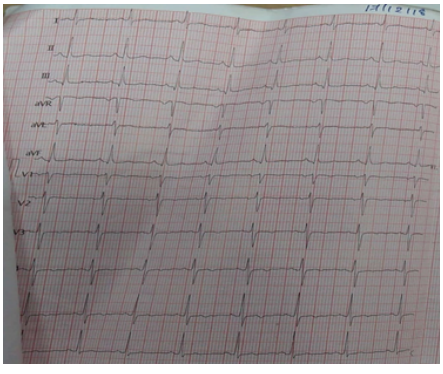

FIGURE 1- ECG of case 1

CASE 2

28-year-old female, a known case of hypothyroidism on levothyroxine therapy, reported to casualty with complaints of dyspnoea, left sided chest pain and palpitations. ECG was suggestive of T inversion in lead II, III, avF, V3-6(figure 2). Cardiac biomarkers were found to be normal. Follow up ECGs showed no fresh changes. 2D-echocardiography was done which was found to be normal. Thyroid function test was done. TSH was found to be 33 uIU/ml with anti-TPO 90.6 IU/ml. Patient's levothyroxine dosage was increased and the patient was discharged. On her monthly follow up patient was found to have significant clinical improvements and normalization of her ECG.

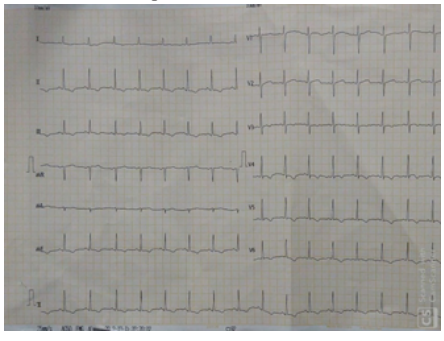

FIGURE 2- ECG of case 2

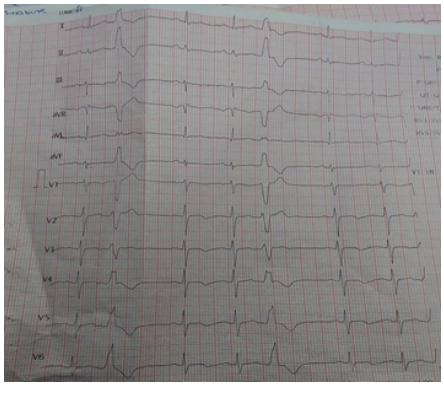

FIGURE 3- ECG of case 3

DISCUSSION

Hypothyroidism is a common disorder in OPD and when patient presents with classical symptoms and signs, it is easy to recognize. Most common symptoms of hypothyroidism include myalgia, lethargy, dry skin, hoarseness, constipation, cold intolerance, menorrhagia and weight gain. Some patients may present with features of depression. Signs of hypothyroidism include bradycardia, Woltmans sign, diastolic hypertension, pedal edema. However, some patients may present with atypical symptoms like chest pain mimicking myocardial infarction.[1] Some enzymes like SGOT, lactic dehydrogenase (LDH), and creatine phosphokinase (CPK), are measured in patients with myocardial infarction , but they are present in a variety of tissues and are released into serum under a variety of circumstances other than myocardial infarction [2] like cardioversion [3,4], tachyarrhythmias [4], polymyositis, pulmonary embolism. Hypothyroidism also can lead to persistent elevation of serum creatine kinase, lactate dehydrogenase. The rise in enzyme in hypothyroidism is attributed to leakage from increased cell permeability. [5] 90% patients with hypothyroidism may have a raised CK. Most commonly it is due to the CK-MM fraction, although CK-MB fraction may be elevated. The enzyme elevation may resolve after treatment of hypothyroidism. [6] ECG changes in hypothyroidism include T-wave flattening or inversion, low amplitude P waves and QRS complex or prolongation of PR interval and QRS widening. Hypothyroidism may also cause prolongation of the QT interval. [7] CK and its MB fraction may be raised in patients with hypothyroidism but Troponin levels are generally not, even in patients with increased CK-MB. [8] Clinically, the diagnosis of hypothyroidism can be difficult in patients with atypical symptoms. The diagnosis of myocardial infarction (MI) has been based on a history of chest pain with electrocardiographic (ECG) changes in the form of new Q-waves and evolving ST-T wave changes. [9] A high index of suspicion is required to consider hypothyroidism in such cases.

CONCLUSION

Hence, in patients presenting with chest pain and raised CK and/or ECG abnormalities but with a negative Troponin I and with no change in serial ECG, a diagnosis of hypothyroidism may be considered.

In the 3 cases we have reported above, all 3 patients had symptoms mimicking myocardial infarction.

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