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Case Report

SUBCLINICAL HYPOTHYROIDISM PRESENTING AS CARDIAC SYNDROME X

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ABSTRACT
Cardiac manifestations of subclinical hypothyroidism (SCH) are due to a decrease in cardiac output and cardiac contractility, a reduction in heart rate, and an increase in peripheral vascular resistance as with overt hypothyroidism. Dyslipidemia and ischemic heart disease have also been documented. Cardiac syndrome X (CSX) has rarely been reported with subclinical hypothyroidism. We present a rare case of subclinical hypothyroidism with CSX.

Key Words: *Subclinical Hypothyroidism, CSX, Dyslipidemia*

INTRODUCTION

Subclinical hypothyroidism is a Subclinical hypothyroidism (SCH), also called mild thyroid failure and is diagnosed when peripheral thyroid hormone levels are within normal reference laboratory range but serum thyroid - stimulating hormone (TSH) levels are mildly elevated. This condition occurs in 3% to 8% of the general population. The possibility that it is a cardiovascular risk factor has been a subject of debate. Studies have shown slowed left ventricular relaxation time, increased vascular tone at rest, and left ventricular systolic dysfunction with exercise and impaired endothelial function. The cross-sectional Rotterdam Study showed an association of SCH with myocardial infarction and aortic calcification. CSX is a situation where a patient presents with angina pain, ischemic ST/T wave changes in ECG and positive TMT for stress induced ischemia, but coronary angiography reveals normal coronaries. Association of CSX with SCH has been extremely rare.

CASES

A 45 year old female presented to us with chief complaints of exertional chest pain (effort angina) since 2 months, fatigability (not related to exertion) since 2 months, and change in voice since one and a half months. There was no history of exertional dyspnoea, paroxysmal nocturnal dyspnoea, orthopnoea, oedema feet, syncope and cough/haemoptysis. On asking leading questions she gave history of heat intolerance and puffiness of face. There were no urinary complaints. There was no history of diabetes mellitus and hypertension.

On examination – Pulse – 90/min regular, Blood Pressure – 140/80mm Hg in right arm, Jugular Venous Pressure was normal, non-pitting oedema was present in both lower limbs. Cardiovascular and Respiratory system examination was normal, per - Abdomen examination was normal. CNS examination – delayed relaxation of ankle and bisceps jerk was demonstrated. A provisional diagnosis of Hypothyroidism with Ischaemic Heart Disease was made and patient was investigated.

Complete Blood Count, Kidney Function Test, Liver Function Test were normal. X-ray chest was normal. ECG revealed diffused ischaemic t wave inversion from v1 – v6 and sinus rhythm (Figure 1) Thyroid profile revealed FT3- 3.2 pg/ml (normal 1.7-4.2pg/ml) FT4- 1.2 ng/dl (normal 0.70-1.80ng/dl), TSH- 12 µIU/ml (normal 0.30-5.5 µIU/ml) suggestive of subclinical hypothyroidism. Tread Mill Test was positive for stress induced ischaemia. 2D echocardiography was normal. Coronary Angiography was done which revealed absolutely normal coronary arteries, suggestive of the classic Cardiac Syndrome X.

Patient was started on tablet levothyroxine 25 micrograms per day, Tab. Metoprolol 25 mg OD, Tab. Enalapril 5 mgOD, Tab. Atorvaststin 10 mg HS, and was advised to take sublingual nitrate 5 mg when

angina occurs. After 2 months of follow up, angina episodes significantly decreased. Subjectively she was feeling better. A repeat serum TSH was 7 µIU/ml. She was advised to continue treatment.

DISCUSSION

Mild thyroid failure or Subclinical hypothyroidism is a condition where serum T3 and T4 are normal with increased serum TSH levels. It is usually a compensatory phase of decreased thyroid gland activity. Myocardial function has been reported in multiple studies to be subtly impaired in patients with subclinical hypothyroidism. Identified functional abnormalities include impaired myocardial contractility and diastolic dysfunction, at rest or with exercise (Forfar *et al.,* 1985 and Foldes *et al.,* 1987). In one comprehensive study of exercise capacity, patients with mild thyroid failure were shown to have significant impairment of exercise-related stroke volume, cardiac index, and maximal aortic flow velocity. Pulmonary testing in these same patients revealed decreased vital capacity, reduced anaerobic thresholds, and decreased oxygen uptake at the anaerobic threshold (Arem *et al.,* 1996). These data clearly demonstrate that cardiovascular function in subclinical hypothyroidism is slightly impaired and not identical to that in the euthyroid state. Dyslipidemia in form of increased serum levels of total cholesterol and low-density lipoprotein (LDL) cholesterol, abnormal lipoprotein levels has also been demonstrated in many studies. Reduced high-density lipoprotein cholesterol in some studies. Some reports have suggested that even high normal serum TSH values may adversely affect serum lipid and lipoprotein levels.

One recent study reported that patients with mild thyroid failure, and even subjects with high normal serum TSH values, have evidence of endothelial dysfunction, manifested by impaired flow-mediated, endothelial-dependent vasodilatation (Lekakis *et al.,* 1997). The recent Rotterdam Study concluded that patients with subclinical hypothyroidism have a significantly increased prevalence of aortic atherosclerosis and myocardial infarctions (Hak *et al.,* 2000).

ECG changes in subclinical and over hypothyroidism are known to occur. The classic ECG changes in hypothyroidism are low voltage complexes, sinus bradycardia, widespread T wave inversions, QT prolongations, and first degree heart block. Our patient had T inversions in the anterior leads but with the view of tachycardia, we considered the T wave changes to be ischemic in origin rather than the effect of hypothyroidism.

Cardiac syndrome X (CSX) is an entity typically characterised by: predominantly effort induced angina, ST segment depression suggestive of myocardial ischaemia during spontaneous or provoked angina, with normal coronary arteries at angiography, absence of spontaneous or provoked epicardial coronary artery spasm, and absence of cardiac (for example, hypertrophic or dilated cardiomyopathy) or systemic (for example, hypertension, diabetes) diseases potentially associated with microvascular dysfunction. The basic pathophysiology postulated for CSX are coronary microvascular dysfunction (microvascular angina), and abnormal cardiac pain sensitivity (Cannon and Epstein, 1988). The causes of microvascular dysfunction in CSX have not yet been fully elucidated and are likely to be multiple. Structural abnormalities, mainly consisting of medial hypertrophy and/or fibrosis of arteriolar vessels, frequently associated with systemic hypertension, have been described in small series of patients. A frequently reported abnormality is endothelial dysfunction, suggested by a decreased coronary flow response to acetylcholine, atrial pacing, and other endothelium mediated vasodilator stimuli, and believed to be caused by impaired nitric oxide (NO) release and/or activity (Chen *et al.,* 2002). Interestingly the same pathology has been recently being described to occur in subclinical hypothyroidism leading to cardiac dysfunction. Most syndromes X patients' exhibit pronounced functional abnormalities of cardiac adrenergic nerve fibres that can explain the base line tachycardia in our patient (Lanza *et al.,* 1997).

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