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Acute Myocardial Infarction in a Young Male: An Unusual Manifestation of Hypothyroidism

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Abstract: People in developing countries suffer from coronary artery disease at a relatively younger age. Dyslipidemia is considered as established risk factor for cardiovascular diseases. Hypothyroidism constitutes a significant cause of secondary dyslipidemia. Secondary dyslipidemia causing acute myocardial infarction in a young adult is rare. We describe a case of acute myocardial infarction in a young male due to hypothyroidism.
Keywords: Dyslipidemia, Hypothyroidism, Myocardial infarction.

INTRODUCTION

Thyroid hormones have profound effects on lipoprotein metabolism and also on other metabolic parameters related to cardiovascular disease risk [1]. Hypothyroidism, a state of thyroid hormone deficiency is a well known cause of secondary dyslipidemia predisposing to development of atherosclerosis. Hypothyroidism causing acute myocardial infarction in young is very rare. We report here a case of acute myocardial infarction as an initial presentation of hypothyroidism in a young male without any other cardiovascular risk factors.

CASE REPORT

A 24 year old, non obese male presented to the emergency room with history of classical ischemic chest pain of four hours duration. There was no history of diabetes, hypertension, and premature cardiovascular deaths in the first degree family members. On examination patient was hemodynamically stable with pulse rate of 50/min and supine blood pressure 100/60mm Hg. No markers of atherosclerosis seen. Systemic examination was unremarkable.

Complete haemogram, blood sugar, renal function test, liver function test, serum electrolytes were normal. A twelve lead surface electrocardiogram (Fig. 1) showed significant ST segment elevation in anterolateral leads with reciprocal changes in inferior leads. Cardiac biomarkers (Creatinine Kinase MB and Troponin I) were elevated and also total cholesterol (371mg/dl) and low density lipoprotein (LDL) cholesterol (280mg/dl).

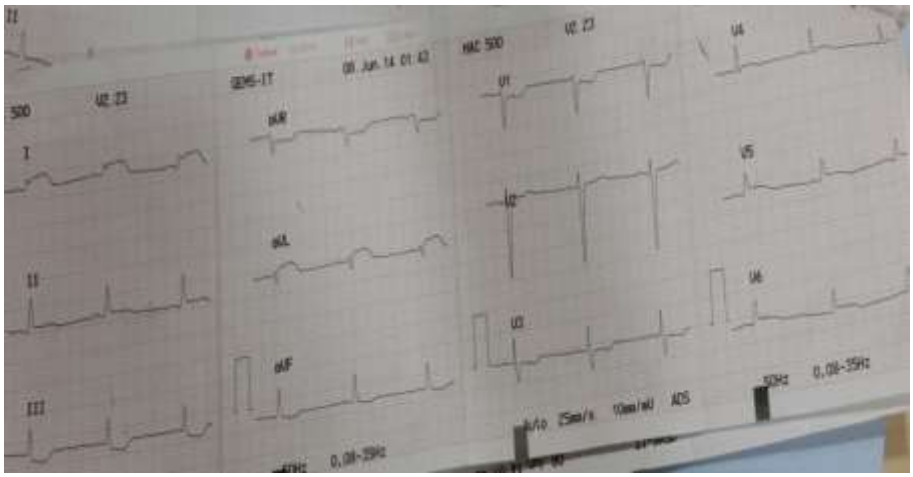

Fig. 1: Admission ECG showing STsegment elevation in I,avL,V5,V6 and ST segment depression in III, avF,avR,V1

Immunological and thrombophilia workup, Chest X-ray and 2D Echocardiogram were normal. Thyroid profile revealed hypothyroid state [TSH ->100 uIU/ml (Ref. Range 0.27-4.20uIU/ml),T3- 0.969 nmol/L (1.30-3.10 nmol/L), T4-19.35 nmol/L (66 -174 nmol/L)]. Patient was treated with stat dose of antiplatelets and statins. Thrombolysis was done with streptokinase. There was good response to thrombolysis as evidenced by resolution of ST segment elevation and disappearance of chest pain. Coronary angiogram [Fig. 2(A), 2(B), 2(C)] revealed normal coronaries. Patient was discharged after five days of uneventful stay in hospital with antiplatelets, statin and Levothyroxine.

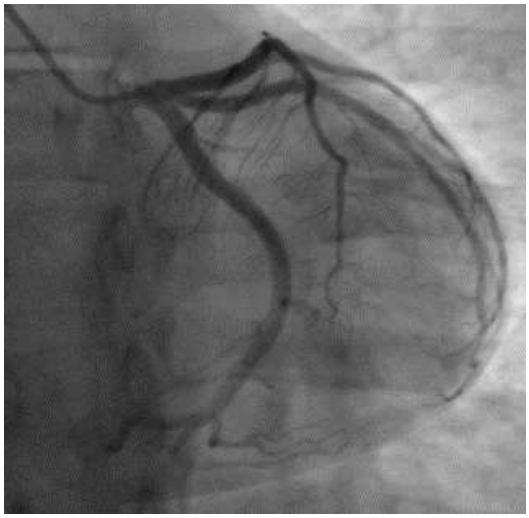

Fig. 2(A): Coronary angiogram in right anterior oblique caudal view illustrating a dominant, normal left circumflex artery

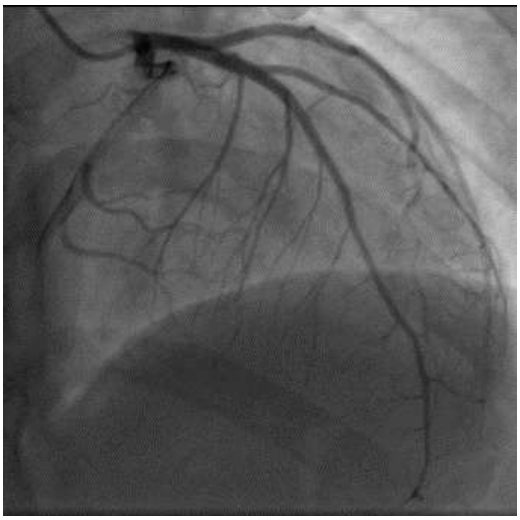

Fig. 2(B): Coronary angiogram in anterior-posterior cranial view – left anterior descending and diagonals are normal

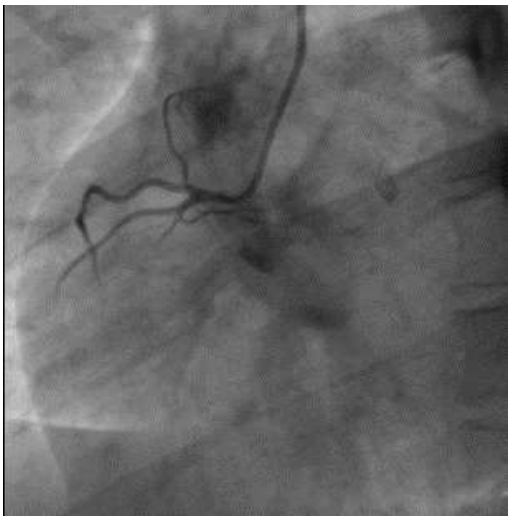

Fig. 2(C): Coronary angiogram showing non-dominant, small caliber and normal right coronary artery

DISCUSSION

Association between hypothyroidism and atherosclerosis was first noted by Greenfield in 1878[2]. Hypothyroidism doubles the risk of atherosclerosis compared to normal individuals [3]. The contributing factors for increased incidence are many and include elevated LDL cholesterol, diastolic hypertension, hyperhomocysteinemia and endothelial dysfunction [4].

Increased levels of total cholesterol and LDL cholesterol are due to decreased clearance of the particles as a result of reduced number LDL receptors on hepatic cells. LDL oxidation is also enhanced making the particle more atherogenic [4].

Diastolic hypertension is common in hypothyroidism due to increased peripheral vascular resistance and arterial stiffness. Hypothyroidism increases plasma homocysteine levels by modulating genetic expression of enzymes involved in homocysteine metabolism, changes in folate metabolism and decline of kidney function. There is increased prevalence of metabolic syndrome and waist-to-hip ratio in hypothyroidism [5].

Hypercoagulable state observed in hypothyroidism is attributed to decreased fibrinolytic activity [6].

Among all the metabolic and coagulation abnormalities due to hypothyroidism, only dyslipidemia was present in our patient.

CONCLUSION

Hypothyroidism is known for its protean manifestations, but hypothyroidism presenting as acute myocardial infarction in a young adult without any other risk factor is rare. This case highlights the need for thyroid profile estimation in all young dyslipidemic patients presenting with acute coronary syndrome.

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