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ACUTE MYOCARDIAL INFARCTION IN YOUNG DUE TO ANTITHROMBIN III DEFICIENCY

Cardiology

Dr. Azizul Haque Associate Professor, Department of Cardiology, Calcutta National Medical College.
Dr. Mita Bar* Senior Resident, Cardiology, Medical College Kolkata. *Corresponding Author
Dr. Prokash Chandra Bagchi Assistant Professor, Cardiology, Medical College, Kolkata.

ABSTRACT

AMI [Acute Myocardial Infarction] is uncommon in teenagers& mostly due to non-atheromatous disease, hyper-coagulable states,or substance abuse. Here is a case of a 16 years boy developing AMI due toAntithrombin IIIdeficiency in blood with normal coronary angiography in absence of other risk factors.

KEYWORDS

Acute myocardial infarction; young; Antithrombin III deficiency

INTRODUCTION

AMI[Acute Myocardial Infarction] is unusual in teenagers and may be due to various causes, including procoagulant states, congenital coronary anomalies, premature atherosclerosis, collagen vascular disorders, and substance abuse in absence of conventional CAD [Coronary Artery Disease] risk factors. Coronary arteries may be normal angiographically.

CASE REPORT

A 16-year-old boy, referred from local hospital ,Purba Medinipur (W.B) was admitted to the coronary care unit of Calcutta National Medical College,Hospital with history of vomiting, central chest pain & profuse sweating of 7 hrs duration. He was normotensive, nondiabetic,without any addiction history. He had no pasthistory of chest pain,respiratory distress, and palpitation. There was no family history of CAD, dyslipidemia or history of sudden cardiac death in family. His ECG showed ST-segment elevation in inferior leads (II,III,aVF)(Figure -1) and serum troponin T was positive.His pulse rate was 106/min, BP was 100/60mmHg. Auscultatoryfindings revealed no murmur and clear lung bases. His anthropometric parameters were: weight -35 kg, height 162 cm, BMI- 13.7 kg/m2. He was diagnosed as a case of acute inferior wall myocardial infarction and was thrombolysed with streptokinase. He was given aspirin, Clopidogrel, Atorvastatin . Subsequently,the patient became pain free and remained hemodynamically stable. Post thrombolysis followup ECGrevealed significant resolution of ST elevation in inferior leads. Echocardiography revealed hypokinesia of inferior wall with an ejection fraction of 42%. Blood biochemicalexamination revealed a fasting blood glucose of 91mg/dl , total cholesterol 137 mg/dl,LDL 80mg/dl, HDL 41 mg/dl, and triglyceride 104 mg/dl, Urea 32 mg/dl, creatinine 0.88 mg/dl, Hb-13.4 gm/dl, T.C.-12600/cmm,Platelet- 2.18 lakh/cmm, CPK-4713, CPK-MB-98 .Serum Lp(a) [estimation done by Particle enhanceimmunoturbidimetric test] was 8.90 mg/dl, Serum Homocysteine – 10.26 µmol/L,Factor V Leiden-Mutation Detection-Negative,Protein C Functional Activity – 77.2% (normal 70-171),Protein S Antigen-Free(Immuno-turbidometry)-73.9% (67.5-139%), Anti Thrombin Activity(Functional)- 58.8 % (87-131).

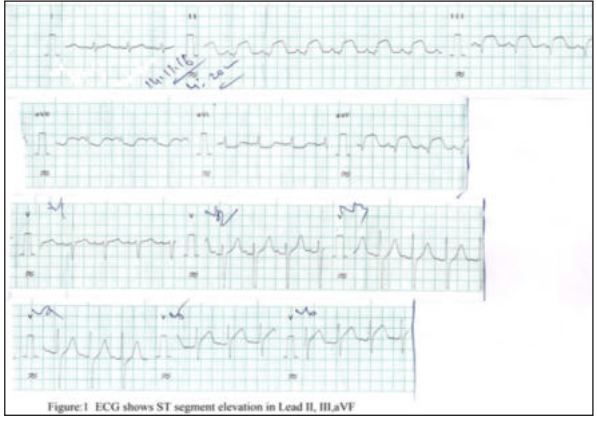

Figure:1 ECG shows ST segment elevation in Lead II, III,aVF

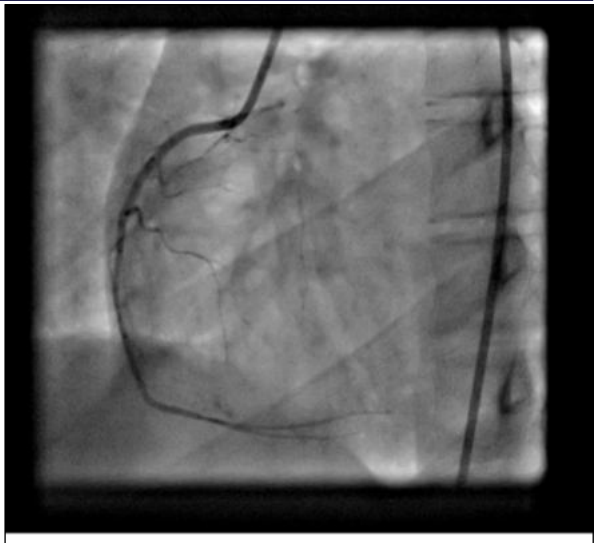

Figure: 2 Coronary Angiography shows normal Right Coronary Artery

Coronary angiography was done which revealed normal epicardial coronary arteries(Figure -2). The patient made a good in-hospital recovery and was discharged after 7 days.

Levels of Protein C, Protein S and Antithrombin III was repeated after 2 months. The repeat report revealed persistently low Antithrombin III level and normal Protein C, Protein S level.

DISCUSSION

Acute myocardial infarction is rare in young adults. Myocardialinfarction[MI] in young adults can be broadly dividedinto two groups: those with angiographically normal coronary arteries and those with coronaryartery disease of different etiology[1].The pathophysiologyof MI in the presenceof normal coronary arteries remains unclear.The possible mechanisms with normal coronary arteries include coronary vasospasm, in situ thrombosis or embolization with subsequent clot lysis and recanalization,minimal atherosclerosis, hypercoagulable states. There is strong evidence to suggest that smoking is an important predisposing risk factor for MI in the presence of normal coronary arteries[2]. Cocaine, marijuana, amphetamine abuse is associated with various cardiac complications including myocardial infarction by various mechanisms including coronaryvasospasm and hypercoagulability[3]. In our case there was no history of smoking or substance abuse.

Disorders of the coagulation system should be considered when any suspicion of idiopathic thrombosis or embolism appears likely,

including protein C, protein S and antithrombin III deficiency. Protein C, Protein S and Antithrombin III deficiency have been associated with a predisposition to venous thrombosis and thromboembolism. Arterial thrombosis is less frequent and may require other vascular risk factors.Decreased level of Antithrombin III activity was the risk factors in our patient. Possibly in-situ thrombus in coronary artery with its subsequent lysis with streptokinase may be responsible for the MI and subsequent normal CAG [Coronary Angiography] in this patient. Any possible source of thrombus with subsequent embolization into coronary artery were excluded by Doppler echocardiographic study.

Antithrombin III is the major plasma protease inhibitor of thrombin and other clotting factors in coagulation.Congenital AT III deficiency is an autosomal dominant disorder & leads to an increased risk of venous and arterial thrombosis in young adulthood.There is a case report of recurrent myocardial infarction in a young football player with antithrombin III deficiency[4]. Acquired AT III deficiency occurs in DIC[Disseminated Intravascular Coagulation], haemolytic uremic syndrome and venoocclusive disease(in patient undergoing bone marrow transplantation),sepsis,liver failure, nephrotic syndrome. Acquired causes of hypercoagulable states had been excluded in our case.

This case suggests that the deficiency of Antihhrombin III should be born in mind when a young patient presented with Acute myocardial infarction with normal coronary arteries in absence of conventional risk factors.

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