Case Report

ADOLESCENT MYOCARDIAL INFARCTION WITH NORMAL CORONARY ARTERY: A CASE REPORT

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Abstract

Acute myocardial infarction may occur in adolescent with angiographically normal or near normal coronary arteries but the pathophysiology remain unclear. This mechanism includes in situ thrombosis or embolization with subsequent clot lysis and recanalization and vascular endothelial dysfunction, per se or combined. An one adolescent male having acute myocardial infarction (inferior with posterior with right ventricular infarction) was reported with no anomalous coronary artery nor history of risk factors for coronary artery disease and no history of drug abuse. He was thrombolysed in addition to per oral clopidegrol (600mg) and aspirin (300 mg) and other conventional anti-ischemic treatment. Two hours later the patient was pain free and there was complete resolution of ST segment. Five days later he was scheduled for coronary angiography.

Key words: Adolescent myocardial infarction, coronary artery.

Introduction

Acute myocardial infarction is rare in childhood and adolescent. Although adults acquire coronary artery disease from the deposition of atheroma and plaque, which causes coronary artery spasm and thrombosis, children usually have either an acute inflammatory condition of the coronary arteries or an anomalous origin of the left coronary artery[1]. Acute myocardial infarction may occur when the coronary arteries are normal or near normal[2], it affects primarily younger persons. Myocardial infarction with normal coronary arteries is likely the result of multiple pathophysiologic mechanisms. These mechanisms include in situ thrombosis or embolization with subsequent clot lysis and recanalization and vascular endothelial dysfunction, per se or combined[1] coronary artery spasm. Intrauterine myocardial infarction also does occur, often in association with coronary artery stenosis[1].

According to the data from Nationwide Inpatient Sample (NIS) from 1998-2001, the incidence and outcome of adolescent myocardial infarction were 157 cases per year or 6.6 events per 1 million patient-year[3].Within the subset of adolescents with acute myocardial infarction, the incidence was higher in individuals aged 16 -18 years than in individuals aged 13 -15 years. The hospital survival for acute myocardial infarction in adolescent is excellent and mortality rate is 0.8%[3].

Case Report

A 13 year 6 months old boy from middle income family presented with history of typical chest pain for 8 hours associated with profuse sweating and vomiting. He does not have any symptoms of any systemic disease. He had otherwise led a healthy life and there was no history of medications prior to admission in the hospital. There was no history of smoking, alcohol, cocaine or other drug abuse and no family history of premature atherosclerotic disease, heart failure or sudden cardiac death. His body mass index was 19.38 kg/m^2.

He was found to be anxious; diaphoretic apart from that physical examination was unremarkable. He had sinus bradycardia (53 beat/min) and blood pressure was 80 over 60 mm Hg, ECG showed Q wave and 2 mm ST segment elevation in the inferior leads with 1 mm ST segment depression and T wave inversion in lead V_1 and V_2 with ST elevation in the V_4R -V_6R (Fig:1-5). 2D

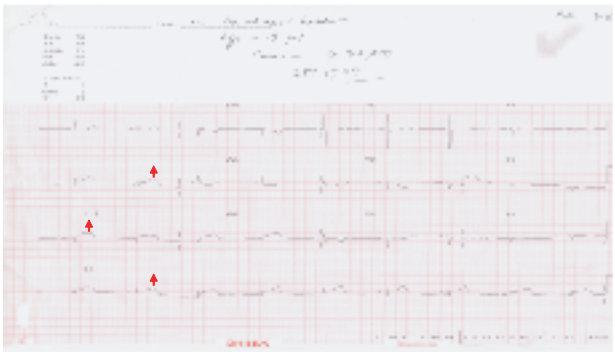

Fig-1: ST Elevation in the II, III, aVF; ST Depression; tall R in V_1 and T inversion in V_1 and V_2.

echocardiography revealed basal infero-posterior segment hypokinesia and global LVEF revealed 50%. Carotid artery duplex ultrasound was normal.

Initial laboratory findings included a white blood cell

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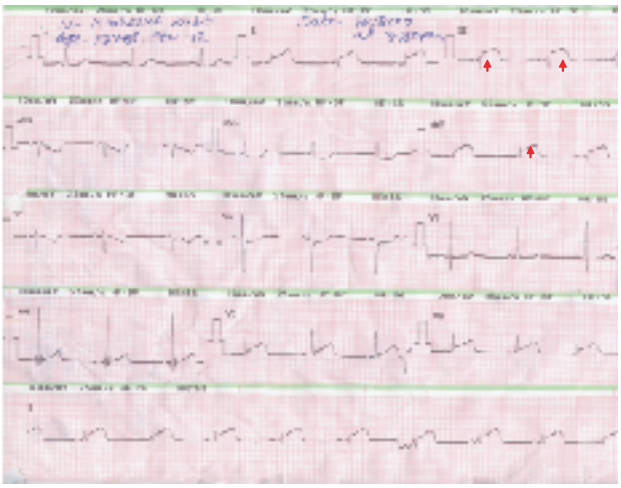

Fig -2: More Pronounced change in the same lead

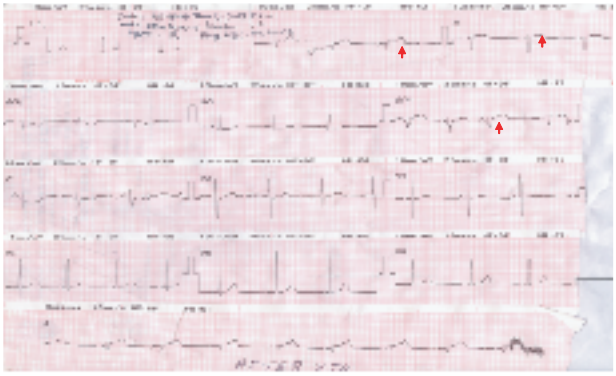

Fig-3: ST segment elevation coming down following thrombolysis

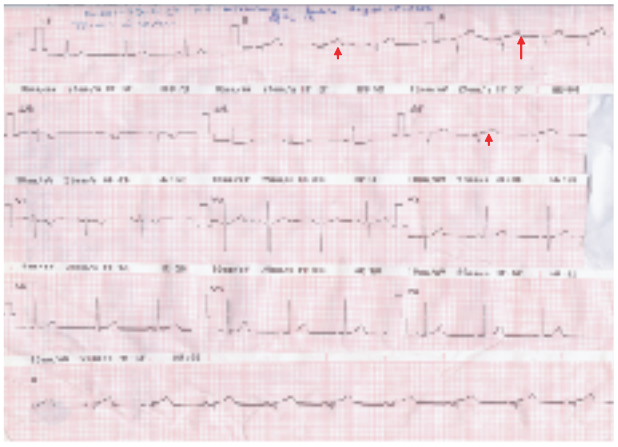

Fig -4: 12 lead ECG on the following day

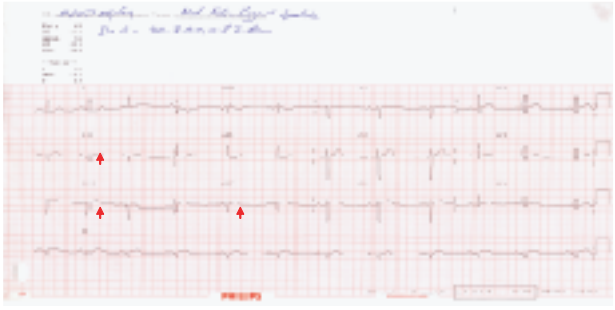

Fig-5: ECG. 2 days after thrombolysis.

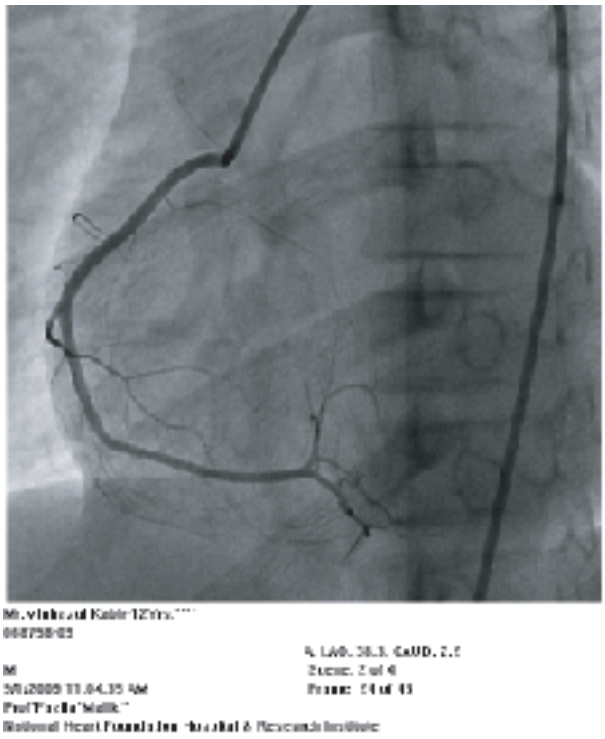

Fig-6: Right Coronary artery in LAO caudal view.

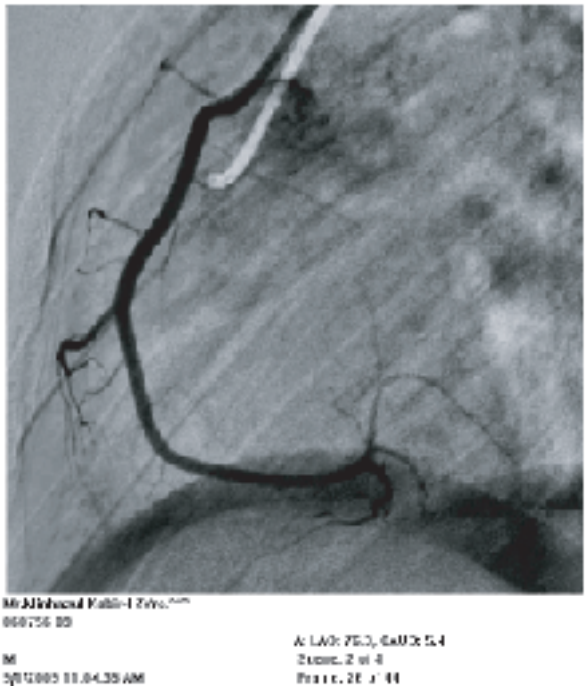

Fig -7: Right Coronary artery in LAO caudal (DSA) view.

count of 13000/mm^3, and normal hematocrit, platelet count, urea, creatinine, CRP, thyroid function tests, anti-phospholipid antibody C-ANCA, P-ANCA, lipid profile were within normal limit. RA test, HBsAg, ANA, Chlamydia antibody and H.pylori antibody were negative, VDRL and TPHA were non-reactive.

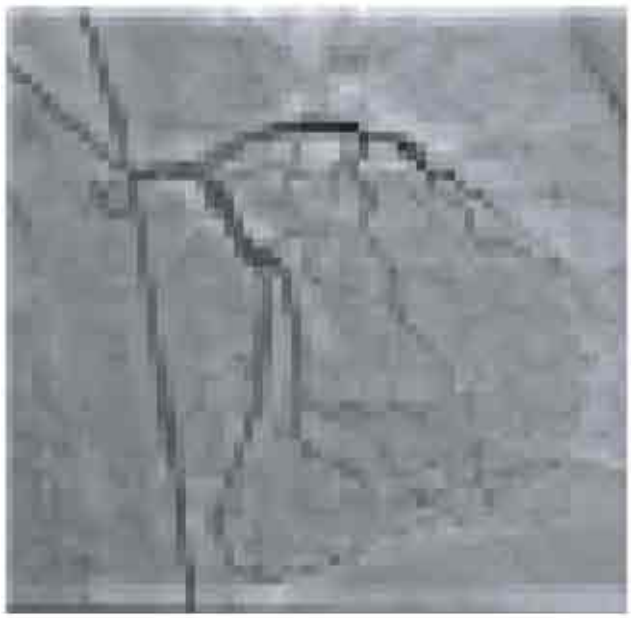

Fig-8: LCA:RAO(11.9)/Caudal (23.9) showing normal Luminography

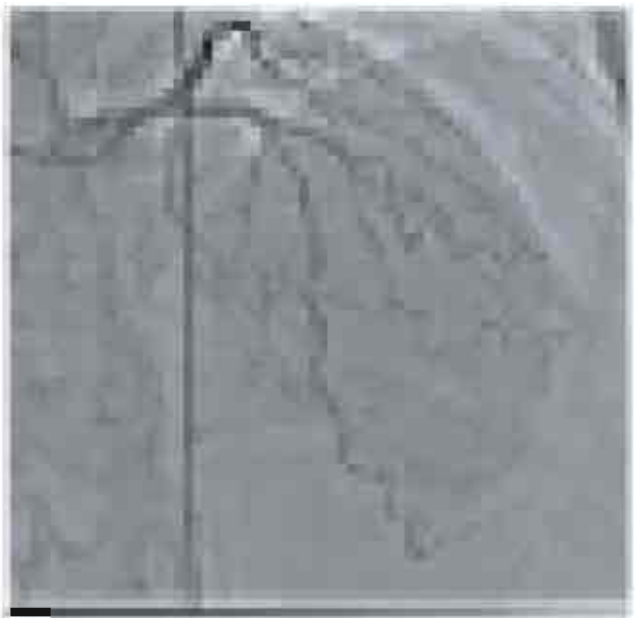

Fig-9: LCA .RAO(4.1)/Cranial(39.9) normal luminography

Further evaluation revealed normal Apo lipoprotein A-1, Apo lipoprotein B, protein S and protein C was >140(normal value 70-140), homocystein level was 12.55µmol/L (normal value 5-15 µmol/L).

He was thrombolysed in addition to per oral clopidegrol (600mg) and aspirin (300 mg) and other conventional anti-ischemic treatment. Two hours after the patient was pain free and there was complete resolution of ST segment. Five days later he was scheduled for coronary angiography.

Coronary angiography revealed normal left coronary artery system with very negligible irregularities in the mid segment of the right coronary artery supposed to be recanalyzed (Fig: 6 and 7).

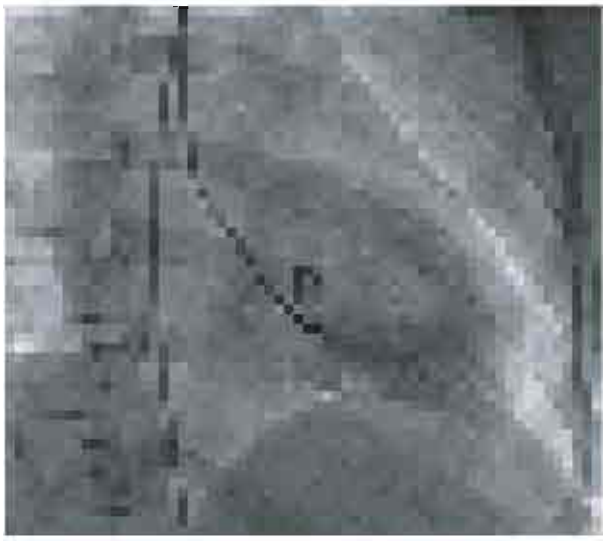

Fig-10: LV graphy (RAO) showing inferior wall hypokinesia

Discussion

Myocardial infarction is usually associated with obstructive coronary artery disease but it may be associated with normal coronary artery. The overall prevalence rate of myocardial infarction with normal coronary arteries is considered to be low, varying from 1% to 12% depending on the definition of "normal" coronary arteries, which usually includes no luminal irregularities(striet definition) or arteries with some degree of stenosis (in most cases, less than 30% stenosis)[4,5,6].

Although the majority of the cases of acute myocardial infarction is caused by atherosclerotic coronary artery disease, acute myocardial infarction can occur in people with coronary arteries that appear normal or nearly normal in an angiography[6]. The pathogenic mechanism of myocardial infarction in patients with normal arteries remain unknown. A single etiology for myocardial infarction with normal coronary arteries does not exist. The possible mechanisms causing myocardial infarction with normal coronary arteries are coronary vasospasm, coronary thrombosis, hypercoagulable states, coronary embolism and coronary trauma[5]. Although myocardial infarction with normal coronary arteries has been reported with many other conditions, a strong association with cigarette smoking has been demonstrated. It has been shown that there is increased platelet consumption in young smokers without clinical evidence of coronary artery disease[7]. Coronary artery spasm has been shown to cause myocardial infarction in patients with normal coronary arteries. Vasospasm can cause vascular endothelial injury leading to platelet aggregation and

coagulation system activation with resultant thrombosis and myocardial infarction[8,9]. Cocaine use is associated with various cardiac complications including myocardial infarction. Cocaine use results in acute myocardial infarction by various mechanisms including coronary vasospasm and hypercoagulability[10]. Amphetamine and marijuana use can result in myocardial infarction but the data are limited[11].

Disorders of the coagulation system should be considered when any suspicion of idiopathic thrombosis or embolism appears possible, including a protein C, protein S and antithrombin III deficiency[12]. Spontaneous coronary artery dissection is a rare cause of acute myocardial infarction in young women[13]. Myocardial bridging is usually asymptomatic, but has been related to acute myocardial infarction in patients as young as 15 years in the absence of risk factors for coronary artery disease and without evidence of coronary atherosclerosis. Coronary artery embolism secondary to infective endocarditis is a well known etiologic factor in the production of acute myocardial infarction in the presence of normal coronary arteries[14].

Coronary artery anomalies, such as origin of the left main coronary artery from the pulmonary artery or the anterior sinus of Valsalva, must be considered as a potential cause of myocardial infarction in infants and young children [15].

Coronary arteritis due to various systemic inflammatory diseases must also be considered as a cause of myocardial infarction (e.g. Kawasaki syndrome, acute rheumatic fever, polyarteritis nodosa, Takayasusu's arteritis, SLE, syphilis and rheumatoid arthritis etc[16].

In our case, coronary angiography revealed very marginal irregularities in the mid segment of the right coronary artery which is thought to be recanalization owing to resolution of the thrombus following thrombolysis. It could have been confirmed if we had done the intravascular ultrasonography (IVUS) to see the plaque and its morphology as it has been proved that acute plaque complications occur abundantly on minor stenosis more than severe stenosis[17].

However, patients with acute myocardial infarction and normal coronary arteries have usually been reported to have an excellent prognosis[2].

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