

Received: 15 July 2014 • Accepted: 07 August 2014



doi:10.15412/J.JBTW.01030903

# Acute Myocardial Infarction with Normal Coronary Arteries Following a Blunt Chest Trauma; a Case Report

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## ABSTRACT

Acute myocardial infarction secondary to blunt injury is rare. Even a mild trauma, such as a direct blow to the anterior chest, can cause myocardial infarction. Our article reported that a 28-year-old man who was in a non-penetrating trauma leading to damage of the left anterior descending coronary arteries causing acute myocardial infarction. Coronary angiography was normal and conservative management considered for this patient. We believe focal spasm or transient coronary thrombosis was the cause of myocardial infarction in this patient.

**Key words:** blunt chest trauma, traumatic coronary injury, acute myocardial infarction, normal coronary artery

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## 1. INTRODUCTION

Blunt trauma can cause various cardiac injuries ranging from non-significant arrhythmias to fatal condition such as myocardial rupture, acute myocardial infarction (AMI), cardiac contusion, ventricular septal defect and valve damages; AMI resulting from blunt trauma is rare but is an important cause of cardiac damage. Incidence of coronary artery injury is about 2%, extremely uncommon and it carries a high risk of mortality and morbidity (1). The most traumas causing myocardial infarction came from road traffic accident, followed by sporting accident. Early diagnosis is difficult due to the nonspecific clinical picture presented by patient (2). In this presentation, we aimed to report a case of myocardial infarction that occurred minutes after he received a blow on the chest while fighting.

## 2. METHODOLOGY

### 2.1. Case report

In August 2013, a 28-year-old male patient referred from another hospital to the heart center in Afshar hospital, Yazd for coronary angiography three days after a minor blunt chest trauma. He had been diagnosed with acute

myocardial infarction (AMI) and underwent medical treatment including Aspirin, Clopidogrel, unfractionated heparin because Emergency services such as primary coronary angioplasty and vascular interventions were not available there. The patient had no notable medical or surgical history such as hypertension, diabetes, dyslipidemia and illicit drug use. The patient was a non-smoker type and had not been taking drugs. The family history was negative for coronary artery disease, cardiomyopathy or thrombotic disease and there were no pathological findings related to other systems. On physical examinations within 24 hours of admission, Pulse rate was 110 per min, blood pressure was 130/70 mm of Hg, the respiratory rate was 22 per min and he was afebrile. Examination of the neck revealed normal carotid pulses with no bruits. The chest examination disclosed a normal apical impulse and normal heart sounds. Peripheral pulses were symmetric with normal amplitudes. There was no peripheral edema. The Pulse oxygen saturation was 92% while receiving 3 L/min oxygen therapy via nasal cannula. The creatinine kinase serum level was 8017 U/L, with an MB fraction of 755 U/L, and the troponin-I serum level was elevated.

### 3. RESULTS AND DISCUSSION

The 12-lead electrocardiogram taken at the time of admission showed an extensive Anteroseptal wall Myocardial Infarction, the ST depression and inverted T waves in I, II, III, aVF and the ST elevation in leads V1-V5 with Q waves from V1 to V4 (Figure 1).

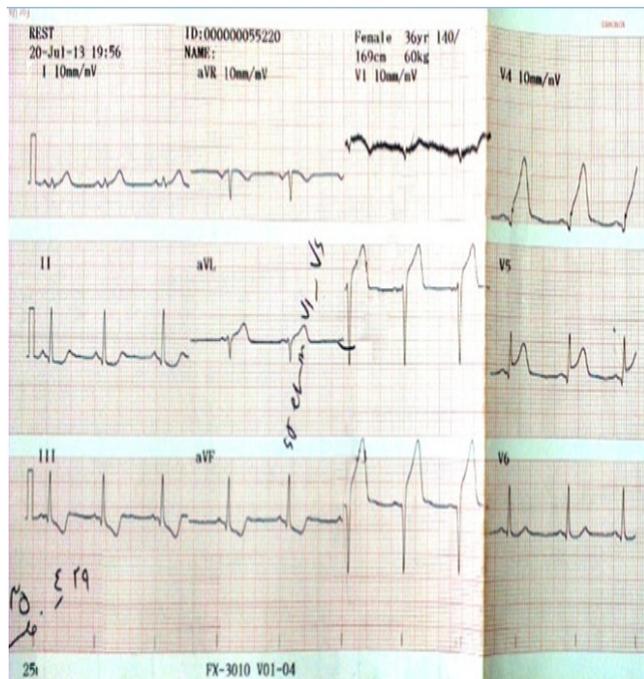


Figure 1. Electrocardiogram showing an acute extensive anteroseptal myocardial infarction

The Transthoracic echocardiogram performed revealing severe left ventricular (LV) systolic dysfunction (ejection fraction of 33% obtained by Simpson method), akinetic changes at the anterior, left ventricle anteroseptal and apical wall (Figure 2).

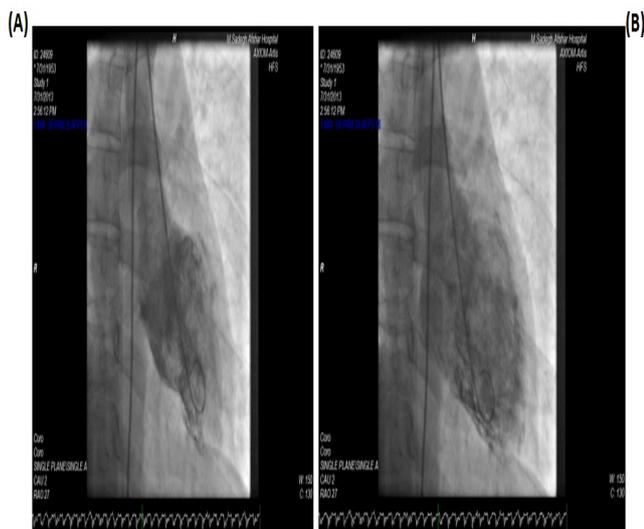


Figure 2. Left Ventriculogram. (A) Ventriculogram shows akinesis of anterior, anteroseptal and apical wall of the left ventricle in systole. (B) Ventriculogram in diastole. Coronary angiography showed normal coronary arteries

There was no evidence of an associated aortic disruption. In the present case, no valvular or pericardial abnormalities were seen by transthoracic echocardiography. In our

department, the patient was monitored in the coronary care unit (CCU), appropriate medications were continued, and the patient was submitted to coronary angiography. Cardiac catheterization showed normal coronary arteries (Figure 3).

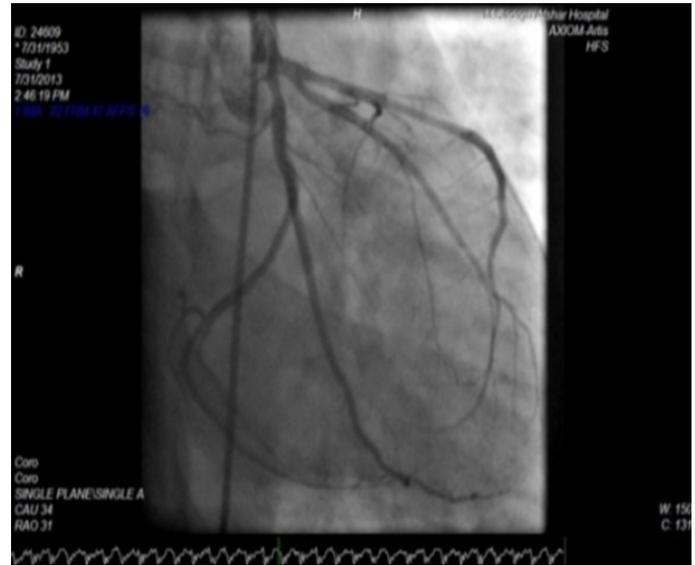


Figure 3. Cardiac catheterization

The Patient was stable during his admission and he discharged from the hospital under treatment with Aspirin, ACE-inhibitor, Beta-blocker, Clopidogrel treatment. It is also noted that patients with low ejection fraction and heart failure are more prone to blunt trauma which may damage the myocardium, cardiac valves and pericardium; which can lead to serious complications, such as: arrhythmias and sudden death. Our patients exposed to these effects. Blunt chest trauma leads to a wide range of injuries. Cardiac injury, secondary to chest trauma, is one of the most important injuries including cardiac arrhythmias, coronary fistula, coronary artery damage, ventricular aneurysm, cardiac rupture, and myocardial infarction (1-4). Although coronary artery atherosclerosis is the most common cause of AMI, blunt chest trauma is one of the non-atherosclerotic mechanisms leading to AMI in young adults (2). Although the pathogenesis is not clear, it seems that some mechanisms of coronary damage following blunt trauma could be due to prolonged focal coronary spasm, coronary embolism, dissection, thrombosis, an intimal tear and sub intimal hemorrhage (2, 4). The left anterior descending (LAD) coronary artery is the most commonly affected vessel (71.4%), followed by the right coronary artery (19%), left main artery (6.4%) and circumflex artery (3.2%) and the chest is the most common clinical symptom (4). Injury of the coronary vessel can lead to myocardial infarction (5) immediately or after some hours, in several cases, only minutes passed between the time of the blow to the chest and the onset of cardiac ischemic pain. This can be difficult to diagnose due to cardiac contusion with MI after a blunt trauma but 12-lead ECG and measurements of biochemical marker levels and echocardiography is helpful (2, 4, 5). *Coronary angiography should be considered*

early in such cases to distinguish between myocardial contusion and coronary occlusion (5). AMI from blunt chest trauma has been managed in several ways and has been controversial and differs according to the concomitant injuries (2, 4). Coronary artery bypass grafting, Percutaneous Coronary Intervention (PCI) and conservative management are considered as treatment options for this condition (1, 2, 4). Conservative management includes thrombolytic therapies reported for the patients with minor injuries. Bypass surgery may be safer than other therapies for managing Post traumatic myocardial infarction (1, 4). Beta blocker can reduce the myocardial oxygen demand is potential for arrhythmias but the beta blockers should be avoided in cocaine-induced myocardial infarction because the coronary spasm may worsen. The ACEI can be useful in reducing long term left ventricular dysfunction related to remodeling (5). Another recent study found acute myocardial infarction with angiographically normal coronary arteries which does not seem to be an uncommon finding in younger age groups; and cigarette smoking is highly prevalent in patients with AMI and normal coronary arteries at angiography (6, 7). The most frequent disorder in myocardial infarction with normal angiograms is a coronary artery disease, which predisposes the artery to vasospasm and thrombosis. Vasospasm can cause vascular endothelia injury leading to platelet aggregation and coagulation system activation with resultant thrombosis and myocardial infarction (8). Benacerraf *et al* and Vingent *et al* reported patients with myocardial infarction associated with probable coronary artery spasm (9, 10) Conti has documented that the most likely cause of ST-segment elevation in a patient with normal coronary arteries is spasm. Thrombotic occlusion could occur with prolonged spasm and a myocardial infarction could develop (3). The spontaneous lysis of the thrombus may explain the finding of a normal angiogram and so the delay between trauma and coronary arteriography may falsely increase the number of cases with "normal" coronary arteries in angiography (6, 9, 10). The twenty-eight-year-old patient reported no history of smoking or illicit drug use. The historical, electrocardiographic, enzymatic and ventriculographic evidence of AMI was associated with normal coronary arteries.

#### 4. CONCLUSION

In our patient we believe that the focal spasm or transient coronary thrombosis due to chest trauma were the causes of MI and hypothesized mechanisms for myocardial infarction without obstructive Coronary artery disease (CAD). Based on a normal angiography, it seems that spontaneous lysis of the thrombus occurred or spasm was resolved before coronary arteriography was carried out.

#### ACKNOWLEDGMENT

No mentioned acknowledgment by any authors.

#### AUTHORS CONTRIBUTION

This work was carried out in collaboration between all authors.

#### CONFLICT OF INTEREST

The authors declared no potential conflicts of interests with respect to the authorship and/or publication of this article.

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