Acute Myocardial Infarction after Blunt Trauma to the Chest

Case Report and Review

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Abstract

Significant cardiac damage can occur as a result of blunt trauma to the chest. The common complications of such an event include sudden cardiac death, arrhythmias, cardiac contusion or rupture of vital cardiac structures such as the ventricular wall, septum, valves, papillary muscles or the chordae tendinae. Acute myocardial infarction is a rare complication and it can occur even after seemingly minor trauma.
A case is presented about a previously healthy 34-year-old man who experienced an AMI after mild chest trauma. The case presentation is followed by review of this topic. Current information on epidemiology, pathogenesis, diagnosis and treatment are outlined and specific recommendations for emergency physicians are made.

Introduction

Patients with trauma to the chest are commonly seen in emergency departments (EDs), and injuries may range from life threatening to trivial. Cardiac trauma is an often-overlooked cause of significant morbidity and mortality in these patients, and its incidence is as high as 15% (1). Close to one million cases a year of cardiac injury after blunt chest trauma are reported in the United States (2). The following injuries can occur: cardiac contusion, rupture of ventricular wall, septum, valves, papillary muscles or the chordae tendinae (1-3). Some patients exhibit non-lethal arrhythmias whereas others present in cardiac arrest from ventricular fibrillation caused by cardiac concussion (*commotio cordis*) (4-5). Even in patients with few or no visible signs of chest wall damage, 55% will have some myocardial dysfunction (6).
We report a case of acute myocardial infarction (AMI) after blunt chest trauma. The review will provide current update on epidemiology, pathogenesis and management of this entity.

Case Report

A 34-year-old previously healthy, physically fit male came to the ED of a major urban hospital experiencing severe squeezing retrosternal chest pain. He had been struck in
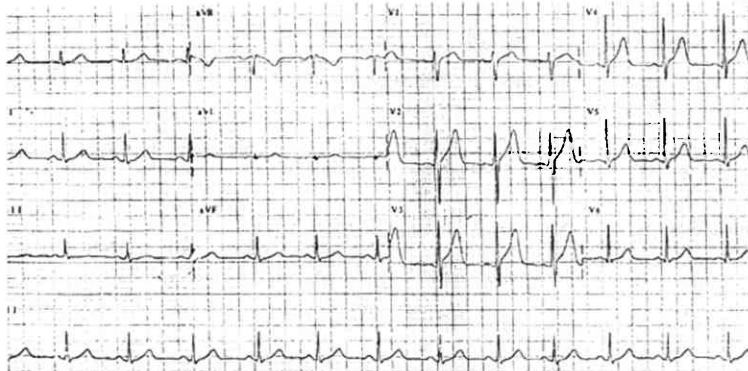
Figure 1. Initial ECG of the patient: peaked T waves in the precordial leads V2-V4.
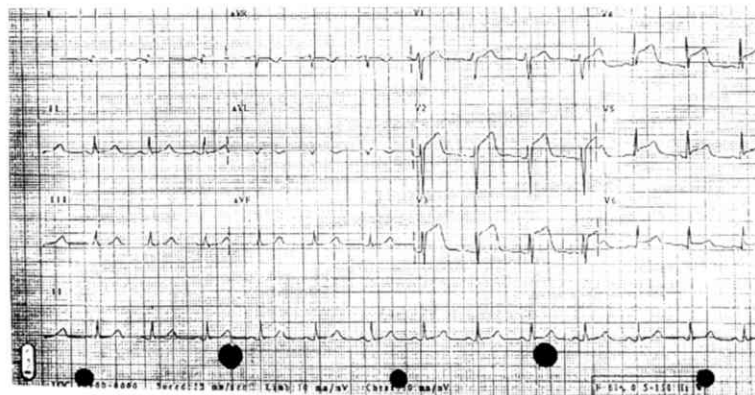

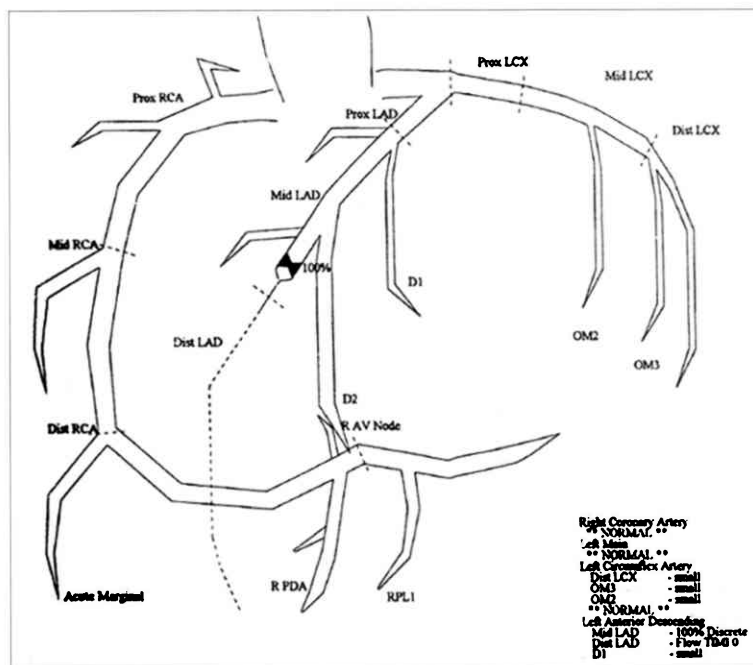*Figure 2. Second ECG of the same patient: ST segment elevations in V2-V6.*

Figure 3. Coronary angiography (CA): partially obstructing thrombus in proximal LAD with 100% occlusion of the distal third.

the chest with a hard-kicked ball while playing soccer earlier that day, but continued to play. The pain started about one hour after he stopped exercising and continued for four hours. The pattern and type of pain was highly suggestive of myocardial ischemia. Associated symptoms included nausea, vomiting and diaphoresis. The patient did not have any cardiac risk factors and he also denied taking any illicit drugs. The rest of his history and the entire physical examination were normal. A presumptive diagnosis of AMI was made. He was placed on monitors, an intravenous line was started, and he was administered supplemental oxygen, sublingual nitroglycerine and 160 mg of acetylsalicylic acid (ASA). A first electrocardiogram (ECG) showed peaked T waves in the precordial leads V2-V4 (**Figure 1**). The second one 30 minutes later showed ST segment elevation in leads V2-V6, consistent with anterolateral AMI (**Figure 2**). The cardiologist was consulted for advice on the best definitive treatment modality in the setting of AMI and chest trauma. He recommended thrombolytic agents (TA). During the administration of TA, the patient had an episode of ventricular fibrillation from which he was successfully resuscitated with a single 200 joules defibrillation. Shortly after the arrest, he was admitted to the coronary care unit in stable condition: he was alert, oriented and had normal vital signs.

Coronary angiography (CA) did not identify atherosclerotic plaques. In the left anterior descending artery (LAD) there was a thrombus extending the entire length of the proximal to middle portion of the vessel causing an 85% narrowing of the lumen but not affecting blood flow. The distal LAD was completely occluded by emboli from the thrombus (**Figure 3**). An area of akinesis was noted that involved the anterolateral and apical segments of the heart and the ejection fraction was 38%. The decision was made to leave the LAD thrombus alone and to treat the patient conservatively with heparin, coumadin, and clopidogrel.

The patient remained pain free and his ST segment elevations on the ECG resolved, although he did develop Q waves in the affected

precordial leads. The cardiac enzymes drawn in the ED were normal. Serial enzymes peaked at levels of LDH = 1573, CK = 2756, CK-MB = 197 (relative index of 13%), before returning to normal. Work up for hypercoagulable states was normal. Echocardiography performed confirmed slight dilatation of the left ventricle and mild akinesis involving the apical septum and the apical region of the left ventricle. The ejection fraction was 40%.

He was discharged in good condition after eleven days, to be followed up by the cardiologist and the cardiac rehabilitation program. Discharge medications were: ECASA, metoprolol 100 mg bid, enalapril 7.5 mg bid, clopidogrel 75mg qd and coumadin 5mg qd.

Months later, he developed further chest pain. Exercise stress test at that time was normal at eleven minutes of the Bruce protocol. He underwent CA which showed patent coronary arteries, including the previously occluded distal LAD. Compared to earlier his ejection fraction had improved to 50%.

Methodology

Two electronic database searches using PubMed, EMBASE and MEDLINE were carried out independently by two of the authors in June 2000 and in April 2002. Additionally the Cochrane Library data was searched in 2002. The Mesh terms used were: myocardial infarction, chest injury. Other search words included AMI, blunt chest trauma, *commotio cordis*, emergency medicine. Eighty-seven articles were identified in the world literature between January 1, 1980 and April 1, 2002. This list contained case reports, case series and review articles. Seventy-two articles written in English, French, Italian, and Spanish were selected. Additionally, a Danish case report was used for the epidemiology section. Articles in English were assessed independently by two of the authors. One author reviewed all the others. Article selection criteria included relevance for current practice in areas of epidemiology, pathogenesis, diagnostic and therapeutic interventions. Agreement between both individuals was necessary for inclusion into the current review. The final references selected for this review, except a few key articles, were published after 1992.

Discussion

Epidemiology:

AMI after blunt trauma to the chest is uncommon. Fifty-two cases were reported by 1991 (7). Since then another twenty-two cases were recorded worldwide, including the two most recent ones in November 2001 (8-9). Motor vehicle collisions (MVC's) account for more than half of all the reported cases. The remainder occurred in falls and sporting events. The majority of the sports-related incidents involved the patient being struck in the chest with some sort of ball or other high-speed projectile. Soccer ball injuries were common (8,10,11).

Pathogenesis:

Risk factors for trauma related AMI include: atherosclerotic coronary artery plaques, hypercoagulable states and connective tissue disorders such as Marfan Syndrome, Ehlers-Danlos Syndrome, or pseudoxanthoma elasticum (12).

In trauma, the myocardium is primarily at risk for damage from direct contusions and less commonly from interruption in coronary blood supply (1). Myocardial injury due to actual disruption of the coronary circulation in the setting of trauma is rare and, in the majority of cases, it affects the LAD. Proximity of the LAD to the anterior chest wall makes it the most susceptible vessel to be injured from blunt trauma to the chest (12-14). The right coronary artery (RCA) is second (2,13,15,16). Most cases occur in

younger, active people who do not have clinical coronary artery disease (CAD). Nevertheless, atheromatous changes can begin early in life and the plaques do not need to be of a clinically significant size to disrupt the coronary blood flow. A small region of a plaque breaks open when it is subjected to shearing forces or blunt trauma. In healthy hearts the natural elasticity of arterial tissue allows it to temper without any adverse effect the mechanical forces liberated by a blow over the sternum. An atherosclerotic artery is more like a rigid tube as it loses its elastic properties. Mechanical energy applied to this type of vessel causes deformity and rupture of the arterial wall and plaque followed by thrombus formation and occlusion of the coronary vessel (10).

Patients without pre-existing atherosclerosis can develop a traumatic lesion of the intima leading to dissection and subsequent thrombosis (12,14).

Hypercoagulable conditions make individuals more prone to developing thrombi, and patients with connective tissue disorders are more likely to dissect the coronary artery and form an aneurysm (12).

Pathogenesis in all other patients involves any of the following: rupture of the vessel itself, thrombosis from direct trauma, vasospasm, dislodgement and embolization of fibrous plaques, or pseudoaneurysm formation after dissection of the intima (17).

Diagnosis:

The diagnosis of AMI is simple when symptoms are consistent with typical cardiac ischemic pain and investigations support this conclusion. In other patients the diagnosis is challenging as other system injuries are more prominent and these may distract the physician from the innocuous sounding chest pain. Some patients may ignore the chest discomfort because of major pain elsewhere. Others will develop chest pain hours to days after the trauma and the connection between the injury and the chest pain is ignored (10,18,19). Sometimes it is not until the patient experiences hemodynamic compromise, or the pain persists despite treatment for other presumed causes, that myocardial ischemia is considered (13).

The mechanism of injury must be ascertained; specifically the type of trauma (MVC, fall, blow by blunt object, etc) and an estimate of the magnitude of impact. The velocity, mass and type of object are important factors. Description of the patient's chest pain, associated symptoms and review of previous atherosclerotic disease manifestations may suggest myocardial ischemia.

Assessment of risk factors for CAD including the use of illicit drugs is mandatory. On physical exam, the clinician will look for injuries that could account for the patient's chest pain. These would include: fractures or dislocations of the ribs or sternum, contusions, abrasions, or lacerations of the chest wall. Pulmonary contusions, pneumothoraces or hemothoraces should also be ruled out. Physical examination cannot readily rule out other causes of posttraumatic chest pain such as injuries to the cardiac structures, esophagus, sub-diaphragmatic intra-abdominal structures.

Initial investigations for patients with post traumatic chest pain include an ECG and a chest X-ray (CXR). Both are cost-effective tools that can easily be done in any trauma situation. The value of the CXR is to rule out other pathology. Serial ECGs can identify the myocardial origin of the pain. If the coronary obstruction is complete, an AMI pattern will be seen on ECG. In myocardial contusion, or incomplete coronary obstruction, the ECG will either be normal or will show nonspecific ST-T wave changes. Comparison with old tracings can clarify the temporal status of the ECG findings.

Patients with chest pain typical for AMI and a diagnostic ECG need urgent definitive treatment and cardiac enzymes results do not provide additional useful information in the immediate decision making process. Stable patients with atypical chest pain or those with non-diagnostic ECG findings should have serial cardiac enzymes drawn as the next diagnostic step in the ED. Significant injury to the myocardium from a contusion or AMI will result in elevated levels of serum CK - MB fraction and troponin.

In situations where the initial work up is either inconclusive or suggestive of AMI, echocardiography and/or CA is recommended (3,17,20-22). The echocardiogram can rule out a hemopericardium and it can evaluate the function of the cardiac valves and the myocardium. CA can assess myocardial function, delineate the sites of coronary artery narrowing or obstruction, outline atherosclerotic disease or vasospasm, or outline a dissection and pseudoaneurysm formation (1,3,15).

Management:

Priorities in management are dictated by the patient's clinical presentation. Immediate therapy must focus on resuscitation and hemodynamic stabilization. Resuscitative efforts have to be tailored to the presumed etiology of the potentially life threatening event. Once resuscitation is completed, more specific therapeutic and diagnostic measures are indicated. Suspicion of AMI in stable patients with chest pain after trauma warrants appropriate monitoring and targeted symptomatic treatment. Once AMI has been confirmed, early definitive management must be considered. The definitive therapeutic options available are: TA, percutaneous transluminal coronary angioplasty (PTCA) or urgent coronary artery bypass grafting (CABG) (15).

Significant trauma is generally a contraindication for TA therapy because of concerns regarding increased bleeding. The majority of patients with blunt trauma to the chest should not receive TAs, and it is recommended that decision for their use should be made in consultation with a cardiologist. Clear guidelines on the use of TA in this setting are currently not available. Consideration for their use should includes situations where the trauma to the chest is deemed mild and in cases where the other associated injuries do not preclude their use (10,23). Rescue angioplasty must be considered in all patients with failed thrombolysis.

PTCA is the preferred definitive treatment modality. It is the most commonly used invasive procedure and it yields very good to excellent results (15). It is less invasive than CABG. However, emergency CABG is in some situations the only alternative (13,24,25).

The current recommendation for those patients in whom angiography confirms dissection of the intima and subsequent thrombosis is to withhold definitive reperfusion treatment modalities. Patients have done well with anticoagulation and anti platelet treatment alone and some have successfully healed the lesion as demonstrated by repeat CA (3,14,17).

Conclusion

AMI secondary to trauma to the chest is a rare event, it often occurs in young individuals and it is responsible for significant morbidity and mortality. ED's must search for recent chest trauma in younger physically active patients with unexplained or atypical chest pain. We recommend that the assessment of "risk factors" for AMI in young healthy patients should include questions about recent blunt trauma to the chest. Initial investigations of patients with suspicious chest pain should include serial ECGs,

cardiac enzymes and a CXR. Consultation with a cardiologist is recommended in equivocal cases. Stable patients with unclear diagnosis may undergo a screening echocardiogram. Patients with definitive diagnosis of AMI require urgent angiography. The definitive treatment modality of choice is PTCA. Consideration for using thrombolytics in stable patients should be made in consultation with a cardiologist.

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