Acute myocardial infarction (AMI) is a rare, potentially fatal and often unrecognized complication of blunt thoracic trauma (BTT). Heart injury is diagnosed in less than 10% of all cases of blunt thoracic trauma. Medical literature is scarce in publications about AMI caused by BTT (AMI-BTT). There is no specific protocol on this issue. We describe two cases of AMI-BTT in young patients who were previously healthy and with no risk factors for coronary artery disease. The diagnostic tools and therapeutic approach are discussed based on a literature review. We conclude that the emergency physician should be alert for the possibility of AMI in victims of BTT, regardless of the intensity of the trauma.

Acute myocardial infarction (AMI) is described as a rare complication of blunt thoracic trauma (BTT), although literature reviews do not have accurate data about the true incidence of this condition. Early diagnosis is difficult due to the nonspecific post-trauma clinical picture presented by patients. BTT may cause damage to the myocardium, cardiac valves, coronary arteries and pericardium, leading to serious complications such as arrhythmias and sudden death. In promoting acceleration, deceleration or direct compression of the chest, the trauma can cause acute myocardial infarction through the following proposed mechanisms: dissection of coronary arteries, coronary thrombosis, vasospasm and rupture of atherosclerotic plaque. We present two case reports of patients who evolved with AMI after a BTT, as well as a review of literature.

Case Reports

Case 1 - CRVA, male, 31 years old, previously healthy and with no risk factors for coronary artery disease. Patient was a victim of BTT (he was hit in the chest by a ball during a soccer game. He evolved with intense and persistent epigastric pain irradiating to the posterior chest region. He was seen at the emergency room in his town about 24 hours after the trauma. Patient remained under clinical observation with persistent pain despite the administration of analgesics. Routine physical and laboratory examination were within the normal limits. An exploratory videolaparoscopy was performed to clarify the diagnosis. ST segment elevation was seen during anesthesia monitoring and this finding was confirmed in the ECG. Cardiac enzymes were elevated (troponin T). The echocardiogram showed moderate to severe systolic dysfunction (ejection fraction of 36%), pseudonormal diastolic function, dilated chambers, akinesia of anterior wall and apical aneurism. Drug treatment for acute coronary insufficiency was started, and the patient was transferred to a Coronary Unit of a hospital in the capital city of Belo Horizonte. Coronary angiography showed right coronary and circumflex arteries free of lesions, anterior descending artery with extensive thrombosis in its middle third, and left ventricle with anteroapical akinesia (Figs. 1 and 2). Clinical treatment was maintained with angiotensin-converting enzyme (ACE) inhibitor, beta-blocker, spironolactone, acetylsalicylic acid, clopidogrel and anticoagulation with low molecular weight heparin (LMWH) that was limited to the hospitalization period. The patient evolved to Killip II during hospitalization, responding to clinical treatment and was discharged from hospital with compensated heart failure of ischemic etiology secondary to coronary insufficiency.

Key words
Myocardial infarction, trauma, chest pain.
to AMI, and was followed up at the outpatient clinic. Five months later, the patient underwent coronary angiography which showed anterior descending artery with a normal flow despite unchanged anteroapical akinesia.

**Case Report**

**Case 2**: WM, man, 44 years old, previously healthy and with no risk factors for coronary artery disease; the patient was involved in a road accident and suffered a tibia fracture and BTT caused by the safety belt. Four days later he was transferred to the hospital in this capital for an elective surgery of tibia fracture correction. Upon the preoperative evaluation, patient presented with chest pain and dyspnea on minimum exertion. The electrocardiogram showed electrical inactivity and ST segment elevation in the anterior wall and inverted T wave in the inferior wall. The echocardiogram evidenced systolic dysfunction (ejection fraction of 33%) and anteroapical akinesia. Specific clinical treatment was initiated. Coronary angiography evidenced an image suggestive of a pseudoaneurysm, since it was a case related to BTT. However, an aneurysm of the middle third of the anterior descending artery with anteroapical akinesia could not be ruled out (Figs. 3 and 4). Patient evolved with acute pulmonary edema compensated with intensive clinical treatment. Patient then underwent angioplasty and placement of a tubular stent in the anterior descending artery. The partial clinical follow-up (control) after two months confirmed dilated cardiomyopathy and compensated heart failure of ischemic etiology secondary to AMI.

**Discussion**

Acute myocardial infarction secondary to a blunt thoracic trauma is rarely described as a complication in cardiac injuries related to trauma. This issue is rarely approached in systematic studies and it is more frequently found as case reports in literature revised; therefore, its prevalence and the incidence of associated abnormalities in ancillary examinations may be underestimated. Although coronary artery atherosclerosis is the most common cause of AMI, 20% of acute myocardial infarctions in young adults have a non-atherosclerotic etiology such as coronary artery embolism, hypercoagulation status, congenital coronary abnormalities, dissection of coronary arteries, coronary artery spasms (including the use of cocaine), vasculitis and mediastinal irradiation. BTT is another possible underlying mechanism of acute myocardial infarction in young patients. Clinically significant cardiac injury occurs in approximately 5 to 20% of patients with non-penetrating thoracic trauma, and up to 76% in severe cases of BTT.

Potential mechanisms of non-penetrating cardiac injury include fast acceleration or deceleration, direct chest trauma, heart compression between the sternum and thoracic segment of the spine, and fast increase of intra-aortic pressure due to abdominal or lower limb compression. The mechanisms that contribute to myocardial infarction may include intimal injury, subintima hemorrhage, intraluminal thrombosis and spasm.

Any coronary artery may become involved, although the anterior descending artery is the most commonly cited artery in case reports followed by the right coronary and circumflex arteries.

Coronary artery injury is more frequently diagnosed in patients under 45 years of age (case 2) victims of road accidents. Minor traumas have rarely been reported as the cause of coronary artery injury (case 1).

Early diagnosis is usually difficult because of low frequency, inespecific clinical picture and the level of suspicion by the assistant physician. Chest or abdominal pain following the trauma can be in most instances attributed to a contusion of bones and soft tissues, which can mask the pain of cardiac origin.

The high level of clinical suspicion, presence of the fourth heart sound and appropriate diagnostic tests must be considered in patients who suffer a BTT. Electrocardiogram must be performed in all suspected cases. Measurement of
cardiac enzymes, chest X-ray and echocardiogram can help in the diagnosis of cardiac trauma. The electrocardiogram (ECG) may present inespecific abnormalities associated with BTT in up to 70% of cases, with abnormal ST segment and T wave associated with sinus tachycardia being the most frequent electrocardiographic findings, in addition to intrequent reports of conducion disorders and arrhythmias. In both cases mentioned above, the ECG was the starting point for diagnosis before clinical suspicion.

Abnormal levels of the cardiac enzyme CKMB [creatin kinase and its MB isoenzyme] in BTT with no coronary artery injury are not related with prediction of complications or long-term prognosis. The increase of this enzyme can not be considered specific for diagnosis of infarction in patients with trauma, due to its presence in the skeletal muscle and in other organs. Serum measurements of troponins T or I help in the diagnosis of post-BTT AMI for having higher specificity; they can also be associated with prediction of cardiac complications related to BTT, especially when associated with an abnormal ECG.

The echocardiogram can show segmental deficits and it must be requested in suspected cases of cardiac trauma. It is also useful in the differential diagnosis of pericardial effusion and valve injury. However, the segmental deficits found can be secondary to myocardial contusion (directly in the muscle) or to acute myocardial infarction.

In cases of suspected diagnosis of acute myocardial infarction, coronary angiography must be performed for confirmation of the diagnosis and prompt initiation of specific treatment involving the reperfusion of the affected artery.

We consider that the two cases presented here are very likely to have a cause-effect relationship between trauma and infarction. However, we can not rule out the hypothesis of the solely temporal association among the events or the presence of coronary injuries previous to the trauma (such as the case of an aneurysm, for example).

We conclude that the clinical examination with high level of suspicion and an electrocardiogram in all cases of possible cardiac trauma must be part of the initial medical care of patients who have suffered a BTT. Acute myocardial infarction must early be considered in the differential diagnosis of patients who are victims of BTT, regardless of the intensity of trauma.

Potential Conflict of Interest
No potential conflict of interest relevant to this article was reported.

References