Acute Apical Myocardial Infarction After Blunt Chest Trauma Incurred During a Basketball Game

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Acute myocardial infarction resulting from blunt chest trauma is an infrequent but potentially lethal complication of severe trauma such as that associated with motor vehicle accidents. The occurrence of acute myocardial infarction after minor blunt chest trauma incurred during sports is quite rare. A literature search found only three reported cases between 1980 and 1999.[1,2] Family physicians are often involved in treating sports injuries in an office practice or the emergency department or as team physicians. This entity merits a place in the differential diagnoses whenever examining a patient with blunt anterior chest trauma.

Case Report

A 30-year-old man came to the emergency department complaining of severe substernal chest pain with associated radiation into both arms, diaphoresis, and shortness of breath. He had been playing basketball 2 hours earlier and sustained a blow to the anterior chest by the elbow of another player. The blow "knocked the wind" out of the patient, but he continued to play for a short while. The pain began substernally and worsened with movement. When the pain began to radiate to his left arm, he quit the game, went home, and took a nonsteroidal antiinflammatory medication, which did not relieve the pain. The pain continued to increase, and he became short of breath, prompting him to seek medical attention.

The patient had no notable medical or surgical history. His family history was remarkable only for his father having had a myocardial infarction at the age of 51 years. The patient had a 15-pack-year smoking history and did not drink alcohol. He was married and a member of the armed forces. Current medications were pseudoephedrine and naproxen. Review of systems was negative.

When examined, his temperature was 97.0°F, pulse 75 beats per minute, respiratory rate 22/min, blood pressure 145/77 mm Hg, and oxygen saturation 99% on 2 L of oxygen via nasal cannula. In general, he was an athletic-appearing man who looked uncomfortable, apprehensive, and pale. He had no stigmata of collagen vascular disease. The only findings during the remainder of his physical examination were tenderness and erythema at his mid sternum at the site of injury. There were no physical findings suggestive of a sternum fracture, rib fracture, or pulmonary injury.

An immediate electrocardiogram (ECG) (Figure 1) showed 1-mm ST elevation in leads II,III, aVF, V_3, and V_4 indicative of anterior and inferior myocardial injury. One 0.4-mg sublingual dose of nitroglycerine decreased his pain but also made him hypotensive. He became free of pain on administration of an intravenous nitroglycerine drip and 3 mg of morphine. Despite these measures his ECG readings remained unchanged. A comprehensive blood chemistry panel, cardiac enzyme levels, troponin I level, and a chest radiograph were all normal.

Because of the persistent injury pattern on the ECG, the patient underwent emergency cardiac catheterization, which showed an intimal dissection of the proximal left anterior descending coronary artery at the takeoff of the first septal perforator with an associated clot burden. The intimal flap and surrounding thrombus combined to form a 95% occlusion (Figure 2). The clot had embolized distally and occluded the transapical portion of the left anterior descending coronary artery, resulting in anterior and inferior ischemia. No serious atherosclerotic disease was seen in the remaining coronary arteries. The ventriculogram showed a preserved ejection fraction and apical hypokinesis. There was no evidence of an associated aortic dis-

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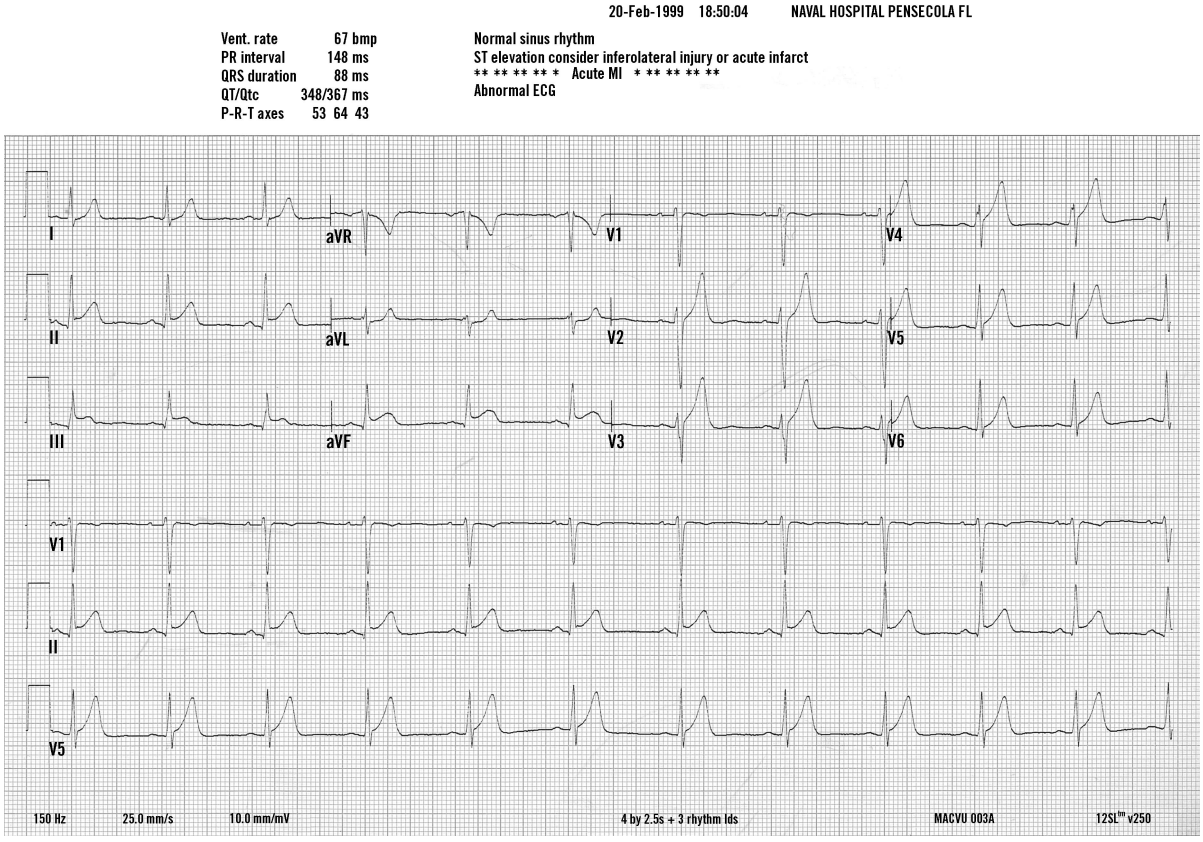

Figure 1. Electrocardiogram showing 1-mm ST elevation in leads II,III, aVF, V_3, and V_4 indicative of anterior and inferior myocardial injury.

ruption. Treatment was started with anticoagulant therapy and antiplatelet therapy, and the patient underwent balloon angioplasty. The stenosis was reduced from 95% to 50% (Figure 3), the patient became free of pain, and the ECG findings resolved.

The patient continued anticoagulation and antiplatelet therapy. A second catheterization 3 days later showed partial healing of the intimal tear with a 40% stenosis remaining in the left anterior descending coronary artery. The patient sustained an apical infarction with a peak creatinine kinase level of 1,590 U/L and an MB fractionation of 15.5%.

He was released from the hospital on warfarin, clopidogrel, and aspirin therapy to minimize the risk of further thrombus formation on the remaining intimal flap. His treatment plan included discontinuance of warfarin and clopidogrel after a follow-up angiogram showed complete healing of the intimal tear. He remained asymptomatic during the follow-up period.

Discussion

A literature search of the last 20 years found 17 cases of acute myocardial infarction resulting from blunt chest trauma.[3] Initial symptoms might not be clearcut, as chest pain and shortness of breath can result from a blow to the chest, an associated rib fracture, or a pulmonary injury. In all cases of acute myocardial infarction found in the literature, patients complained of symptoms suggesting cardiac chest pain and had ECG patterns consistent with acute myocardial infarction. Prompt ECG examination is therefore an excellent initial step.[4] Diffuse flattening of T waves and mild ST depression or elevation account for approximately 50% of ECG findings in cases of blunt chest trauma, with the remaining 50% being conduction abnormalities and dysrhythmias.[5,6] These changes most often represent trauma-induced pericarditis or cardiac contusion.[4] All reported cases of acute myocardial infarction showed an injury pattern consistent with a localized lesion representing specific coronary

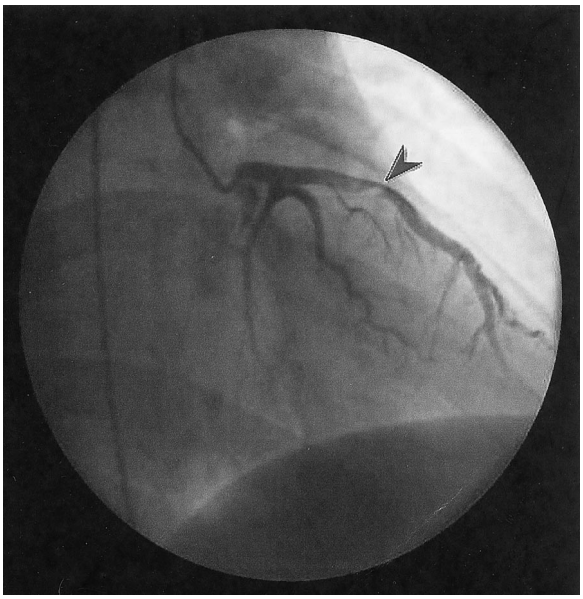

Figure 2. Angiogram showing 95% occlusion caused by intimal flap and surrounding thrombus.

artery involvement, whereas no reported cases of cardiac contusion did so.[1,2,6.]

Until recently the prevailing thought was that coronary artery occlusion occurred only when there was preexisting atheromatous disease.[7–9] Cases have now shown that coronary occlusion occurs in the absence of existing coronary artery plaques.[10,11] Animal studies have confirmed that blunt chest trauma can result in coronary occlusion in a plaque-free coronary artery by causing intimal tears with subsequent thrombus formation or spontaneous thrombus formation without an associated tear.[12]

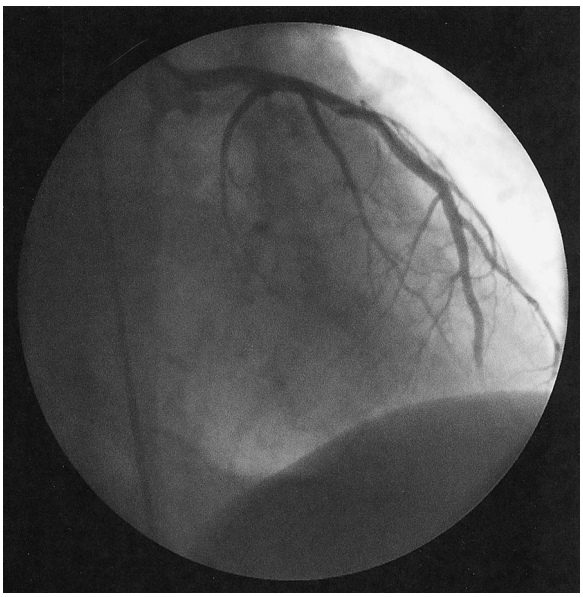

Figure 3. Angiogram showing stenosis reduced from 95% to 50% after anticoagulant therapy, antiplatelet therapy, and balloon angioplasty.

The most frequently injured vessels are the left anterior descending coronary artery (76%) and the right coronary artery (12%).[3] ECG changes indicating injury to areas supplied by a specific vessel or a known injury pattern should prompt rapid treatment and intervention for acute myocardial infarction. For diffuse or equivocal injury ECG findings, transesophageal echocardiography has been shown to be a quick and effective means by which to differentiate between cardiac contusion and coronary vessel tear.[13] Angiography remains the diagnostic reference standard.

Treatment has been controversial. Systemic anticoagulation and thrombolytic therapy are not always options in cases of severe trauma; however, with minor trauma these therapies can be less problematic. Traumatic intimal dissections generally heal within 6 months.[3,11] Treatment, therefore, should focus on initial stabilization, minimization of thrombus formation, and prevention of infarction. Although experience with the use of medical therapy alone compared with angioplasty is limited, one study showed that 30% of those treated with medical therapy alone later developed coronary artery aneurysmal disease.[3] As with any myocardial infarction, β-blockers can reduce the myocardial oxygen demand and potential for arrhythmias. Angiotensin-converting enzyme inhibitors can be useful in reducing long-term left ventricular dysfunction related to remodeling.

The possibility of coronary disruption should be considered in any patient complaining of chest pain as a result of trauma. Evaluation, including ECG examination, should take place promptly. Symptoms and patterns of injury shown on the ECG indicative of coronary occlusion should be treated as such. Transesophageal echocardiography is useful in equivocal cases to discern between coronary occlusion, contusion, or other myocardial damage. Immediate resuscitative measures are indicated, as well as empiric treatment for acute myocardial infarction with anticoagulation, antiplatelet therapy, and β-blockade, unless contraindications exist. Experience is limited with systemic thrombolytic therapy, but success has been shown with balloon

angioplasty and intraluminal thrombolysis; therefore, rapid cardiac catheterization is recommended.

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