Postcardiac Injury Syndrome Following Acute Myocardial Infarction Related to Blunt Chest Trauma

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Abstract

Blunt chest trauma can cause acute myocardial infarction, which may also be associated with pericarditis. However, such cases are rare. We herein report a case of a 57-year-old man suffering from acute myocardial infarction due to a blunt chest trauma and postcardiac injury syndrome after discharge with spontaneous resolution of a total coronary occlusion.

Key words: blunt chest trauma, acute myocardial infarction, postcardiac injury syndrome

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Introduction

Postcardiac injury syndrome (PCIS) following acute myocardial infarction (AMI) related to blunt chest trauma has not been previously reported. Previous autopsy studies have shown that the incidence of coronary artery injury secondary to blunt chest trauma is approximately 2% (1). PCIS can also develop after cardiac trauma, cardiac surgery, AMI, and some intravascular procedures (2). We herein report a rare case of PCIS that occurred after AMI related to blunt chest trauma and spontaneous recanalization of a totally occluded left anterior descending artery (LAD).

Case Report

A 57-year-old man with hypertension was transported to our hospital one hour after an iron shelf dropped onto his chest while he was working in a factory. The patient was healthy before the accident but complained of chest pain upon arrival to our hospital.

On physical examination, the patient’s vital signs were stable. Ecchymosis was present over the anterior chest wall. Chest X-rays were normal. An electrocardiogram (ECG) showed a sinus rhythm with a convex ST segment elevation on the whole leads without reciprocal change (Fig. 1A). Laboratory testing revealed positive cardiac markers with a creatine phosphokinase/creatine kinase-MB isoenzyme level of 825/187 U/L and troponin T level of 1.62 ng/mL.

Chest computed tomography showed a fracture of the distal portion of the sternal body with minimal retrosternal hemorrhagic fluid collection and no aortic dissection. The heart function was also evaluated; transthoracic echocardiography revealed normally sized cardiac chamber dimensions with preservation of the left ventricle systolic function (ejection fraction=56%), akinesia of the left ventricular apex, and no valvular dysfunction or pericardial effusion (Fig. 2A, B).

Coronary angiography revealed total occlusion of the distal portion of the LAD (Fig. 3A). The left circumflex coronary artery and right coronary artery were normal. We attempted to resolve the total occlusion of the LAD, however, the guidewire repeatedly entered the false lumen. Intravascular ultrasound (IVUS) showed the occlusion site of the LAD consisting of heterogeneous echogenicity. The lesions made it difficult to distinguish the blood and intima (Fig. 3b). When we injected the contrast dye into the distal segment of the LAD using a micro-catheter, we noticed that the lesion was the true lumen through the false lumen (Fig. 3B). Additionally, IVUS showed that the IVUS catheter was located in the false lumen (Fig. 3c, d). The patient remained hemodynamically stable at that time without chest pain or dyspnea. Therefore, we decided not to perform percutaneous coronary intervention and instead commenced conservative treatment.

Follow-up transthoracic echocardiography showed findings similar to those of the previous examination without pericar-
phosphokinase/creatine kinase-MB isoenzyme level of 270.98 U/L and troponin T level of 0.048 ng/mL. Additionally, laboratory testing revealed a normal eosinophil count (209.6×10⁹/L) and normal protein fraction. However, we did not check the auto-myosin Ab and auto-actin Ab levels. Transthoracic echocardiography showed finding similar to those of the previous examination, however, a moderate amount of pericardial effusion had newly developed (Fig. 2C). Nonsteroidal anti-inflammatory drugs (NSAIDs) were administered for 2 weeks. The patient steadily improved until the day of discharge. The pericardial effusion resolved by the time of the follow-up echocardiographic examination.

Six months after the accident, we performed follow-up coronary angiography. Coronary angiography revealed recanalization of the distal portion of the LAD that had previously appeared to be a total occlusion (Fig. 4). Ten months after the accident, the patient remained asymptomatic.

**Discussion**

There are no previously reported cases of PCIS after AMI due to blunt chest trauma. A variety of cardiac injuries resulting from blunt chest trauma have been reported, such as cardiac arrhythmia, valve injury, myocardial contusion, cardiac rupture, and myocardial infarction due to coronary artery injury. Traumatic coronary artery injury is a rare, life-threatening event (3). Traumatic coronary artery damage that can result in myocardial infarction after blunt trauma includes intimal tear, dissection, rupture of an existing plaque, spasm, vessel rupture and external compression from epicardial hematoma (4). Traumatic coronary artery injury frequently affects the following vessels in decreasing order of frequency: left anterior descending artery (76%), right coronary artery (12%), and left circumflex artery (6%) (5). Because affected patients are usually young and have single-vessel disease, the prognosis for most patients with AMI after blunt chest trauma is relatively favorable. The early diagnosis of AMI after blunt chest trauma is important because it may minimize the myocardial damage and reduce mortality. Traumatic coronary artery injury can be treated with coronary artery bypass grafting, angioplasty with stent placement, or conservative medical management (5-7). We attempted to perform angioplasty with stent placement, however, the guidewire repeatedly entered the false lumen. The culprit lesion in the LAD was distally located, and we speculated that a distal lesion would be less likely to affect the left ventricular systolic function. Therefore, we decided not to perform percutaneous coronary intervention.

PCIS is an inflammatory process involving the pleura and pericardium. It occurs secondary to cardiac injury and does not involve chamber perforation. The diagnosis of PCIS depends on characteristic clinical features such as a fever, chest pain, pleuropericarditis and pulmonary involvement. Abnormal laboratory findings include leukocytosis, elevated levels of inflammatory markers, and chest X-ray abnormalities (8). The patient reported herein had the typical clinical

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**Figure 1.** Electrocardiogram. A: An ECG showed a sinus rhythm with a convex ST segment elevation on the whole leads without reciprocal change at 1st admission. B: At the time to discharge, an ECG showed deep T wave inversion findings on precordial leads and limb leads. C: An ECG showed a sinus tachycardia with T-wave inversion in the precordial leads at the second admission.
**Figure 2.** Transthoracic echocardiography. A, B: Initial transthoracic echocardiography showed normally sized cardiac chamber dimensions with preservation of the left ventricle systolic function, akinesia of the left ventricular apex, and no pericardial effusion. End-diastolic (A). End-systolic (B). C: Follow-up transthoracic echocardiography showed findings similar to those of the previous examination, however, a moderate amount of pericardial effusion (arrow) had newly developed.

**Figure 3.** Coronary angiography. A: Coronary angiography revealed total occlusion of the distal segment of the LAD (a: normal segment of the LAD, b: total occlusion site of the LAD; heterogenous echogenicity was observed within the lumen. Moreover, the lesions made it difficult to distinguish the blood and intima). B: The more distal segment of the occlusion site was achieved using repetitive wiring from the false lumen to the true lumen. When we injected the contrast dye into the distal segment of the LAD using a micro-catheter, we noticed that the lesion was the true lumen through the false lumen (c, d: IVUS catheter was located in the false lumen).
features of PCIS. He exhibited pleural effusion, pericardial effusion, elevated levels of inflammatory markers, and typical symptoms of pericarditis after AMI related to blunt chest trauma. The pathophysiology of PCIS has not yet been elucidated. The most widely accepted hypothesis is that the development of an autoimmune response against heart antigens leads to generalized pericardial inflammation and pericarditis (2, 9). Treatment of PCIS includes NSAIDs, colchicine, steroids, and aspirin. We treated our patient with NSAIDs, and he steadily improved until the day of discharge. The amount of pericardial effusion also decreased. Moreover, follow-up coronary angiography revealed spontaneous recanalization of a totally occluded LAD. We believe that mild injuries of the coronary artery during the intervention, such as unrecognized perforation by a guidewire repeatedly entered into the false lumen, may have caused small bleeding into the pericardial space and triggered PCIS. Additionally, spontaneous recanalization of LAD may be achieved by the repetitive wiring from the false lumen to the true lumen.

In summary, AMI related to blunt chest trauma is rare. Additionally, the case of PCIS after AMI related to blunt chest trauma and spontaneous recanalization of the totally occluded coronary artery has not been previously reported. We herein reported a rare case of PCIS that occurred after AMI related to blunt chest trauma and the spontaneous resolution of total occlusion of LAD. Physicians should therefore pay careful attention to the possibility of the development of PCIS following AMI related to blunt chest trauma. An early diagnosis and management is critical in such cases.

The authors state that they have no Conflict of Interest (COI).

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