CASE REPORT

Importance of Carefulness in Accurate Diagnosis
Myocardial Injury Caused by Severe Blow

Satoshi Higuchi,¹ MD, Kenichi Matsushita,¹ MD, Yoshihiro Niina,¹ MD, Konomi Sakata,¹ MD, Toru Satoh,¹ MD and Hideaki Yoshino,¹ MD

Summary
Blunt chest trauma may lead to cardiac involvement such as myocardial contusion, coronary artery dissection, cardiac rupture, or myocardial infarction. Early detection and treatment of complications such as these are essential. We describe a case status post collision with an iron ball and discuss how to detect myocardial infarction. We emphasize the importance of careful interview, physical examination, and electrocardiogram even in seemingly healthy patients. A severe blow, such as that described, can impair coronary artery flow and may potentially cause myocardial infarction.

Key words: Blunt trauma, Coronary angiography, Myocardial infarction

Discussion
Our patient demonstrated myocardial infarction, complicated by blunt chest trauma. Careful interview and physical examination were key to accurate diagnosis. Some patients who suffer from blunt chest trauma may simultaneously develop myocardial infarction, although its incidence is low.

What is the cause of such adverse events? A previous review revealed causes such as thrombus formation due to intimal tear and dissection, focal spasm, vascular rupture, fissuring of atherosclerotic plaque, and external hematoma compression.1) The distribution of delayed enhancement on CMR was consistent with assumed occlusion of the middle left anterior descending (LAD) artery in the present case. Regarding the frequency of each affected coronary artery after blunt chest trauma, Christensen et al. reported

Differential diagnosis is one of the most important tasks a physician performs. Healthy subjects, working at construction sites, can also concurrently display disease with external trauma. Detailed interview and careful physical examination are essential for accurate diagnosis and appropriate treatment. We present a male patient with myocardial infarction, who initially presented with blunt chest trauma.

Case Report
A 69-year-old male with hypertension sustained chest trauma following a side collision with a 3-kg iron ball during a decommissioning operation. He developed a throbbing sensation at the trauma site and was admitted to our hospital.

On initial examination, we observed normal blood pressure, heart rate, respiratory rate, and body temperature (140/87 mmHg, 67 beats per minute, 16 breaths per minute, and 36.4°C, respectively). The first and second heart sounds had normal loudness, and a fourth heart sound was heard. Neither the third heart sound nor murmur was heard. The bruised trauma site, where the patient reported increased pain with breathing or when pushed with a finger, showed internal bleeding. No rib bone fractures were observed on chest X-ray. When asked about the pain, the patient reported a different, nonthrobbing, pain, describing it as chest tightness that concurrently appeared with the blow.

On electrocardiogram (ECG), elevated ST-segments in the precordial and high lateral leads were observed (Figure 1A). An echocardiogram revealed hypokinesis on the anteroseptal wall with no epicardial effusion. As shown in Figure 2, we found no stenosis or coronary dissection on emergent coronary angiography (CAG). Creatinine kinase (CK) was observed at 1,286 IU/L, Creatinine kinase-myocardial band (CK-MB) at 143 ng/mL, and troponin I at 40,425 pg/mL. We diagnosed the patient with myocardial infarction in addition to injury. The feeling of chest tightness disappeared the following day. The throbbing pain disappeared a week later. Cardiac magnetic resonance imaging (CMR) revealed delayed enhancement on the anteroseptal and anterior endocardial areas (Figure 3A), with no indication of hemorrhage (Figure 3B). An acetylcholine provocation test was negative. We prescribed imidapril to prevent possible lesion remodeling. A follow-up ECG revealed ST resolution (Figure 1B). The patient’s cardiac function remained unchanged after a year.

From the ¹Division of Cardiology, Department of Internal Medicine II, Kyorin University School of Medicine, Tokyo, Japan.
Address for correspondence: Kenichi Matsushita, MD, Division of Cardiology, Department of Internal Medicine II, Kyorin University School of Medicine, 6-20-2 Shinkawa, Mitaka, Tokyo, 181-8611, Japan. E-mail: kenichi-matsushita@umin.ac.jp
Received for publication June 21, 2017. Revised and accepted October 24, 2017.
Released in advance online on J-STAGE May 23, 2018.
doi: 10.1536/ihj.17-345
All rights reserved by the International Heart Journal Association.
Figure 1. (A) An electrocardiogram (ECG) disclosed ST elevation in the precordial and high lateral leads. (B) A follow-up ECG showed ST resolution.

Figure 2. Emergent coronary angiography showing no stenosis or dissection in the left (A) and right (B) coronary arteries.

Figure 3. (A) Delayed enhancement of cardiac magnetic resonance imaging was noted (arrow). (B) T1-weighted image showed no indication of hemorrhage.

that LAD was most frequently affected (71.4%), followed by the right coronary artery (19.0%), left main trunk (6.4%), and left circumflex artery (3.2%).\(^\text{31}\)

The hypothetical mechanisms of infarction included
the direct impact of contusion, external hematoma compression, temporary thrombus formation, or vasospasm. As CMR revealed no hematoma, and an emergent CAG showed no coronary artery dissection, the severe blow might not have directly affected the myocardium; external hematoma compression was less likely. We ruled out vasospasm through acetylcholine provocation test. Sinha et al. reported a coronary thrombus without dissection in a young woman, without atherosclerotic risk factors. A sudden change in the diameter of a tube reduces the velocity of fluid near the wall of the tube. Flow retarded at the surface has low kinetic energy and, therefore, cannot enter a high-pressure zone, consequently separating from the vessel wall, moving toward the inner flow. At the boundary layer separation, shear stress equals zero; therefore, this condition may induce hyperviscosity and increase rouleaux formation. Blunt trauma to vascular walls could, therefore, lead to thrombosis. In our patient, we did not perform anticoagulation therapy because the emergent CAG revealed no thrombus. Regarding anticoagulation therapy, patients should be individually managed, taking into consideration the benefits and risks associated with the therapy.

In conclusion, we reported a patient with myocardial infarction who initially presented with blunt chest trauma. Careful medical interview and physical examination can be important clues to hidden diseases even in seemingly healthy subjects.

Disclosures

Conflicts of interest: The authors have no conflicts of interest to declare.

References

1. Christensen MD, Nielsen PE, Sleight P. Prior blunt chest trauma may be a cause of single vessel coronary disease; hypothesis and review. Int J Cardiol 2006; 108: 1-5.