Clinical Case Report

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Myocardial infarction following a blunt chest trauma

A case report

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Abstract

Rationale: Blunt cardiac injury (BCI) is a common complication after blunt chest trauma, which can lead to mild arrhythmia, severe chamber or valvular rupture, or even death. Myocardial infarction following blunt chest trauma is a rare but fatal condition.

Patient concerns: A 38-year-old, previously healthy, man was admitted to our hospital with a complaint of dyspnea. He had a history of being hit in the chest by a high-speed screw while working in a factory 3 months before he was admitted to the hospital.

Diagnosis: After performing coronary angiography and echocardiography, he was finally diagnosed with myocardial infarction.

Interventions: He received optimized medications, including diuretics, β-blockers, and cardiac stimulants.

Outcomes: At the 4-year follow-up, the patient was diagnosed as having chronic heart failure with a reduced ejection fraction.

Lessons: Owing to the first doctor's lack of experience and knowledge with this case, the patient was misdiagnosed and treatment was delayed, which subsequently led to heart failure.

BCI can lead to myocardial infarction if patients are misdiagnosed and treatment is delayed. Thus, surgeons and physicians should consider cardiac complications in patients with chest trauma to reduce the incidence of its misdiagnosis.

Abbreviations: BCI = blunt cardiac injury, CAG = coronary angiography, ECG = electrocardiogram, IVUS = intravascular ultrasound, LAD = left anterior descending artery, LCX = left circumflex artery, RCA = right coronary artery.

Keywords: blunt trauma, cardiac injury, coronary angiography, myocardial infarction

1. Introduction

In the United States, trauma is the leading cause of death among the young population (1–44 years old).[1] Among trauma victims, cardiovascular injuries are the second most common cause of death after central nervous system injuries, with more than 900,000 cases of blunt cardiac injuries (BCIs) in the United States each year.[2] Causes of BCI include motor vehicle collisions, falling from a height, being struck on the chest by a ball, and even a blow on the chest by a fist.[3] Moreover, there are several cause of BCI that lead to death, including transmural rupture of 1 or more cardiac chambers, commotiocordis, valvular injury, coronary artery injury leading to acute myocardial infarction (AMI), and pericardial tamponade.[4] Coronary injury leading to AMI is a rare but life-threatening situation. Angina is usually easily masked by chest trauma, unfortunately leading to misdiagnosis and delayed treatment. A few reports have presented cases of myocardial infarction after blunt chest trauma, but only a few these cases were confirmed by coronary angiography (CAG). Further, no intravascular ultrasound (IVUS) examination was performed in these cases, and these cases were not followed up well.

We report a case of blunt chest trauma in a 38-year-old, previously healthy, man who got hit by a high-speed screw that led to the dissection of the left anterior descending (LAD) artery, as confirmed by CAG and IVUS.

2. Case report

A 38-year-old man was admitted to the hospital for blunt chest trauma. The patient was generally healthy and had no risk factors of coronary artery disease. He was hit in the chest by a high-speed screw that was of approximately 6-cm diameter while working in a factory. He flew approximately 4 m away because of the force and pressure of the high-speed screw. He lost consciousness at once, but regained consciousness several minutes later. He was then transferred to the emergency room of a local hospital immediately. His Glasgow Coma Scale score was 15. He complained of intense and persistent chest pain, tightness, and dyspnea. Chest computed tomography showed bilateral pleural effusion and sternal and rib fractures. Thus, a chest tube was inserted for several days. Then, he was discharged without

Editor: N/A.

Written informed consent was obtained from the patient.

The authors declare that they have no conflict of interest.

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Medicine (2019) 98:4(e14103)

Received: 16 August 2018 / Received in final form: 12 December 2018 / Accepted: 18 December 2018

http://dx.doi.org/10.1097/MD.0000000000014103

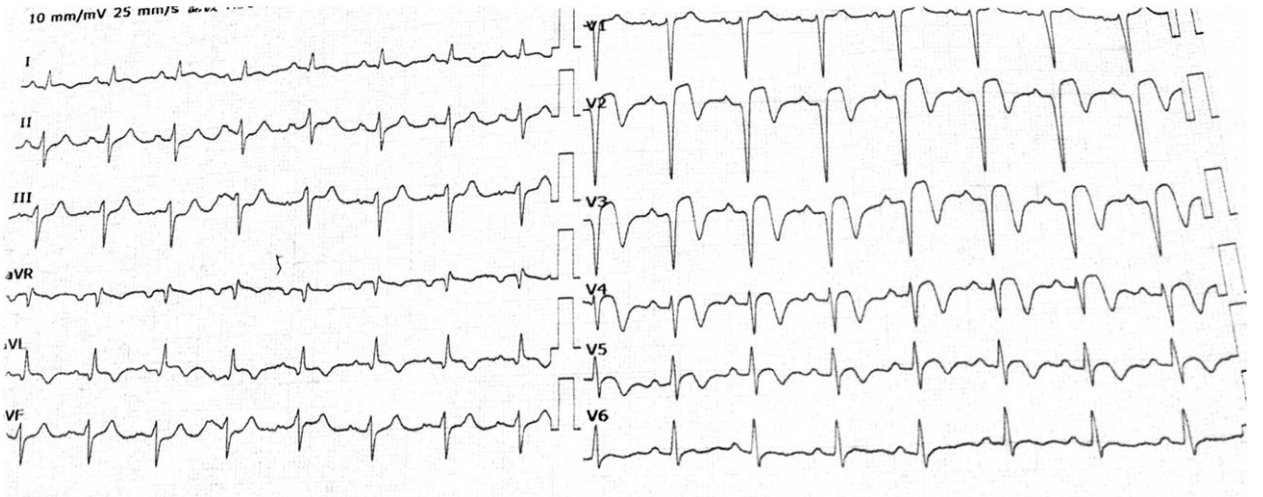

Figure 1. The patient's first electrocardiogram after the blunt trauma shows ST evaluation and T wave inversion in the I, avL, and V2-V5 leads and the left anterior branch block.

undergoing electrocardiogram (ECG) or other cardiovascular examinations.

Three months after hospital discharge, during a routine follow-up check-up, pleural effusion was still present with accompanying symptoms of chest tightness, shortness of breath after physical activities, and even dyspnea at night, which prompted his admission in our hospital. His vitals during admission were as follows: a regular heart rate of 98 beats/min, blood pressure of 110/70 mmHg, and respiratory rate of 20 breaths/min. Physical examination showed normal carotid and jugular venous pulsation. Bilateral thoracic respiratory movement and activity were normal, tactile language fibrillation of the right lung was abate, right lung percussion presented with dullness, and breath sounds were abate. There was no obvious rhonchus and moist rale during lung auscultation. There were no murmurs upon cardiac auscultation. Examinations of the abdomen and extremities revealed normal findings. The first ECG (Fig. 1) showed ST evaluation and T wave inversion in the I, avL, and V2-V5 leads and in the left anterior branch block. Troponin-I test was negative, and the NT-proBNP level was 706 pg/ml (normal range: 0–104 pg/ml), suggestive of an old anterior myocardial infarction. Transthoracic echocardiography showed a 26.4-mm left atrium, a 51.8-mm left ventricle, and a left ventricular (LV) ejection fraction (EF) of 32% (M mode). The anterior LV wall and interventricular septum showed thinning at 6.6 mm, with remarkably reduced movement echo.

When examining the patient's carotid, intracranial, and lower extremity arteries, no obvious atherosclerotic plaques were found. All findings supported the diagnosis of old myocardial infarction (OMI), rather than arteriosclerosis. However, the patient was hemodynamically stable. After undergoing diuretic therapy, the patient was administered β-blockers, statins, and cardiac stimulants, which relieved his discomfort. Coronary computed tomography angiography showed moderate stenosis in the proximal LAD artery. To confirm the stenosis and examine the condition of the coronary intimal, CAG was performed at 3 months after the chest trauma, which showed approximately 70% narrowing of the lumen in the proximal LAD (Fig. 2) and the coronary segment had a curved lesion. The left circumflex artery (LCX) and right coronary artery (RCA) were normal. CAG results confirmed the diagnosis of OMI, but due to the cost burden, the patient refused IVUS examination. The patient underwent conservative therapy and was discharged from the hospital several days later.

One month later, on December 9th,2014, he returned to the hospital for percutaneous coronary intervention (PCI) therapy. After undergoing drug therapy, his conditions had improved slightly. Physical examination and ECG revealed almost normal findings. Transthoracic echocardiography showed improvement in LV systolic function and EF at 45%. He had full antiplatelet therapy with aspirin and clopidogrel. Then, he underwent another CAG at 4 months after the chest trauma, which showed a slight improvement of stenosis. There was approximately 60% narrowing of the lumen (Fig. 3). IVUS was performed and revealed that the lesion severity was 55.9%, and the plaque of low echo appeared before thrombus formation after the subintimal tear. We completed the examination without further intervention because only 55.9% narrowing of the lumen was found and the patient had stable hemodynamics without evidence

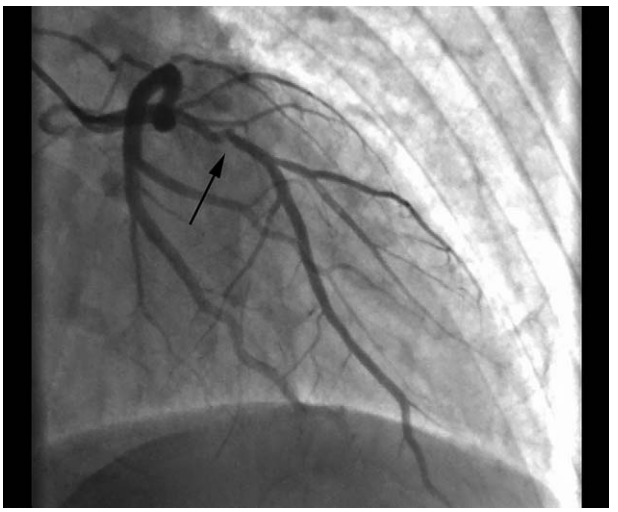

Figure 2. Coronary angiography image from the right anterior oblique shows a curved lesion with approximately 70% narrowing of the lumen in the proximal of left anterior descending coronary artery.

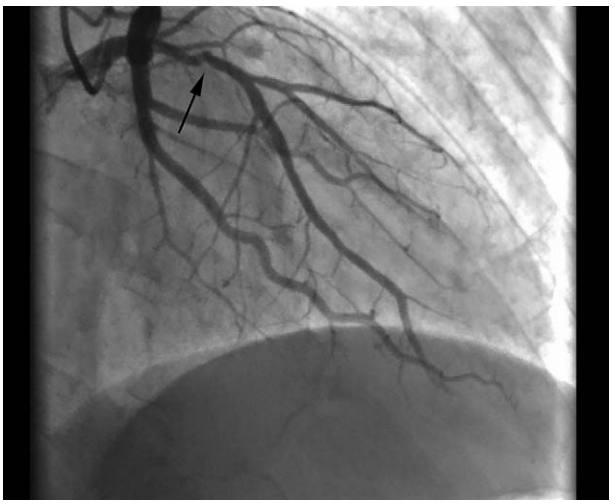

Figure 3. Coronary angiography image from the right anterior oblique shows that the curving of the lesion is slightly reduced compared to that in the its previous condition.

of constant myocardial ischemia. The patient was prescribed optimal medication, and his discomfort gradually alleviated. In July 17, 2018, 4 years after the trauma, follow-up evaluation revealed that the patient still experiences shortness of breath after working, but is relieved soon after taking a rest. Physical examination showed the following results: blood pressure of 114/80 mmHg, heart rate of 66 beats/min, absence of obvious abnormalities upon lung and heart auscultation; and absence of obvious edema in both lower extremities. Follow-up ECG revealed the following findings (Fig. 4): rS in V2-V4 leads, T wave inversion in the I, avL, and V2-V5 leads and the left anterior branch block. Transthoracic echocardiography (Fig. 5) showed left heart enlargement with 39-mm left atrium, 54.7-mm left ventricle, and LV EF of 41% (Simpson's). Thinning of the LV anterior wall and interventricular septum was also noted, with the thinnest part being 4.7 mm; movement had almost disappeared and the echo was enhanced. However, the carotid, intracranial, and lower extremity arteries were still normal. Moreover, we found that the medications prescribed to treat heart failure was not religiously taken after the discharge; thus, we newly prescribed 23.75-mg metoprolol, 2.5-mg benazepril, and 20-mg spironolactone to be taken once daily, and coenzyme to be taken every 10 hours.

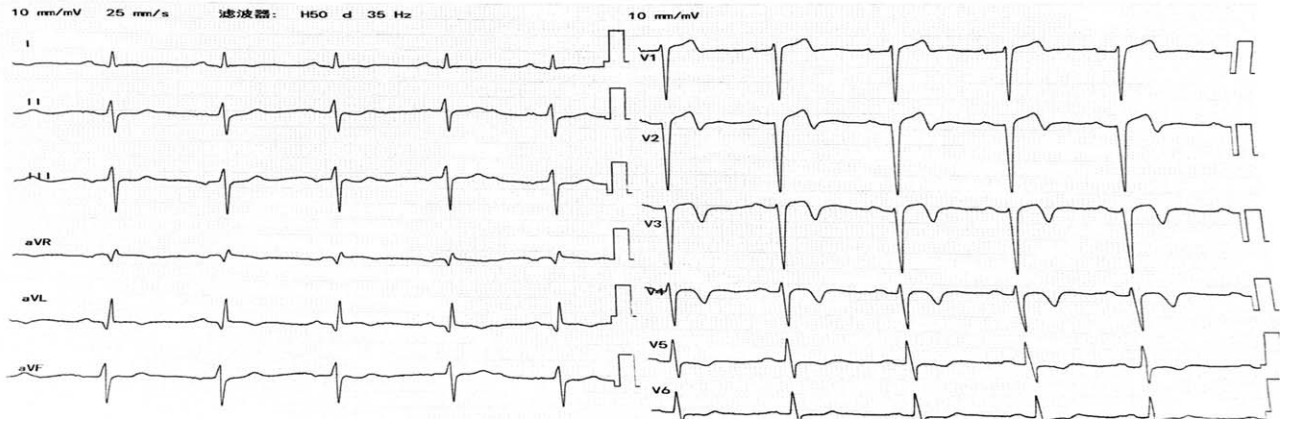

Figure 4. The image shows rS in V2-V4 leads; T wave inversion in the I, avL, and V2 to V5 leads; and the left anterior branch block.

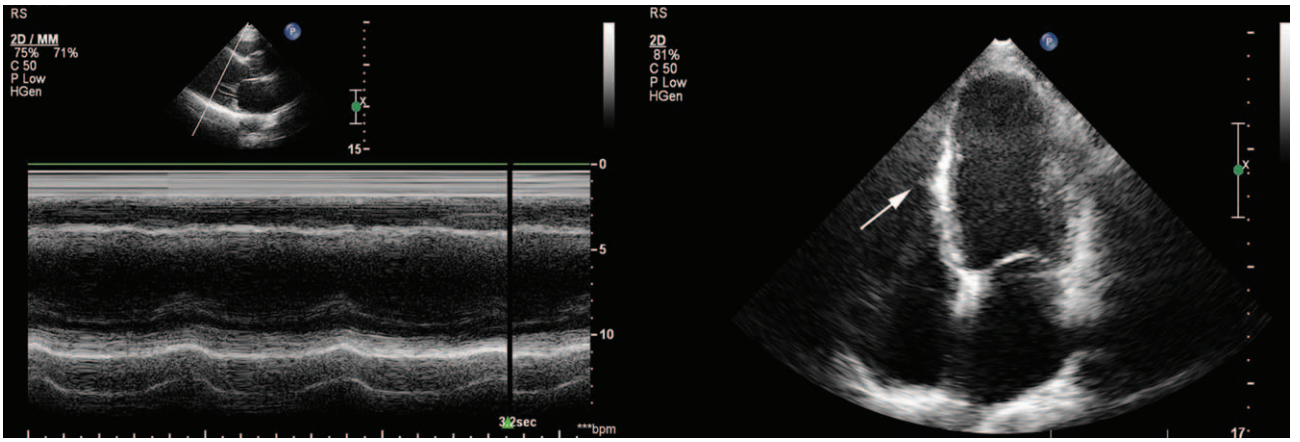

Figure 5. M-mode echo shows that the movement of interventricular septum was almost disappeared. Thinning of the interventricular septum is seen, with the thinnest part being 4.7-mm thick, and the echo was enhanced.

3. Discussion

The exact incidence of BCI is not clear, varying from less than 1% in clinical studies to over 30% in autopsy studies.[5] Motor vehicle collisions are the major cause of BCI, accounting for about 50% of cases, followed by pedestrians being struck by motor vehicles (35%) and motorcycle crashes (9%).[6,7] BCI is one of the mechanisms of non-arteriosclerotic myocardial infarction in the population of younger than 45 years old.[8] The most commonly affected coronary artery is the LAD, which was also shown in our case. The probable explanation is its anatomic position on the anterior wall of the heart. The second most commonly affected artery is the RCA, which is most vulnerable at its origin—perhaps because of acceleration/deceleration injury. The left main (LM) artery and LCX are seldom involved.[9] The main type of coronary injury is coronary dissection due to either direct contusion or an acceleration/deceleration force.

In our case, the patient was hit on the left side of the chest by a high-speed screw when he was working in a factory. Given the high speed and small diameter of the screw, the deceleration force was focused on a specific point in the chest with large compression to the heart. Unfortunately, on the basis of the angiography and IVUS results, we believe that the force was accidentally applied on the proximal LAD, resulting in unnatural curving of the coronary artery, subsequently leading to a coronary intimal dissection and myocardial infarction.

Prompt differential diagnosis of the myocardial infarction caused by BCI and appropriate management of this condition are critical in ensuring a favorable patient outcome. However, these are not easy as many of these cases are often unrecognized and treated late because traumatic chest pain sometimes masks anginal pain and is interpreted as being secondary to chest contusion or is masked by combined injuries. The physicians who initially treated the patient were unaware of the potentially devastating complications of blunt chest trauma, such as LM or proximal LAD dissection. Unfortunately, in our patient, the diagnosis was delayed, leading to an anterior wall infarction and LV dysfunction with a poor prognosis.

According to the practice guidelines of the Eastern Association for the Surgery of Trauma, all patients suspected of having BCI should undergo ECG during admission (Level I evidence).[10] BCI could be completely ruled in patients without new ECG abnormalities because most ECG changes after BCI tend to be transient, intermittent, and evolving. Instead, these patients should be given continuous cardiac monitoring[10] (Level II evidence) and undergoing cardiac Troponin-I (cTnI) test to determine the presence of myocardial injury. With high specificity and sensitivity, cTnI plays an important role in the differential diagnosis of cardiac injury.[11] Echocardiography is necessary in patients with compromised hemodynamics to rule out mechanical complications, such as cardiac tamponade, ventricle rupture, or valve injury.[12] The combination of normal ECG and negative cTnI (<0.4 ng/ml) findings 4 to 6 hours postinjury almost completely exclude a clinically significant BCI, with negative predictive values ranging from 98% to 100%.[13] These patients may be safely discharged home.

For patients with suspected myocardial infarction after trauma, emergency CAG should be performed as soon as possible to determine the culprit vessel. The management of myocardial infarction after blunt trauma remains controversial and lacks guidelines; management mainly includes conservative drug therapy, emergency PCI treatment, and emergency surgical coronary artery bypass graft (CABG) treatment. Emergency PCI should be the best treatment choice for patients without obvious hemorrhage in other organs because it allows rapid revascularization of the culprit vessel and is minimally invasive.[14,15] Early conservative treatment following delay of PCI is an optional method in cases accompanied by hemorrhage in other organs.[16] CABG has a good outcome for patients compared with PCI, but its application is limited in cases of multiple trauma due to the substantial risk of bleeding.

It is unknown whether the use of stents during PCI treatment can be used for patients with trauma. However, we believe that if the intima of the culprit vessel tears, leading to thromboembolism that could affect the distal blood flow, then the stents should be used. Otherwise, the torn intimal has no effect on the distal blood flow and will have less possibilities of thrombosis formation. Moreover, we do not recommend performing stent implantation in patients without angina, because it was reported that the intima tears are self-healing.[17] In our patient, although the proximal LAD artery was significantly abnormal, its normal distal blood flow and IVUS results allowed us to finally proceed with conservative management. For selecting the appropriate stent type, we believe that in the younger population with no obvious risk factors for coronary heart disease, bare-metal stents should be prioritized, instead of drug-eluting stents, because bare-metal stents require short-term anticoagulant therapy,[18] thereby lowering the bleeding risk, particularly in patients with trauma in other organs. Once bleeding occurs, the discontinuation of anticoagulant drugs can lead to lower risk of stent thrombosis and restenosis when using bare-metal stents than when using drug-eluting stents.

The incidence of BCI in China is unknown and most of the Chinese people are hesitant toward autopsy examinations; thus, the mortality from BCI remains unclear. Primary hospital clinicians in China have lesser experience and lack sufficient knowledge in the care of patients with BCI caused by severe trauma. Cases such as ours, in which the patient was misdiagnosed and had delayed treatment, leading a poor prognosis, are typical in China. Therefore, improving the physicians' knowledge about posttraumatic cardiac injury and developing guidelines for BCI management as soon as possible are necessary.

Author contributions

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Writing – original draft: Xiangdong Li, Zhiyuan Wang.
Writing – review and editing: Xiangdong Li, Zhiyuan Wang, Yushuang Yang, Fanbo Meng, Yuquan He, Ping Yang.

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