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Traumatic coronary artery dissection misdiagnosed as stress-induced cardiomyopathy in a patient with multiple trauma

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ABSTRACT

Traumatic coronary artery dissection resulting from blunt trauma, is a relatively rare and life-threatening event. We present a case report of a 42-year-old male who presented with electrocardiogram abnormality and cardiac enzyme elevation following a fall from a height of 3 m. The patient was misdiagnosed with stress-induced cardiomyopathy because of the absence of clinical signs of acute coronary syndrome. The patient was subsequently diagnosed with traumatic coronary artery dissection using coronary angiography, and the relevance of the trauma was confirmed using intravascular ultrasonography (IVUS). Herein, we highlight that trauma team should maintain a high suspicion of traumatic coronary artery dissection, although the early recognition of traumatic coronary artery dissection can be difficult. Additionally, the importance of coronary angiography with IVUS modalities for the evaluation of traumatic coronary artery dissection is highlighted.

Introduction

Blunt chest trauma can lead to various forms of cardiac contusions and structural injuries, including coronary artery dissection. While traumatic coronary artery dissection is a rare, life-threatening event, patients typically present from acute coronary syndrome to sudden cardiac death [1]. The recognition of traumatic coronary artery dissection can be difficult because of the lack of guidelines to assist with diagnosis and treatment [2]. Here, we present a case of misdiagnosed traumatic coronary artery dissection in a patient with multiple trauma without the typical chest pain.

Case

A healthy 42-year old male presented to the emergency department with a fall from a height of 3 m during roof construction. The patient complained of sharp pain in the left lateral chest wall, and his initial vital signs were as follows: blood pressure 130/80 mmHg, heart rate 104 beats/min, with mild tachycardia, and Glasgow Coma Scale score 14 with drowsy mentality. There were no specific

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findings in the patient's medical history. After primary and secondary surveys in the trauma bay, the patient was transferred to Computer Tomography (CT) room. On his left side, the patient had multiple rib fractures, traumatic hemopneumothorax, scapular fracture, humerus fracture, and splenic laceration (Fig. 1A, B, C, D). His initial injury severity score was 17. The initial laboratory examination showed elevated troponin levels (2.05 ng/ml), and an initial electrocardiogram (ECG) showed ST elevation in leads V2–V6 (Fig. 2A).

The cardiac two-dimensional echocardiography showed abnormal findings, with apex wall motion and left ventricle ejection fraction of 36 %. The cardiologist considered the patient's diagnosis as a stress induced cardiomyopathy rather than myocardial infarction (MI), considering the absence of typical symptoms such as prominent substernal chest pain and dyspnea, the patient's young age, and no underlying disease. The patient was admitted to the traumatic intensive care unit and monitored ECG and anticoagulation medications. Three days later, improvement of the patient's apex wall motion was observed on a 2D-echocardiograph, and troponin level improved to 1.00 ng/ml. Although the patient did not complain of chest pain, ST elevation in leads V2-V6 on ECG persisted (Fig. 2B). Chest CT angiography was performed, complete occlusion was found over a relatively long segment in the proximal left anterior descending (LAD) artery, that had resulted in 90 % stenosis of the vessel (Fig. 3A). The cardiologist decided to perform coronary angiography, which revealed 99 % dissection of the proximal LAD coronary artery.

To determine the relevance of the trauma, intravascular ultrasonography (IVUS) was performed. The IVUS image of the proximal LAD demonstrated a large amount of hematoma that compressed the true lumen, and moderate plaques in the proximal LAD target lesion (Fig. 3B). The remaining coronary arteries showed normal findings.

The patient underwent percutaneous coronary intervention, with the subsequent deployment of drug-eluting stents, which improved the initial 99 % stenosis (Fig. 4A, B). The patient was monitored in the cardiac ICU and was medically stable once. After the patient underwent fixation for a left humerus fracture on HOD 20, he was discharged on dual antiplatelet therapy and a beta-blocker.

Discussion

Traumatic coronary artery dissection is a rare but life-threatening complication of blunt thoracic trauma, with severe cardiac injury accounting for only 5–15 % of cases, originally described in 1988 [1,3]. There is no clear relationship between the severity of chest wall trauma and the development of coronary lesions, making it difficult to diagnose of traumatic coronary artery dissection [4], and there are no guidelines available to assist with identifying patients at risk [2].

The mechanism of injury is thought to be shearing of the coronary vessel wall, causing intimal tear with propagation to the media and the torn intima then creates a flap that obstructs the blood flow [2,5]. Previous reports demonstrated the LAD is most often injured, accounting for 76 % of cases, while the right coronary artery accounts for 12 % of cases and the circumflex artery accounts for 6 % of cases [3,6]. Traumatic coronary artery dissection presents with symptoms of severe substernal chest pain and dyspnea, leading to collapse due to hemodynamic instability. The diagnosis of traumatic coronary artery injury may be missed as it is rare, and the time difference from the actual chest injury to coronary artery trauma can vary from 48 h to sometimes five weeks [7]. Patients with chest trauma have elevated troponin levels, which could be due to structural cardiac injuries such as myocardial contusion, myocardial rupture, or skeletal trauma, and it is important to distinguish these from the elevation in cardiac troponin due to MI from coronary artery injury [8]. In addition, it might also demonstrate regional wall motion abnormalities when there is coronary artery occlusion, or stressed induced cardiomyopathy, as it did in this report, in which the 2D-echocardiography revealed regional wall motion abnormalities of left ventricle apex.

Differentiation between stress-induced cardiomyopathy (SCMP) and MI is important in trauma patients who present with ST and troponin elevation, but this is still difficult. SCMP may mimic MI and is an important condition in patients with underlying stress states. Reversible stress-induced cardiac dysfunction, mostly asymptomatic, is frequently seen as a complication of a multitude of acute stress state abnormalities on ECG, and patients often have an increase in cardiac enzymes. The classic pattern of wall motion abnormalities

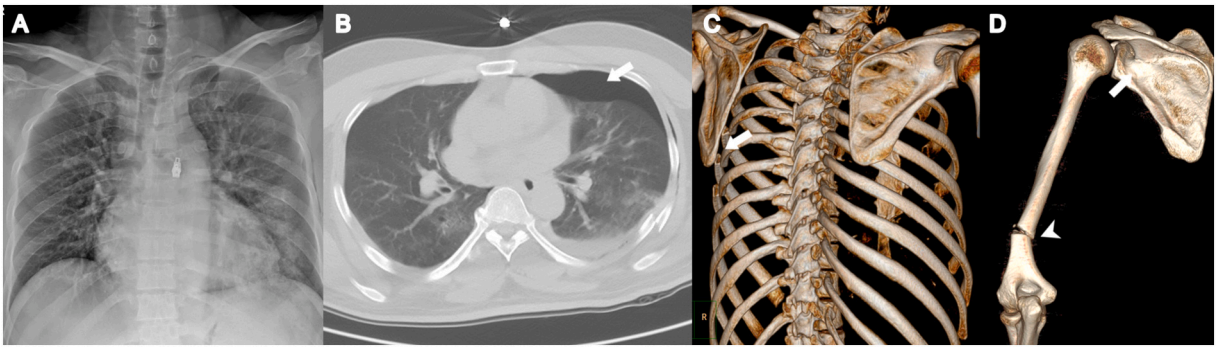

Fig. 1. Initial image work-up in the emergency room. (A) Chest X-ray showing left pulmonary congestion. (B) Computed tomography (CT) scan of the chest, showing left hemo-pneumothorax (white arrow). (C) Three dimensional rib CT scan showing multiple rib fractures of left lateral arc portion (white arrow). (D) Three dimensional scapular CT scan showing left displaced transverse fracture of distal humeral shaft (arrowhead) and scapular fracture (white arrow).

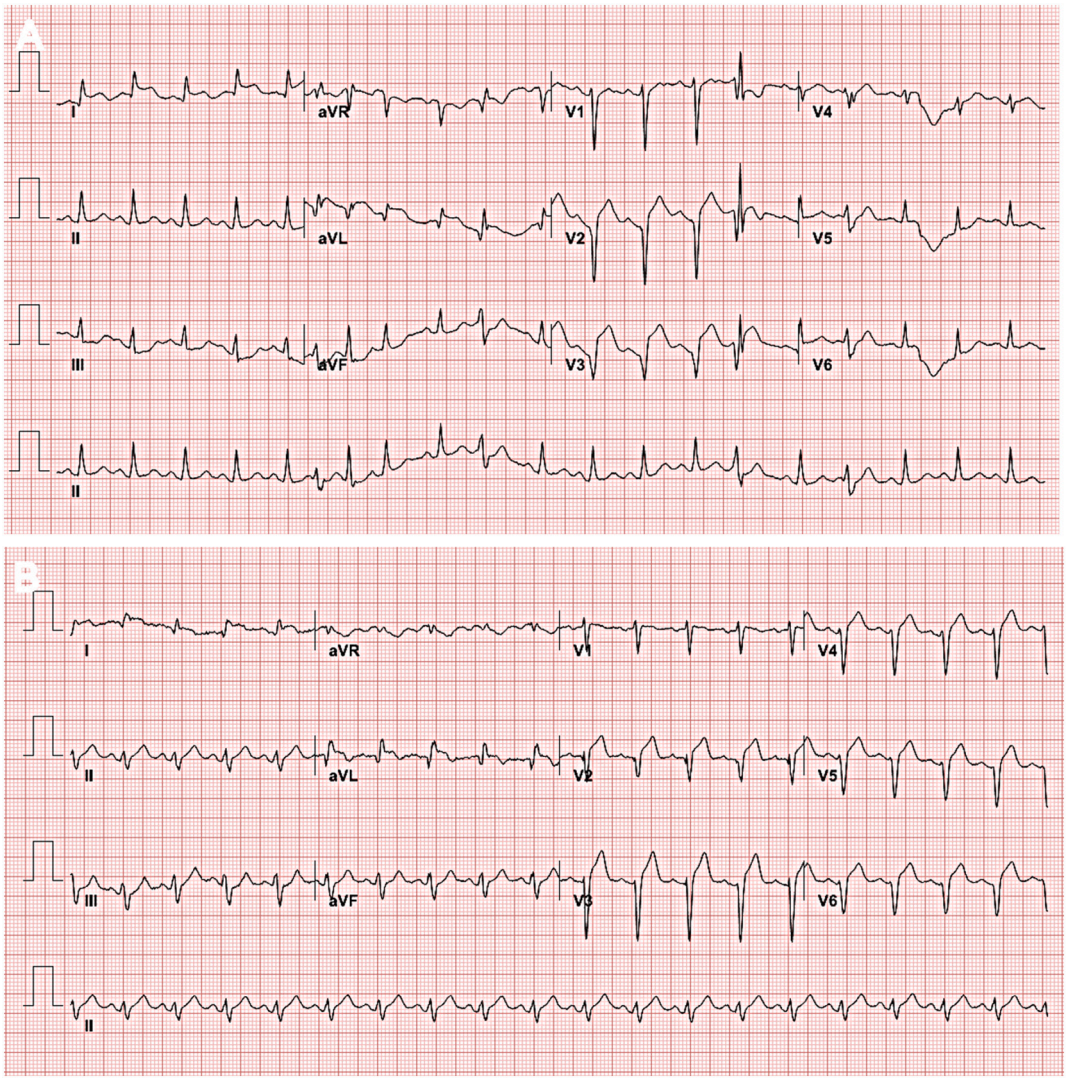

Fig. 2. (A) Initial electrocardiogram showing ST elevation in leads V2–V6, I, and aVL, heart rate 121. (B) On hospital day 3, electrocardiogram showing ST elevation in leads V2-V6, I, II and aVL, heart rate 115.

observed on the 2D-echocardiograph in SCMP is apical hypokinesia of the apex of the heart [9]. While most MIs are attributed to atherosclerotic occlusion, non-atherosclerotic etiologies play a larger role in the population with minimal comorbidities that predispose trauma patients to dissection or occlusion [10]. Distinguishing between traumatic coronary artery dissection and atherosclerotic plaque rupture can be difficult in traumatic situations. In this study, IVUS imaging played an important role in the evaluation of trauma relevance. The outward pushing pattern of the hematoma on the IVUS image was distinguished from atherosclerotic injury caused by atherosclerotic plaques in the vessel wall.

Coronary angiography remains the gold standard of diagnosis [2,3,7]. Traumatic coronary artery dissection can be managed with coronary artery bypass grafting, balloon angioplasty, angiography with stent placement, conservative medical management, or thrombolysis based on careful patient selection [2,3,5]. However, success rates for these treatment modalities are difficult to ascertain, owing to the small sample size and limited patient follow-up [2,8].

In this report, we considered a traumatic coronary artery dissection case of missed and delayed diagnosis mistaken for SCMP in a patient with multiple traumas. The trauma team should maintain a high suspicion of traumatic coronary artery dissection, although the recognition of traumatic coronary artery dissection can be difficult because of the various symptoms and lack of guidelines in assisting with the diagnosis and treatment. Coronary angiography is a simple procedure available with trauma center and should be utilized to either diagnose or exclude traumatic coronary artery dissection irrespective of any available guidelines.

In conclusion, in younger patients with ECG abnormality and cardiac enzyme elevation, the trauma team should consider traumatic coronary artery dissection more closely, even if there are no clinical signs of acute coronary syndrome. Coronary angiography with IVUS image played an important role in the evaluation of trauma relevance in a patient with traumatic coronary artery dissection.

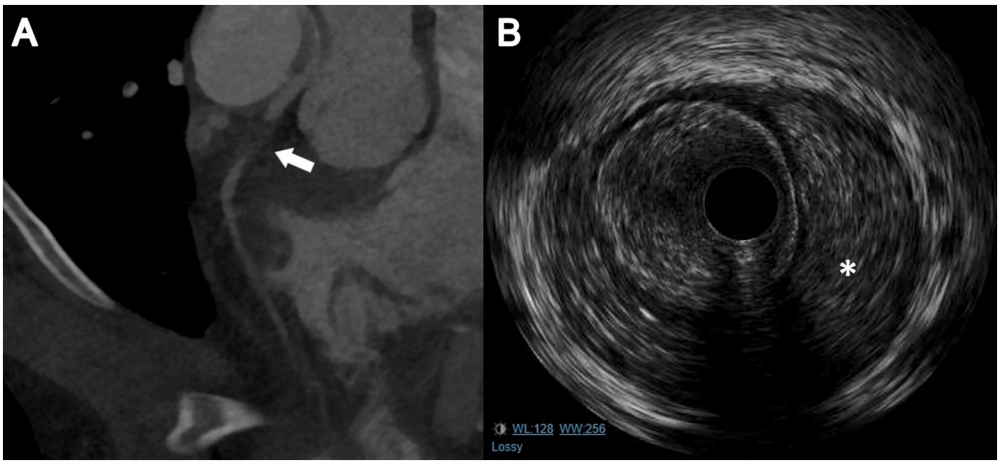

Fig. 3. (A) Chest CT angiography showing complete occlusion of long segment in the proximal left anterior descending (LAD) artery (white arrow). (B) Intravascular ultrasonography image showing a large amount of hematoma (arrowhead) that compressed the true lumen of proximal LAD.

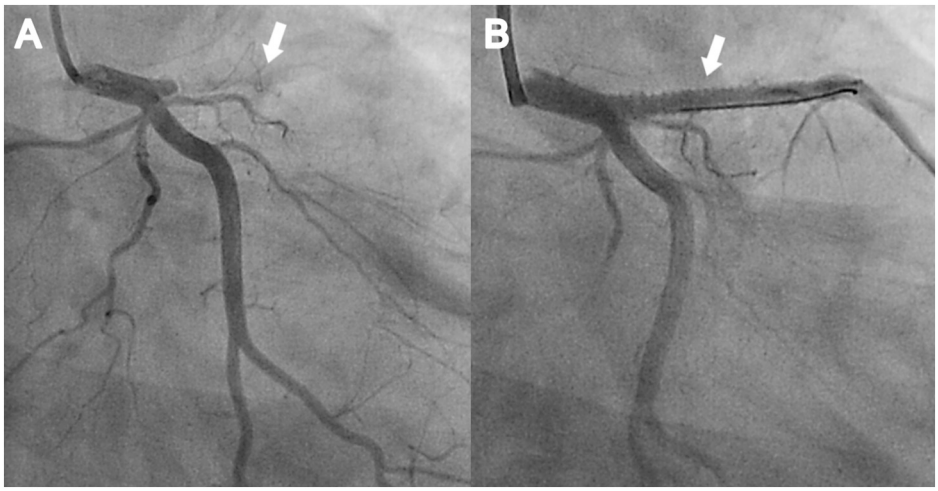

Fig. 4. (A) Coronary angiography showing a lesion in the proximal left anterior descending (LAD) artery with 99 % stenosis (white arrow). (B) Coronary angiography image after placement of drug-eluting stents into the proximal LAD artery (white arrow) showing final 0 % stenosis.

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Code availability

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CRediT authorship contribution statement

JL and SS collected the case data and drafted the original manuscript. JL and SL treated the patients and critically reviewed the manuscript. All authors read and approved the final manuscript.

Declaration of competing interest

The authors declare that they have no competing interests.

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We certify that this case report is our own work and all sources of information used in this report have been fully acknowledged.

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