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CASE REPORT

Right coronary artery dissection with inferior wall ST-segment elevation in a patient with thoracic blunt injury

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Abstract

Background: Blunt chest trauma, commonly caused by traffic accidents, is often associated with musculoskeletal injuries such as rib fractures and lung contusions. These symptoms may obscure more critical conditions like cardiac injury, resulting in delayed diagnosis and treatment. Although rare, blunt trauma can lead to coronary artery dissection and myocardial infarction. **Case**: We report a case of a 61-year-old female who presented to the emergency department with chest pain following a motor vehicle accident involving airbag deployment. Initial Computed Tomography (CT) scans identified multiple rib and cervical spine fractures, and chest pain was presumed to be musculoskeletal in origin. However, a delayed 12-lead electrocardiogram showed ST-segment elevation in the inferior leads, and subsequent coronary angiography confirmed a right coronary artery dissection. Hemodynamic instability and unsuccessful Percutaneous Coronary Intervention (PCI) were followed by emergency Veno-Arterial Extracorporeal Membrane Oxygenation (ECMO) placement. Despite aggressive management, her condition deteriorated, and she ultimately died after withdrawal of life-sustaining treatment. **Conclusions**: Blunt chest trauma can cause life-threatening cardiac injuries, including coronary artery dissection and myocardial infarction. In this case, electrocardiographic findings were key to identifying cardiac involvement, and coronary angiography confirmed right coronary artery dissection. Early use of 12-lead Electrocardiogram (ECG), cardiac biomarkers, and appropriate imaging is essential for timely diagnosis. For hemodynamically unstable patients, rapid intervention such as PCI, Coronary Artery Bypass Grafting (CABG), or ECMO should be considered. Increasing awareness of potential cardiac complications following blunt trauma is critical to improving survival outcomes.

Keywords

Chest pain; Blunt cardiac injury; Blunt trauma; ST elevation MI; Emergency center

1. Introduction

Most blunt chest injuries are caused by traffic accidents and include musculoskeletal injuries such as rib and spinal fractures and lung bruises [1]. Cardiac injury might also occur [2], leading to severe bleeding, arrhythmia, coronary thrombosis, and septum or valve rupture. Very rarely, electrocardiogram and coronary artery disruptions might suggest acute myocardial infarction [2, 3]. This is caused by coronary artery blockage due to blood clots, partial coronary artery convulsion, thrombosis due to coronary detachment, and embolism. The incidence of secondary coronary damage is about 2% [4, 5]. Coronary artery dissection is uncommon in patients visiting the emergency room following blunt chest trauma. As chest pain is the leading symptom, imaging tests are performed first, while 12-lead electrocardiography is often delayed, making it easy to misdiagnose patients with acute myocardial infarction. Furthermore, if a 12-lead electrocardiogram is performed and detects S wave–T wave segment (ST-segment) elevation, it is difficult to determine whether acute myocardial infarction led to the accident or the ST-segment elevation was due to vascular damage caused by the accident. To increase awareness, we report a case with blunt chest trauma and ST-segment elevation who visited the emergency room complaining of chest pain. The study protocol was approved by the Institutional Review Board of Boramae Medical Center (IRB file no. 30-2022-114). The requirement for informed consent was waived by the IRB.

2. Case presentation

A 61-year-old female was in the front passenger seat when the car crashed into a guardrail an hour and 10 minutes before reaching the emergency room. The airbags exploded during the accident. According to the patient's husband, who was the driver and sustained only minor injuries without loss of consciousness, the patient began experiencing chest pain after the trauma and had not reported any chest pain before the accident. She was subsequently transported to the emergency room by ambulance. The patient had no specific history other than high blood pressure and had no history of surgery or previous trauma. The family and social history presented no specific findings. On arrival, the assessment indicated the following: blood pressure, 75/49 mmHg; pulse 43 beats/min; body temperature, 35 °C; respiratory rate, 24 breaths per minute; and oxygen saturation, 96%. The patient's height was 149 cm, and body weight was 64.5 kg. The patient had normal consciousness (Glasgow Coma Scale E4V5M6) and complained of continuous chest pain of 6 points on a numeric rating scale, and difficulty breathing because of it. The first overall trauma assessment indicated that the airway was normal because the patient could talk clearly. A cervical spine protector was applied because the patient lost consciousness at the time of the accident. There were no signs of tension pneumothorax such as lost or diminished breathing sounds on one side of the chest; the patient only reported discomfort due to pain. Since the blood pressure was low (75/49 mmHg), an intravenous route was secured with an 18 G catheter, and 500 mL of plasma solution was quickly administered (Full Drip). Physical deformation was not observed, but a laceration of about 1.5 cm was noted above the upper lip. Echocardiography and Focused Assessment with Sonography for Trauma (FAST) were not performed. Computed tomography (CT) scans were performed immediately after the first trauma assessment and history taking. These included non-contrast brain, chest, and cervical spine (C-spine) + 3-dimensional (3D) CT scans. Brain CT showed no sudden cerebral hemorrhage, Chest CT showed a fracture in the second left rib (Fig. 1), as well as fractures in the bilateral second and third ribs and the right third to eighth ribs. C-spine + 3D CT showed left transverse process fractures in cervical vertebrae 4–7 (Fig. 2). CT scans were performed first because the patient's symptoms and physical examination patterns concorded with pain and other symptoms related to musculoskeletal damage. The 12-lead electrocardiogram, performed immediately after returning from the CT room, showed ST-segment elevation in leads II, III, and augmented Voltage Foot lead (aVF). Inverted electrocardiography showed right coronary artery (RCA) infarction as indicated by ST-segment elevation in leads reverse V4–6 (Fig. 3). The Department of Circulatory Internal Medicine was immediately contacted. After visiting the CT room, the patient's consciousness gradually decreased, the pulse decreased to 34 beats/min, and the blood pressure dropped to 69/40 mmHg. After applying transcutaneous pacing (output, 85 mA; rate, 70 beats/min), 2 mg lorazepam was administered for convulsing with eyeball deviation in the 12 o'clock direction. Cardiopulmonary resuscitation was performed ten minutes later as the patient lost a palpable pulse. Return of spontaneous circulation (ROSC) was achieved four minutes after intubation and administration of 1 mg epinephrine. The laboratory findings upon arrival were nonspecific, except for the increased troponin I (35.5 pg/mL), Creatine Kinase-Myocardial Band (CK-MB) (9.0 ng/mL), glutamic oxaloacetic transaminase (116 IU/L), glutamic pyruvic transaminase (106 IU/L), and glutamine (1980 mg/L). After ROSC, a Levine tube was inserted to deliver three tablets of 100 mg aspirin and eight tablets of 75 mg clopidogrel. As the blood pressure, monitored by an arterial line, remained low (77/39 mmHg), dobutamine was administered at 0.5 mcg/60 kg/min. However, as the blood pressure dropped further (56/20 mmHg), the dobutamine administration rate was increased to 15 mcg/60 kg/min, and half an ampule of epinephrine and

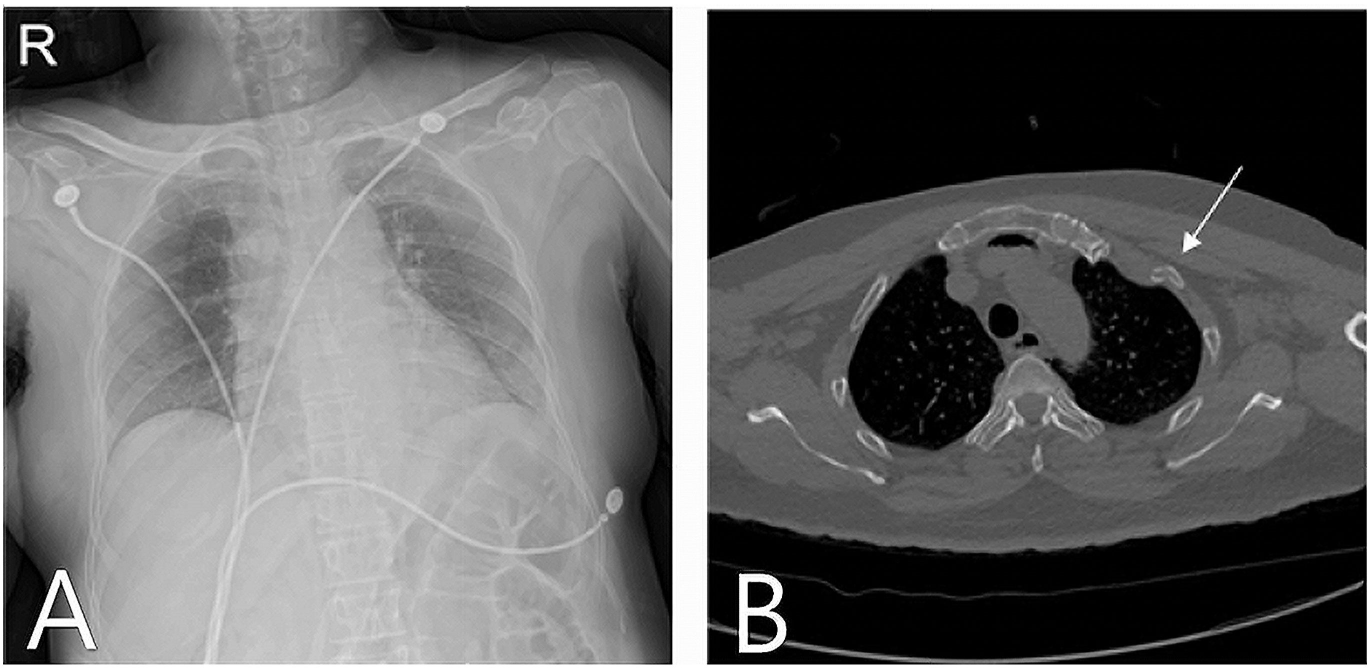

FIGURE 1. Initial imaging findings following blunt chest trauma. (A) The chest radiograph did not demonstrate acute intrathoracic injury. (B) Chest computed tomography showed a left second rib fracture.

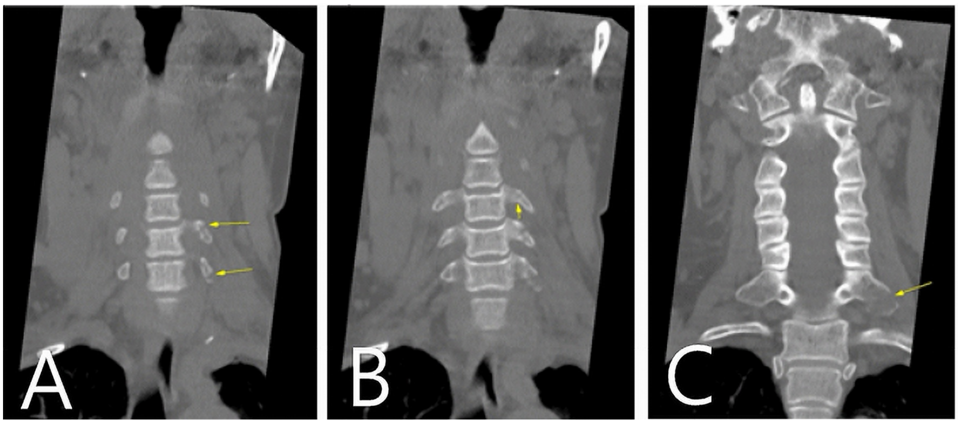

FIGURE 2. Cervical spine computed tomography in coronal view. (A) Left C5–C6 transverse process fractures (arrows indicate the fracture sites). (B) Left C4 transverse process fracture (arrow). (C) Left C7 transverse process fracture (arrow).

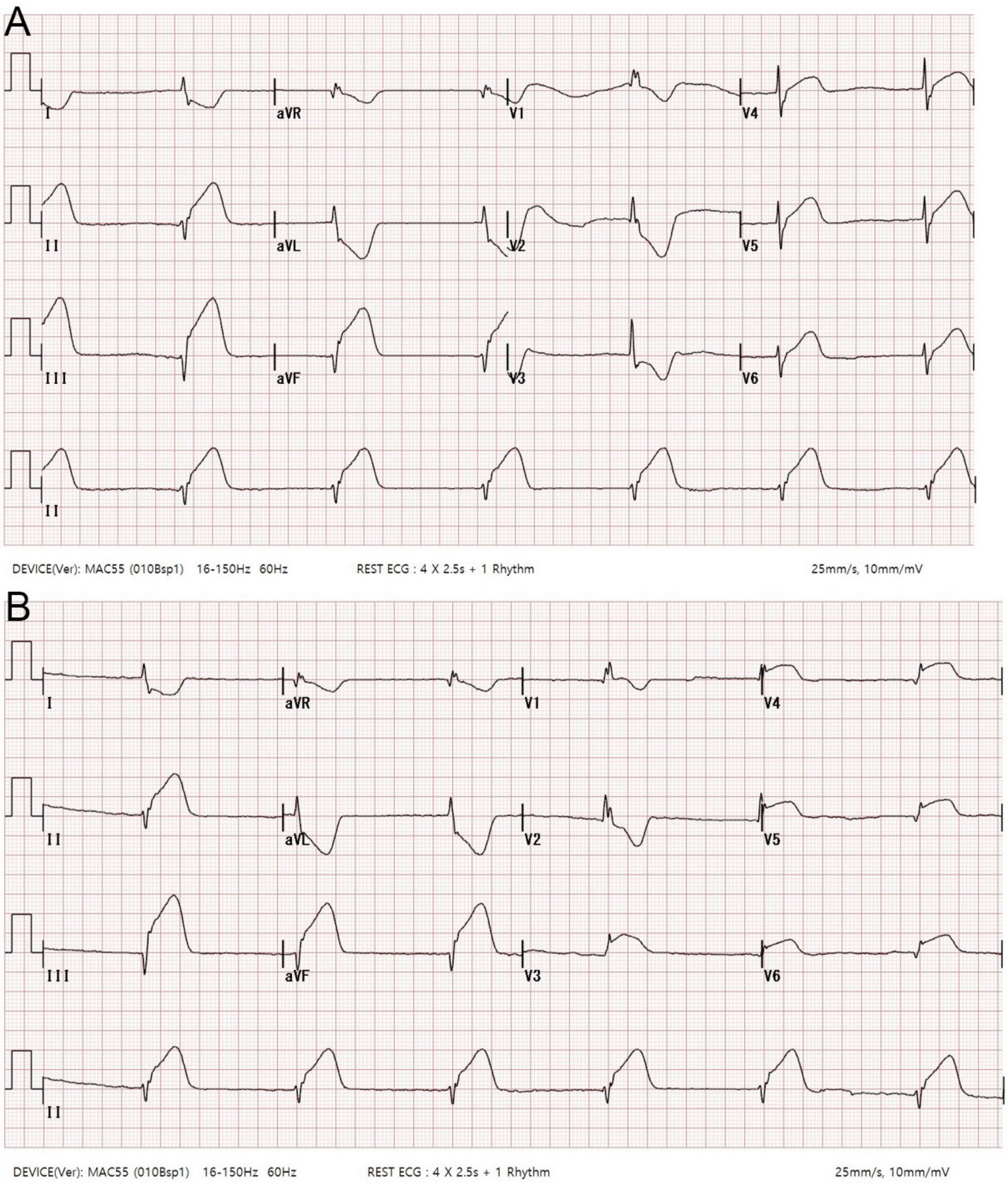

FIGURE 3. Electrocardiographic findings. (A) Electrocardiogram showing diffuse inferior wall ST-segment elevation. (B) Inverted electrocardiogram showing ST-segment elevation in leads V4–6. aVR: augmented Voltage Right lead; aVL: augmented Voltage Left lead; aVF: augmented Voltage Foot lead; ECG: Electrocardiogram.

32 μg/min norepinephrine were administered to maintain the blood pressure. As the pulse dropped to 40 beats/min, 0.5 mg atropine was administered. Once the blood pressure and pulse were stabilized, the patient was transferred to the cardiovascular room for percutaneous coronary intervention (PCI). Angiography revealed a two-vessel disease (2VD), including a dissection in the proximal right coronary artery (pRCA) with the formation of a false lumen. Due to the difficulty in advancing a wire into the true lumen, PCI at the dissection site was unsuccessful. Consequently, a temporary pacemaker was inserted, and surgical options, including transsternal (TS) coronary artery bypass grafting (CABG), were discussed. As a result, a pacemaker was placed, and CABG surgery was planned. The patient was transferred to the surgical intensive care unit, remained intubated, and was on ventilator support (Fraction of Inspired Oxygen (FiO_2): 70%, mode: Pressure-Controlled ventilation (PCV), Pressure: 8 cmH_2O, Respiratory rate: 17 breaths/min, and Positive End-Expiratory Pressure (PEEP) level: 5 cmH_2O), where repeated ventricular tachycardia (V-tach) and pulseless electrical activity rhythm occurred. After 20 minutes, the patient was transferred to the cardiovascular room to insert veno-arterial extracorporeal membrane oxygenation (VA ECMO). Shocks were delivered more than ten times as arrhythmia, such as V-tach, occurred. CABG was not performed immediately after the VA ECMO was inserted because the pupils showed no response, and brain infarction was detected. The patient's prognosis was exceedingly poor. Later, the patient was disconnected from the VA ECMO and life-sustaining treatments were discontinued following the guardian's request. On the 26th day since admission, tracheal extubation was performed after completing the Physician Orders for Life-Sustaining Treatment (POLST) form, and the patient died.

3. Discussion

A study reviewed 76 patients with myocardial infarction secondary to trauma between 1974 and 2005. Of these 49 (63.5%) had traffic accidents, and 55 (72%) had coronary occlusion (n = 43) or dissection (n = 12) [6]. Coronary artery damage in patients with blunt chest trauma is divided into five types: detachment due to endocardial rupture, ischemia caused by artery compression, stenosis caused by external compression or internal edema, coronary artery rupture, and fistula [7]. Dissection starts with the tear of the inner membrane around a plaque. The damage spreads by the blood flow, while the torn inner membrane blocks/disrupts the blood flow and creates a blood clot that causes infarction [8]. In rare cases, coronary artery dissection caused by blunt chest trauma can lead to such myocardial infarction, typically in the distal left anterior thoracic (76% of the cases) or right coronary (12%) artery, resulting in obstruction and thrombosis [9]. The Eastern Association for the Surgery of Trauma recommends a 12-lead electrocardiogram scan early in patients after blunt chest trauma due to possible cardiac injury [10]. However, in this case the patient's pain pattern was non-specific and the cause of chest pain was assumed to be musculoskeletal damage, such as a rib fracture. Due to this, and a higher than normal possibility of spinal shock with cervical fracture, the 12-lead ECG was delayed and the cardiac injury was missed. Early 12-lead electrocardiography and tests for cardiac markers such as troponin I may be helpful in cases such as these [11].

Although cardiac marker tests in patients with blunt chest trauma are controversial, it is recommended to perform them because they can help detect myocardial infarction early, suggest possible cardiac injury, and lead to quick PCI [7, 10]. Furthermore, echocardiography should be performed to identify possible structural and non-structural cardiac damages in patients with hemodynamic instability or persistent new arrhythmias [12].

The treatment of coronary artery injury due to blunt chest trauma varies depending on the patient; however, as using thrombolytic agents after a severe trauma has a very high risk of bleeding, these patients are usually managed by PCI instead [13]. While PCI is used in most cases, surgical angioplasty could be more effective in some. Surgical angioplasty is preferred in the case of left main coronary artery damage (but not right coronary or left anterior descending artery), contraindications for antiplatelet therapy, and patients at a high risk of bleeding [14]. In our case, a right coronary artery dissection was found, but the patient was hemodynamically unstable, and antiplatelet therapy use was limited due to the severe trauma. PCI was attempted, but it was difficult to gain access; consequently, CABG surgery was planned, but it was not performed due to repeated cardiac arrests, the unstable condition, and a poor prognosis. However, ECMO was applied after cardiac arrest, suggesting a potential delay in its application. According to the literature review, ECMO is indicated in cardiogenic shock when systolic blood pressure (SBP) is below 90 mmHg or when maintaining stable perfusion becomes difficult. Additionally, if PCI fails and revascularization is not possible, ECMO should be initiated immediately in cases of progressive hemodynamic instability. In cardiac arrest, the Extracorporeal Cardiopulmonary Resuscitation (ECPR) criteria suggest that ECMO initiation within 21 minutes is associated with better neurological outcomes, while initiation after 30 minutes results in poor prognosis, and after 60 minutes, effectiveness is significantly reduced. Given the patient's progressive hypotension (69/40 mmHg) and failed PCI, earlier ECMO initiation could have provided hemodynamic support, potentially allowing CABG to be performed before further deterioration [15]. Physicians should consider the possibility of coronary artery damage in patients visiting the emergency department following a blunt chest trauma as it has a high mortality rate.

Chest pain and non-specific clinical features after trauma are often underestimated as these are similar to symptoms associated with musculoskeletal damages, such as rib fractures, or obscured by such injuries, making diagnosis difficult. Therefore, 12-lead electrocardiogram and cardiac marker tests should be performed in all patients presenting after blunt chest trauma. Furthermore, CT scans or echocardiography should be considered as they can detect injury to blood vessels. If indicated by the 12-lead electrocardiogram, PCI should be performed quickly in such patients. If PCI fails, CABG should be considered. Additionally, if PCI fails and the patient remains hemodynamically unstable, ECMO should be considered earlier to provide circulatory support and improve treatment outcomes.

4. Conclusions

Blunt chest trauma can result in serious cardiac injuries, including coronary artery dissection and myocardial infarction, which are often overlooked due to non-specific symptoms resembling musculoskeletal injuries. Early 12-lead electrocardiography and cardiac marker tests, such as troponin I, are crucial in detecting cardiac involvement and guiding timely intervention. Imaging tools like CT scans and echocardiography can further identify vascular damage. Prompt PCI is typically the preferred treatment, though CABG should be considered if PCI fails, especially in cases involving severe trauma or high bleeding risk. Delayed diagnosis can result in poor outcomes, emphasizing the importance of early cardiac evaluation in blunt chest trauma patients presenting with chest pain.

5. Limitations

This study presents a single case, which may limit generalizability. Due to the emergent nature of the case, certain diagnostic evaluations such as early echocardiography and serial cardiac markers could not be fully performed. Despite this, the case highlights important clinical considerations in the assessment and management of blunt cardiac injury.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

AUTHOR CONTRIBUTIONS

HJK, SYJ, HJC—designed the study and wrote the initial draft. SYJ and HJC—performed data collection and analysis. HJK—provided critical revisions and clinical interpretation. HJC—contributed to the literature review and manuscript refinement. All authors contributed to editorial changes, read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was approved by the Institutional Review Board of Boramae Medical Center (IRB file no. 30-2022-114). The requirement for informed consent was waived by the IRB due to the retrospective and anonymized nature of the case.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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