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Delayed Recognition of Inferior ST-Segment Elevation Myocardial Infarction After Blunt Chest Trauma With Rib Fractures and Severe Right Coronary Artery Stenosis: A Case Report

Authors' Contribution:

Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient: Male, 56-year-old
Final Diagnosis: Myocardial infarction
Symptoms: Chest pain
Clinical Procedure: —
Specialty: Cardiology
Objective: Unusual clinical course
Background: Blunt thoracic trauma can produce chest wall pain that obscures concurrent acute coronary syndrome. This report describes delayed recognition of inferior ST-segment elevation myocardial infarction (STEMI) after blunt chest trauma with rib fractures and emphasizes the limitations of attributing the event to a trauma-specific coronary mechanism.
Case Report: A 56-year-old man developed persistent chest pain immediately after an anterior chest impact. At a local hospital, computed tomography showed fractures of the left seventh to ninth ribs, and no electrocardiogram (ECG) was initially obtained. Approximately 11 hours after injury, the first ECG showed inferior STEMI. He was transferred 13 hours after symptom onset; his pain had improved, he was hemodynamically stable, and repeat ECG showed ST-segment resolution with Q-wave formation. The cardiac troponin I level was 8.05 ng/mL on arrival and later peaked at 48 ng/mL. Coronary angiography on hospital day 10 showed approximately 90% stenosis of the proximal-to-mid right coronary artery. After balloon predilatation, optical coherence tomography showed a complex lipid-rich lesion with thrombotic features and localized dissection. Because imaging was performed after predilatation, the original mechanism could not be established. Percutaneous coronary intervention restored Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow. Cardiac magnetic resonance imaging 5 days later confirmed an ischemic-pattern infarction in the right coronary artery territory with microvascular obstruction.
Conclusions: Persistent or unexplained chest pain after blunt thoracic trauma warrants early ECG and troponin testing, even when rib fractures are present. Post-predilatation intracoronary imaging can characterize a culprit lesion but cannot, by itself, establish that trauma caused plaque disruption or coronary dissection.
Keywords: Wounds, Nonpenetrating • Myocardial Infarction • ST Elevation Myocardial Infarction • Coronary Vessels • Tomography, Optical Coherence • Magnetic Resonance Imaging
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Introduction

Blunt chest trauma can injure the myocardium, pericardium, valves, great vessels, and coronary arteries. Because chest wall pain and myocardial contusion may resemble acute coronary syndrome, an admission electrocardiogram (ECG) and cardiac troponin measurement are recommended when blunt cardiac injury is suspected [1].

For ST-segment elevation myocardial infarction (STEMI), immediate reperfusion is recommended within 12 hours of symptom onset. In late presenters, primary percutaneous coronary intervention (PCI) remains indicated when ongoing ischemia, hemodynamic instability, or life-threatening arrhythmia is present; routine PCI may be considered in selected stable patients who present 12 to 48 hours after symptom onset [2]. In trauma patients, these decisions must also incorporate the risk of active or delayed bleeding and competing injuries.

Published cases illustrate several mechanisms by which acute coronary events can follow blunt chest trauma. Regueiro et al described right coronary artery (RCA) dissection after blunt trauma [3]; Perez et al reported STEMI after blunt trauma caused by left anterior descending artery occlusion [4]; and Vyas et al documented traumatic RCA dissection presenting as inferior STEMI [5]. More recent reports have described delayed or severe consequences of traumatic RCA dissection [6,7] and emphasized the need to balance bleeding risk against timely coronary

reperfusion in trauma-associated STEMI [8]. These reports demonstrate the diagnostic challenge but do not establish a single mechanism for all post-traumatic myocardial infarctions.

The present report describes a 56-year-old man with anterior or blunt chest trauma, fractures of the left seventh to ninth ribs, and delayed recognition of inferior STEMI associated with severe proximal-to-mid RCA stenosis. The purpose is to illustrate how genuine traumatic chest wall pain can mask a concurrent acute coronary event and demonstrate how postpredilatation optical coherence tomography (OCT) and cardiac magnetic resonance imaging (CMR) can provide lesion- and tissue-level information while leaving the original coronary mechanism uncertain.

Case Report

A 56-year-old man was struck in the anterior chest by a heavy object at approximately 7:00 AM while working at a construction site. Persistent chest pain began immediately. He had a history of hypertension for more than 10 years, which was regularly treated and adequately controlled. He denied smoking or alcohol use and had no known coronary artery disease, diabetes, chronic kidney disease, or previous myocardial infarction.

At approximately 9:00 AM, he presented to a local hospital. Chest computed tomography (CT) showed fractures of the left

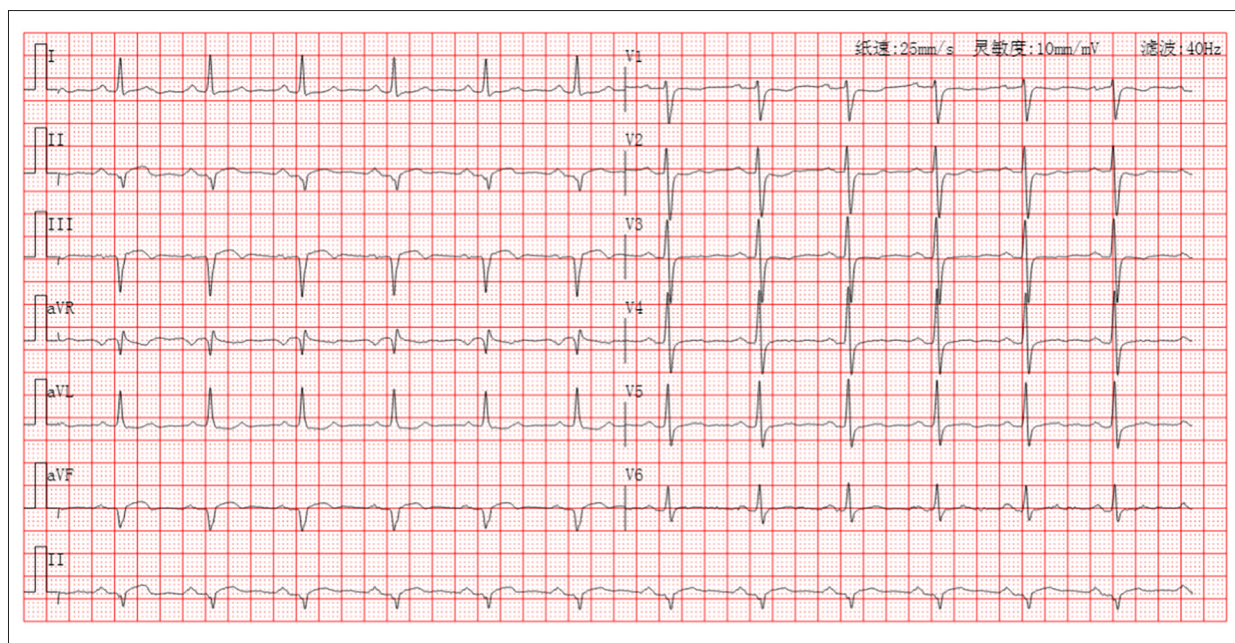


Figure 1. First diagnostic electrocardiogram after blunt chest trauma. The 12-lead electrocardiogram (ECG) obtained at the local hospital approximately 11 hours after injury showed inferior ST-segment elevation myocardial infarction (STEMI). A repeat ECG at the receiving hospital was documented as showing ST-segment resolution with Q-wave formation, but the tracing was not available for publication.

Table 1. Clinical timeline.

Time point	Clinical event
7: 00 AM, day 0	Anterior chest impact; persistent chest pain began
9: 00 AM, day 0	First medical contact; chest CT showed fractures of the left seventh to ninth ribs; no ECG was obtained
6: 00 PM, day 0	First diagnostic ECG showed inferior STEMI; aspirin 300 mg and clopidogrel 300 mg were administered
8: 00 PM, day 0	Transfer 13 hours after symptom onset; pain improved; repeat ECG was documented as showing ST-segment resolution with Q-wave formation
Hospital days 1-9	CCU monitoring and medical therapy; preprocedural heparin was withheld because of concern about delayed bleeding; no recurrent ischemia or hemodynamic instability
Hospital day 10	Coronary angiography showed severe proximal-to-mid RCA stenosis; post-predilatation OCT was performed; PCI restored TIMI grade 3 flow
Day 15 after symptom onset/day 5 after PCI	CMR showed an ischemic-pattern RCA-territory infarction with microvascular obstruction
Day 6 after PCI	Discharged on dual antiplatelet and secondary prevention therapy
1 month	No recurrent symptoms; LVEF improved to 47%, with residual posteroinferior hypokinesis

Abbreviations: CCU, cardiac care unit; CMR, cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; RCA, right coronary artery; STEMI, ST-segment elevation myocardial infarction; TIMI, Thrombolysis in Myocardial Infarction.

seventh, eighth, and ninth ribs. The pain was initially attributed to chest wall trauma, and the patient received analgesic and supportive treatment. No ECG was obtained at first medical contact. At approximately 6: 00 PM, because the pain persisted, the first documented ECG was obtained and showed inferior STEMI (**Figure 1**). Aspirin 300 mg and clopidogrel 300 mg were administered; he was transferred to our hospital at approximately 8: 00 PM, 13 hours after symptom onset.

Upon arrival, the chest pain had substantially improved. The patient was hemodynamically stable, without cardiogenic shock, recurrent severe chest pain, malignant arrhythmia, or overt heart failure. A repeat ECG was documented as showing resolution of ST-segment elevation with Q-wave formation; the repeat tracing was not available in archived materials for publication. No pre-injury ECG was available. The cardiac troponin I level was 8.05 ng/mL on arrival and later peaked at 48 ng/mL; the creatine kinase-MB level was 191 U/L. The archived record did not indicate whether the troponin assay was conventional or high-sensitivity; the manufacturer, 99th-percentile upper reference limit, exact serial sampling times, and timing of the peak value could not be retrieved. Transthoracic echocardiography showed inferior and lateral wall hypokinesis, with a left ventricular ejection fraction of 42%. Lipid levels were: total cholesterol, 4.38 mmol/L; triglycerides, 1.71 mmol/L; low-density lipoprotein cholesterol, 2.34 mmol/L; and high-density lipoprotein cholesterol, 1.71 mmol/L.

Because the patient was a late presenter, had no ongoing ischemic symptoms, had documented ST-segment resolution with Q-wave formation, remained hemodynamically stable, and had recent thoracic trauma with rib fractures, the treating team chose close cardiac care unit monitoring and medical therapy rather than immediate catheterization. Treatment included aspirin 100 mg once daily, clopidogrel 75 mg once daily, atorvastatin calcium 20 mg nightly, rabeprazole 10 mg once daily, ramipril 5 mg once daily, and sustained-release metoprolol succinate 23.75 mg once daily. To reduce the risk of delayed bleeding after the recent thoracic trauma, heparin anticoagulation was withheld prior to coronary angiography and PCI. Complete serial hemoglobin, platelet count, and coagulation results; detailed thoracic CT findings beyond the documented rib fractures; and a formal trauma consultation record were not preserved in the archived case materials. During observation, there was no recurrent chest pain, hemodynamic instability, or sustained ventricular arrhythmia. The chronological course is summarized in **Table 1**.

Coronary angiography was performed on hospital day 10. The left main coronary artery had no clinically significant stenosis, the proximal left anterior descending artery had noncritical plaque, and the left circumflex artery had no clinically significant stenosis. The RCA was considered the culprit vessel, with approximately 90% stenosis of the proximal-to-mid segment (**Figure 2A**). Distal opacification was preserved, and no definite pre-balloon dissection flap was visible on the available angiographic images.

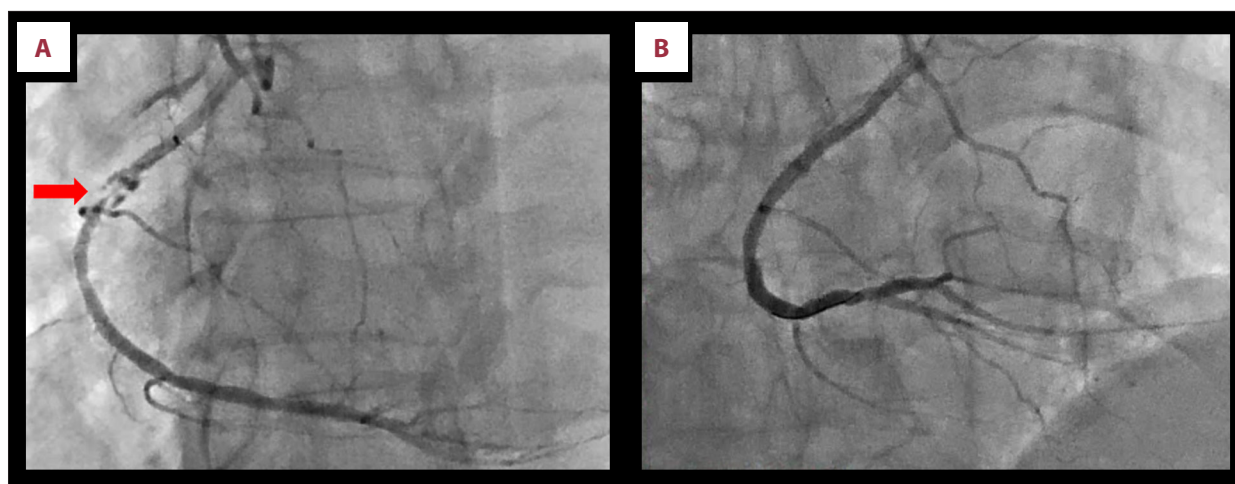


Figure 2. Right coronary angiography before and after percutaneous coronary intervention. (A) Pre-intervention coronary angiography demonstrated approximately 90% stenosis of the proximal-to-mid right coronary artery (red arrow), which was considered the culprit lesion. (B) Final angiography after drug-eluting stent implantation showed no clinically significant residual stenosis and Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow.

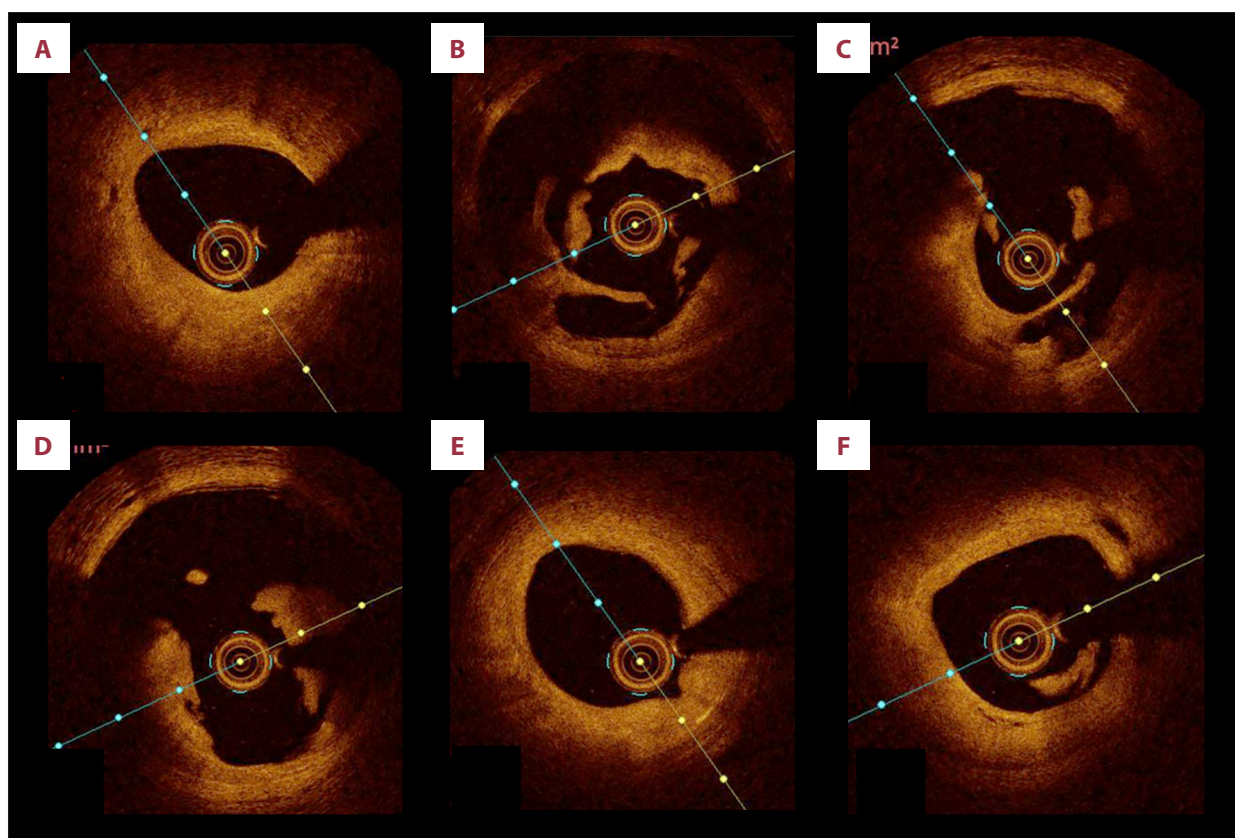


Figure 3. Post-predilatation optical coherence tomography of the right coronary artery. The optical coherence tomography (OCT) examination was reported to show diffuse lipid-rich plaque in the proximal-to-mid right coronary artery. Panels A-F illustrate selected associated features compatible with macrophage accumulation (A), plaque disruption (B), localized dissection (C), red thrombus (D), cholesterol crystals (E), and white thrombus-like or microchannel-like structures (F). OCT was performed after balloon predilatation; thus, localized dissection and other luminal features cannot establish the original trauma-specific mechanism and must be interpreted in the procedural context.

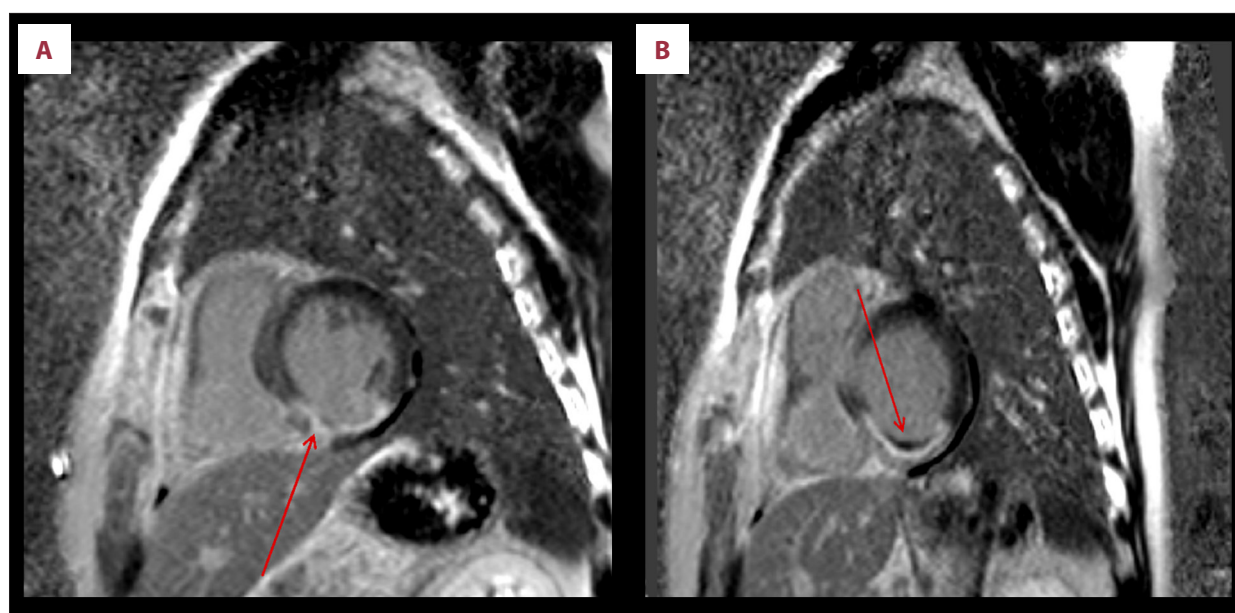


Figure 4. Cardiac magnetic resonance findings of right coronary artery-territory infarction with microvascular obstruction. Cardiac magnetic resonance imaging was performed 15 days after symptom onset and 5 days after percutaneous coronary intervention. Short-axis late gadolinium enhancement images demonstrate an ischemic-pattern hyperenhanced infarct zone involving the inferior and inferoseptal segments in the right coronary artery territory (**A**, red arrow) and a central hypoenhanced core consistent with microvascular obstruction (**B**, red arrow).

The RCA lesion was predilated with a 2.0×15 mm balloon. OCT performed after predilatation was reported to show a complex lipid-rich lesion. The selected images illustrate associated features compatible with plaque disruption, thrombotic material, cholesterol crystals, macrophage accumulation, microchannel-like structures, and localized dissection (**Figure 3**). Because OCT was performed after balloon predilatation, the localized dissection and some luminal features could have been pre-existing, trauma-associated, spontaneous, or procedure-related. The lesion was further prepared with a 2.5×15 mm balloon, treated with a 2.75×24 mm drug-eluting stent, and post-dilated with a 3.0×12 mm noncompliant balloon. Final angiography showed no clinically significant residual stenosis and Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow (**Figure 2B**).

CMR was performed 15 days after symptom onset and 5 days after PCI. Short-axis late gadolinium enhancement images showed ischemic-pattern hyperenhancement predominantly involving the basal-to-mid inferior and inferoseptal left ventricular segments; a central hypoenhanced core was compatible with microvascular obstruction (**Figure 4**). The inferior right ventricular wall was also involved. This coronary-territory pattern supported acute/subacute RCA-territory infarction, rather than chronic scar or isolated myocardial contusion. Left ventricular systolic function was mildly reduced, and a small pericardial effusion was present.

The patient was discharged 6 days after PCI on the same secondary prevention regimen. At the 1-month follow-up, he

remained free of chest pain, dyspnea, syncope, or palpitations. Follow-up echocardiography showed residual postero-inferior hypokinesis and an improved left ventricular ejection fraction of 47%.

Discussion

The main lesson from this case is that a true traumatic chest wall injury and an acute coronary event can coexist; the traumatic diagnosis can delay recognition of STEMI. In the present case, rib fractures plausibly explained part of the pain, but the persistent symptoms ideally would have prompted earlier ECG and troponin testing. The available evidence thus supports a concurrent trauma-associated acute coronary event that was initially masked by rib fracture-related pain; it does not prove that blunt trauma caused coronary contusion, plaque rupture, or primary RCA dissection.

The presentation in our case differs in important ways from previously published cases of trauma-associated STEMI. Regueiro et al and Vyas et al reported inferior STEMI with RCA dissection demonstrated on coronary imaging [3,5], whereas no definite dissection flap was visible on our pre-balloon angiographic images. Perez et al described anterior STEMI caused by left anterior descending artery occlusion after blunt trauma, emphasizing the need for prompt cardiac evaluation [4]. Paparoupa et al and Li et al demonstrated that traumatic coronary dissection

can present with severe electrical or hemodynamic deterioration [6,7]. In a 2026 report, Bahurmuz et al used serial ECG assessment and multidisciplinary bleeding-risk evaluation before PCI in a trauma patient with STEMI [8]. In contrast, our patient was hemodynamically stable after the delayed diagnosis, but the prolonged interval to angiography remains an important limitation rather than a recommended strategy.

The timing of coronary angiography requires explicit qualification. At transfer, the patient was 13 hours from symptom onset, chest pain had improved, ST-segment elevation had resolved with Q-wave formation, and there was no shock, recurrent ischemia, or malignant arrhythmia. Current guidance allows consideration of routine PCI in selected stable patients who present 12 to 48 hours after symptom onset [2]. Waiting until hospital day 10 should be understood as part of this real-world delayed-diagnosis course, not as a preferred STEMI pathway or a practice supported by biomarker normalization. The decision to withhold preprocedural heparin in the present case reflected concern about delayed bleeding after recent thoracic trauma. Because complete serial hemoglobin, platelet count, coagulation, detailed thoracic CT, and formal trauma consultation data were unavailable, the original decision-making process cannot be reconstructed more precisely.

The coronary mechanism remains uncertain. The severe RCA stenosis and lipid-rich, thrombotic OCT features are compatible with type 1 acute coronary syndrome in an atherosclerotic substrate. The immediate onset of pain after impact makes trauma a plausible trigger for plaque destabilization, transient occlusion, thrombosis, or spasm. However, no pre-trauma coronary imaging was available, spontaneous plaque rupture coinciding with the traumatic event cannot be excluded, and the left-sided rib fractures had no demonstrable direct anatomical relationship to the proximal-to-mid RCA lesion. The most supportable interpretation is trauma-associated inferior STEMI in a patient with an atherosclerotic RCA lesion, rather than proven trauma-induced plaque rupture or primary traumatic coronary dissection.

OCT provided high-resolution information about the culprit lesion but was performed only after balloon predilatation. Standardized OCT interpretation can identify thrombus, plaque disruption, macrophage-rich regions, crystal-like structures, and dissection [9]; OCT has been used to guide the management of coronary injury after blunt chest trauma [10]. In the present case, however, predilatation may have altered the lumen and created or extended the localized dissection. Accordingly, OCT findings characterize the post-predilatation lesion but cannot establish the original trauma-specific mechanism.

CMR helped distinguish infarction from isolated myocardial contusion. CMR has previously demonstrated coronary-territory

infarction after blunt chest trauma [11], and multimodality imaging has been useful in trauma-related RCA dissection [12]. In the present case, the ischemic-pattern late gadolinium enhancement followed the RCA territory and contained a central hypoenhanced core consistent with microvascular obstruction. Microvascular obstruction after STEMI is a marker of severe myocardial injury and is associated with adverse outcomes [13]. These findings supported acute/subacute infarction rather than chronic scar or isolated myocardial contusion.

The initial echocardiography report described inferior and lateral hypokinesis, whereas CMR mainly localized the injury to the inferior and inferoseptal segments; the follow-up echocardiography report described posteroinferior hypokinesis. Raw echocardiographic cine loops were unavailable for re-segmentation; the apparent discrepancy likely reflects differences in routine reporting terminology, as well as the greater tissue and segmental specificity of CMR.

This report has several limitations. There was no pre-trauma coronary imaging; the receiving-hospital repeat ECG tracing and original echocardiographic cine loops were unavailable for publication; troponin assay and serial sampling details were incomplete; and serial hematologic and coagulation results and detailed trauma-assessment records could not be fully retrieved. OCT was performed after balloon predilatation, hindering causal interpretation of the observed dissection and plaque morphology. Follow-up was restricted to 1 month. These limitations preclude a definitive conclusion that blunt trauma directly caused the RCA lesion.

Conclusions

Persistent or unexplained chest pain after blunt thoracic trauma should prompt early ECG and troponin testing, even when rib fractures provide an apparent alternative explanation. In the present case, multimodality imaging confirmed an RCA-territory infarction and characterized the culprit lesion, but post-predilatation OCT could not determine whether trauma caused plaque disruption or coronary dissection.

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ChatGPT (OpenAI; GPT-5.6 Sol) was used to assist with English-language editing, structural organization, and preparation of responses during manuscript revision. It was not used to generate or modify clinical data or diagnostic images or to make diagnostic or treatment decisions. All artificial-intelligence-assisted text was critically reviewed and edited by the authors. The authors independently verified the clinical facts, interpretations, and bibliographic references; they take full responsibility for the accuracy and integrity of the final manuscript.

Institution Where the Work Was Performed

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Patient Consent

Written informed consent was obtained from the patient for publication of the case details and accompanying clinical images.

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Figure Authenticity Confirmation

The authors confirm that Figures 1-4 are authentic clinical images from the reported patient. No generative artificial intelligence was used to create or modify any figure. Image processing was limited to cropping, resizing, panel assembly, and the addition or removal of labels and arrows for clarity; these adjustments did not alter the underlying clinical information. No clinical image content was fabricated, duplicated, or selectively altered.