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CASE REPORT: CLINICAL CASE

Simultaneous Acute Myocardial Infarction, Aortic Dissection, Pulmonary Embolism, and Pneumothorax

A Diagnostic and Therapeutic Conundrum

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ABSTRACT

Chest pain is a frequently misdiagnosed presenting symptom, with a broad differential diagnosis that includes critical, time-sensitive cardiopulmonary conditions that necessitate rapid and accurate identification. We present a rare case of concurrent ST-segment elevation myocardial infarction, aortic dissection, pulmonary embolism, and pneumothorax that was successfully managed by a multidisciplinary team. (JACC Case Rep. 2024;29:102610) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 77-year-old African-American man presented following a head-on motor vehicle collision in which the patient was a restrained driver. On arrival to the emergency department, he was alert and in hemodynamically stable condition (blood pressure [BP] 100/80 mmHg, heart rate [HR] 80 beats/min, respiratory rate 20 breaths/min on ambient air), describing new right-sided chest pain without anginal features that improved with analgesics. He was admitted to the intensive care unit for monitoring. Two hours later, the patient experienced acute left-sided crushing chest pain, diaphoresis, and hypotension with mean arterial pressures between 40 and 50 mm Hg, as well as hypoxia requiring supplemental oxygen with high-flow nasal cannula.

LEARNING OBJECTIVES

- This case highlight the importance of maintaining a broad differential diagnosis in any patient presenting with chest pain.
- It may be reasonable to delay door-to-balloon time to rule out aortic pathology if an acute aortic syndrome is suspected in a patient with ST-segment elevations.

MEDICAL HISTORY

His medical history included benign prostatic hyperplasia requiring tamsulosin. He had no previous known cardiovascular history.

DIFFERENTIAL DIAGNOSIS

Given his presentation of chest pain after a motor vehicle accident, a broad differential diagnosis was considered initially focused on trauma-related causes, including rib fractures, aortic dissection, pneumothorax, hemothorax, and hemopericardium. Acute

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ABBREVIATIONS AND ACRONYMS

ACS = acute coronary syndrome

CTA = computed tomography angiography

ECG = electrocardiogram

HR = heart rate

hs-cTnT = high-sensitivity cardiac troponin-T

LAD = left anterior descending coronary artery

PCI = percutaneous coronary intervention

PE = pulmonary embolism

STE = ST-segment elevation

STEMI = ST-segment elevation myocardial infarction

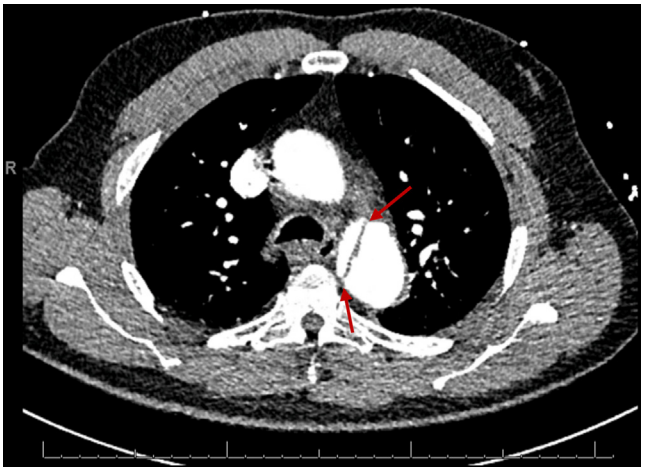
coronary syndrome, acute heart failure (stress-induced cardiomyopathy), and pulmonary embolism were also considered, although they were initially thought to be less consistent with his presentation.

INVESTIGATIONS

Initial laboratory results revealed an upward trending high-sensitivity cardiac troponin-T (hs-cTnT) from 7 to 102 ng/L (normal <22 ng/L for men and <14 ng/L for women, Roche Diagnostics Troponin T Gen 5 assay). A 12-lead electrocardiogram (ECG) showed <2 mm ST-segment elevations (STE) in leads V_3-V_6 that did not meet the criteria for ST elevation myocardial infarction (STEMI). A chest x-ray demonstrated fractures of the left second through eighth ribs and right fifth through eighth ribs. A computed tomography angiogram (CTA) of the chest showed a medial left apical pneumothorax as well as a descending thoracic aortic dissection distal to the left subclavian artery Stanford type B (Figure 1). An echocardiogram showed a left ventricular ejection fraction of 65% without regional wall motion abnormalities, normal right ventricular function, and no evidence of a pericardial effusion.

After the patient experienced acute left sided chest pain with hypotension and hypoxia, repeated workup revealed an ECG with prominent STE in leads V_3-V_6 (Figure 2) and a sharp rise in hs-cTnT to 1,722. Given the concurrent aortic dissection—a relative contraindication for anticoagulation—repeated chest CTA was obtained to evaluate for progression. Imaging showed a stable aortic dissection but demonstrated an acute left upper lobe subsegmental pulmonary embolism (PE) (Figure 3).

FIGURE 1 Computed Tomography Angiogram of the Chest Showing Stanford Type B Aortic Dissection



MANAGEMENT

After a risk-benefit discussion and shared decision making by a multidisciplinary team including interventional cardiologists, vascular surgeons, radiologists, and cardiothoracic surgeons, the patient was administered aspirin and given a continuous heparin infusion for concurrent (STEMI) and PE. Left heart catheterization with coronary angiography was subsequently performed and showed a 100% thrombotic occlusion in the mid left anterior descending artery (LAD) (Figure 4). The lesion was predilated with a 2.0 × 12 Trek Balloon (Abbott Vascular) and post-dilated with a 3.5 × 8 NC Trek Balloon (Abbott Vascular). A 2.5 mm × 38 mm Xience Skypoint drug-eluting stent (Abbott Vascular) was placed in the LAD, resulting in TIMI flow grade III after percutaneous coronary intervention (PCI). Following PCI, he was given 600 mg oral clopidogrel and 80 mg atorvastatin. Inasmuch as the dissection was Stanford type B, the vascular surgery team elected for conservative management with BP and HR control. For treatment of the apical pneumothorax, a chest tube was inserted for low-pressure suction. The chest tube was removed 1 week later after resolution of the pneumothorax. Repeated CTA of the chest showed a stable aortic dissection.

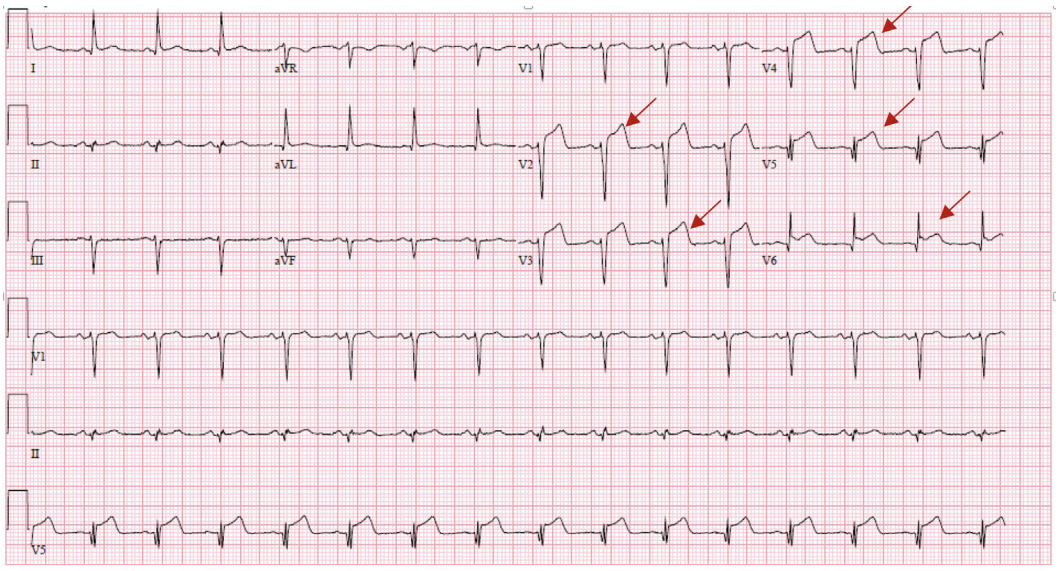
OUTCOME AND FOLLOW-UP

The patient had a 15-day hospital stay and was discharged to cardiac rehabilitation and then subsequently home. He continues to receive follow-up care with a primary physician and a cardiologist at an outside hospital.

DISCUSSION

Chest pain is the second most common reason for adults to present to the emergency department in the United States after injuries.[1] Acute coronary syndrome (ACS), PE, and aortic dissection can all result in chest pain, hence ≥1 of these conditions may be missed during initial work-up. In this case, initial evaluation was confounded by presentation after a motor vehicle accident, and his chest pain was initially thought to be secondary to rib fractures. Although most patients presenting with chest pain have a noncardiac cause, a thorough history is necessary to narrow the differential diagnosis and avoid missing critical conditions.[1] A focused

FIGURE 2 12-Lead Electrocardiogram Showing ST-Segment Elevations in Leads V_2-V_6



cardiovascular examination is not always reliable but may provide clues to the cause of chest pain. The American Heart Association and the American College of Cardiology have outlined an algorithm for evaluating chest pain. In summary, an ECG is recommended in any patient presenting with chest pain without an obvious noncardiac cause. Further testing should be guided by pretest probability.[1-3] For example, tachycardia and dyspnea occur in about 90% of patients with PE, whereas pulse differential with widened mediastinum has an 80% probability of aortic dissection.[1] In our case, the initial ECG was without STE, and hs-cTnT levels were below the 99th percentile, which lowered the probability of ACS. Serial monitoring of ECGs and troponin eventually led to the diagnosis of a STEMI. Although the chest x-ray did not show a widened mediastinum, aortic dissection was still high on the differential, given the history of trauma. Type B dissections are less likely to cause hemodynamic changes or widened mediastinum on chest x-ray than are type A dissections.[4] In most cases including ours, patients who have >1 cardiac cause of chest pain, ≥1 cause is usually an incidental finding.[5] Acute PEs were incidentally discovered during evaluation for aortic pathologic changes.

ACS and PE typically require anticoagulation for management, whereas anticoagulation is

FIGURE 3 Computed Tomography Angiogram of the Chest Showing Subsegmental Pulmonary Emboli

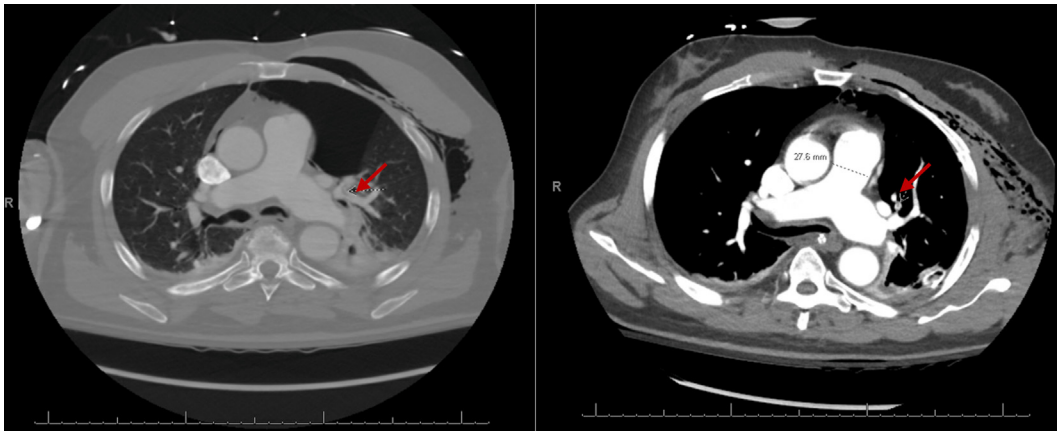
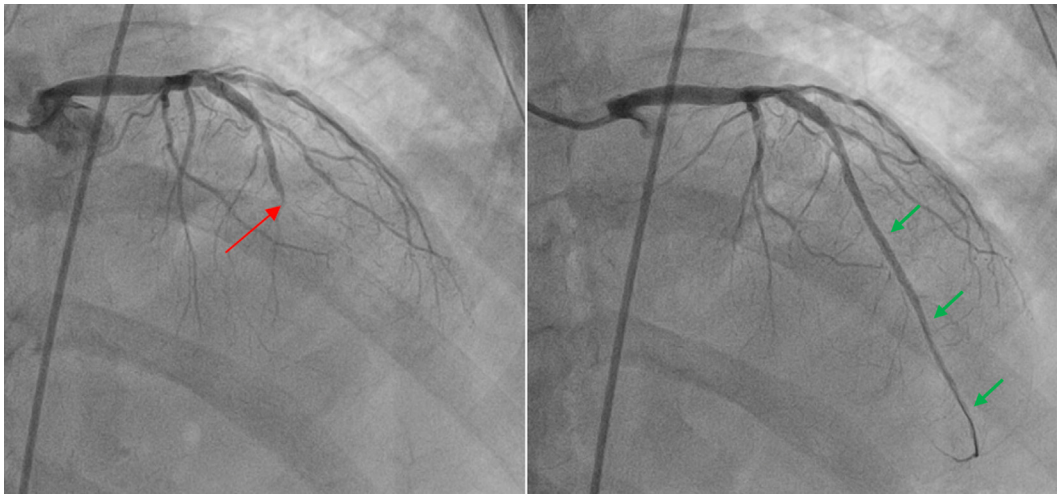


FIGURE 4 Coronary Angiograms Showing Complete Occlusion of the LAD and Revascularization of the LAD After Percutaneous Coronary Intervention



contraindicated in aortic dissection.[2,6,7] When each of these conditions present in an isolated manner, management is clearly guided by well-established guidelines.[1-3,8] However, when ≥2 co-exist, there is often a diagnostic and therapeutic challenge. Type A aortic dissections (involving the ascending aorta) often require surgical interventions, whereas type B dissections can be managed conservatively if uncomplicated or endovascularly if complicated.[2,4] Type A dissections are more challenging to diagnose and manage because they may mimic ACS if the dissection flap extends to the ostium of the coronary artery.[9] Clinicians are often under pressure to achieve a door-to-balloon time <90 minutes when faced with a patient with STEMI and may overlook other possible causes of chest pain. If clinical suspicion for aortic pathologic changes in a patient presenting with STEMI is high, it may be reasonable to delay door-to-balloon time to rule out a dissection, inasmuch as misdiagnosis can lead to catastrophic consequences, especially in non-PCI-capable centers where thrombolytics are used to manage ACS.[8-10] If an aortic dissection is missed prior to PCI, difficulty with catheter engagement or significant aortic regurgitation during PCI should raise the suspicion for acute type A aortic dissection.[9] The role of a multidisciplinary approach for treating patients with concurrent aortic dissection, PE, and ACS should be emphasized. These teams often involve cardiologists, interventional cardiologists, cardiac surgeons, vascular surgeons, radiologists, interventional radiologists, and anesthesiologists. Shared decision making with these teams is often necessary to decide the optimal treatment modality: open surgery, vascular and endovascular procedures, or conservative management.[2] In our case, the patient had a stable type B aortic dissection, and a shared decision was made to pursue conservative management.

CONCLUSIONS

This case highlights the importance of maintaining a high index of suspicion for aortic dissection in patients presenting with chest pain, even in the setting of STE. A thorough clinical evaluation and appropriate use of advanced imaging techniques are crucial for prompt diagnosis and optimal management of these life-threatening conditions. It may be reasonable to delay door-to-balloon time to rule out aortic pathologic changes if an acute aortic syndrome is suspected in a patient with ST segment elevations. The complex interplay between the underlying pathologic conditions in this case further highlights the need for a multidisciplinary approach to patient care.

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