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INTERMEDIATE

CASE REPORT: CLINICAL CASE

Early Recanalization of a Traumatic Coronary Artery Dissection With Medical Therapy Alone

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ABSTRACT

We describe a patient who presents with chest discomfort 30 h after having an accident with an all-terrain vehicle. His follow-up coronary computed tomography angiogram revealed early recanalization of his coronary artery with conservative medical therapy. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:2299–303) © 2020 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/).

HISTORY OF PRESENTATION

A 33-year-old man was admitted to the trauma service after a high-speed motor vehicle collision. He had intimal injuries in the anterior wall of the descending thoracic aorta (**Figure 1**) and right subclavian arteries that were managed conservatively. He was safely discharged home with interval resolution of his injuries on follow-up imaging.

Seven months later, the patient presented to the emergency department of a non-percutaneous coronary intervention (PCI)-capable center for new-onset chest discomfort after having a second motor vehicle accident. He was driving a four-wheel all-terrain vehicle when he hit an unknown object, flew over the handlebars, and woke up with the vehicle on top of him. The following day (~30 h later), he experienced new-onset chest discomfort radiating to his left arm that prompted him to seek medical attention.

In the emergency department, the patient was hemodynamically stable, and physical examination was significant for excoriations on his legs with an unremarkable cardiopulmonary examination. The electrocardiogram (ECG) showed normal sinus rhythm with tall T waves in leads V2 to V4 and ST-segment elevations in V_4 (**Figure 2A**). Troponin level was 0.01 ng/ml. Our center received a transfer request for possible emergent catheterization.

LEARNING OBJECTIVES

- To recognize traumatic coronary artery dissection as a rare but life-threatening condition.
- To review the management of coronary artery dissection and decision-making in the catheterization laboratory.
- To show the effectiveness of medical therapy in the treatment of traumatic coronary artery dissection and the utility of coronary CT imaging for re-evaluation of coronary anatomy.

MEDICAL HISTORY

The patient's medical history included multiple rib fractures, right temporomandibular dislocation, and a splenic laceration associated with the previous motor

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ABBREVIATIONS AND ACRONYMS

CT = computed tomography

ECG = electrocardiogram

LAD = left anterior descending artery

PCI = percutaneous coronary intervention

TCAD = traumatic coronary artery dissection

vehicle accident. He denied personal or family history of cardiovascular disease, early coronary artery disease, or sudden cardiac death.

DIFFERENTIAL DIAGNOSIS

Based on the patient's history of vascular injury 7 months earlier, aortic dissection was on the initial differential diagnosis. Cardiac contusion, hemopericardium, coronary artery dissection, and type 1 acute myocardial infarction were also considered.

INVESTIGATIONS

A chest computed tomography (CT) angiogram in the non-PCI-capable center demonstrated no aortic dissection, with complete resolution of the prior vascular injuries.

MANAGEMENT

En route, the patient received aspirin, ticagrelor, unfractionated heparin, metoprolol tartrate, and morphine. Upon arrival to the PCI-capable center, he was free of chest pain and was hemodynamically stable. ECG showed tall T waves and worsening ST-segment elevations in V_3 to V_4 (**Figure 2B**). He was immediately taken to the cardiac catheterization laboratory. The left anterior descending artery (LAD) was occluded at the ostium flush with findings believed to be consistent with coronary dissection (linear densities) and areas with apparent thrombosis (Videos 1 and 2). The left circumflex artery and right coronary artery were widely patent, with no significant luminal irregularities. There were right to left collaterals that do not fill the distal LAD.

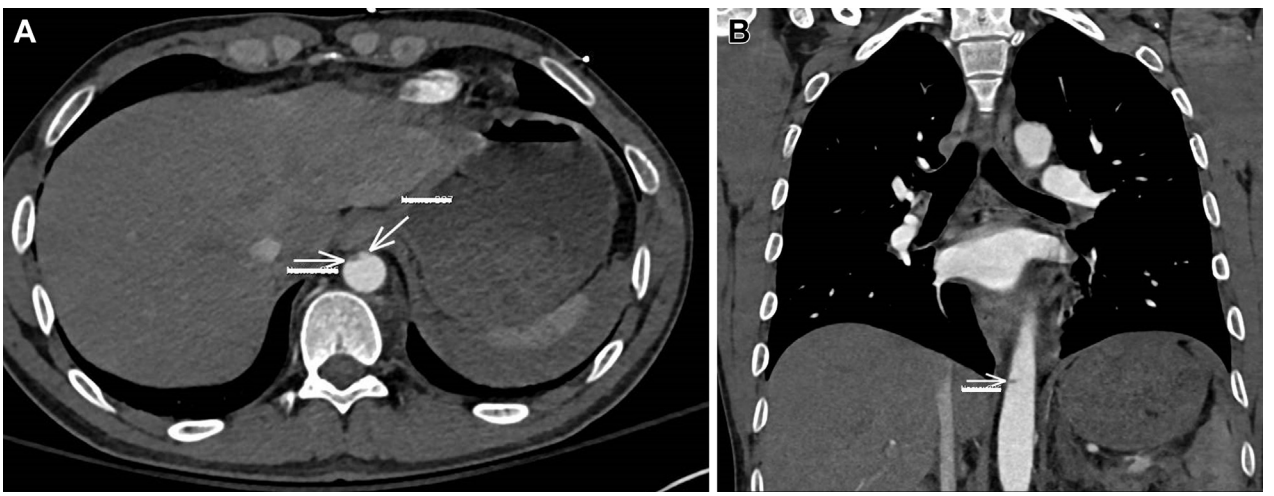
In the PCI-capable facility, the patient's ECG showed worsening ST-segment elevations in V_3 to V_4; however, he remained hemodynamically stable, and chest pressure had resolved. Careful analysis of his coronary angiogram raised concerns for a complex traumatic dissection of the ostium of the LAD of unknown duration with associated thrombosis. There were no significant collaterals filling the distal LAD. The coronaries were not further instrumented, and no PCI was performed because of concerns that wiring the LAD could compromise the left main artery if a false dissection lumen was entered. Intravenous heparin was discontinued to reduce the risk of worsening dissection.

In the coronary care unit, the patient's troponin levels peaked at 33.39 ng/ml. Basic metabolic panel and blood counts were within normal ranges. A transthoracic echocardiogram revealed a left ventricular ejection fraction of 40% to 45% with a hypokinetic anteroseptal wall in the mid to apical segments (Videos 3 and 4).

On hospital day 3, the patient was discharged home on aspirin, ticagrelor, metoprolol succinate 150 mg, lisinopril 2.5 mg, and atorvastatin 80 mg with close follow-up in the post-myocardial infarction clinic.

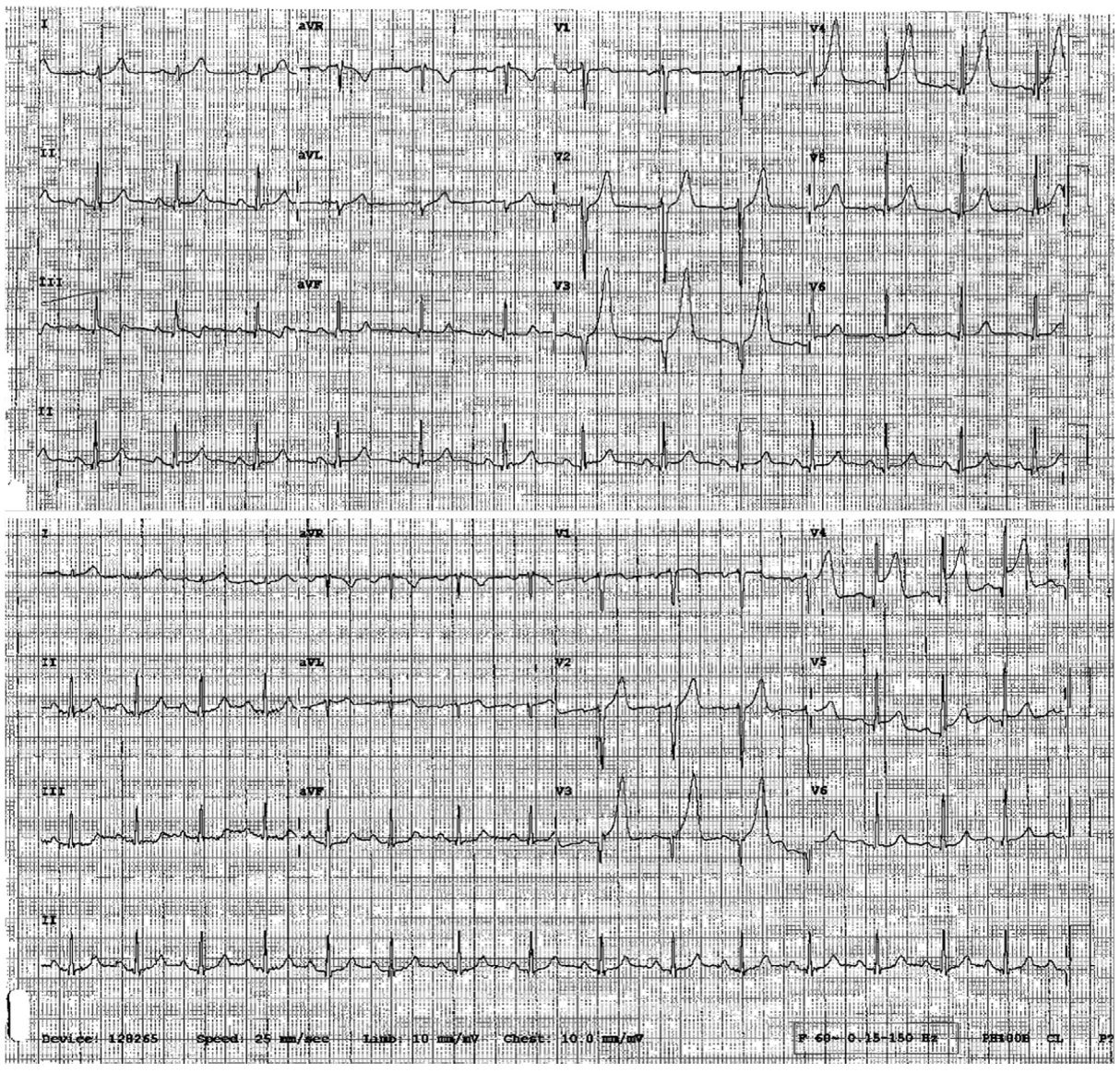
Six days after discharge, the patient returned to the emergency department for evaluation of sharp pain in

FIGURE 1 Intimal Injury to the Thoracic Aorta



Intimal injury of the thoracic aorta in the axial **(A)** and coronal **(B)** planes.

FIGURE 2 Electrocardiographic Findings at Presentation and Prior to Coronary Angiogram



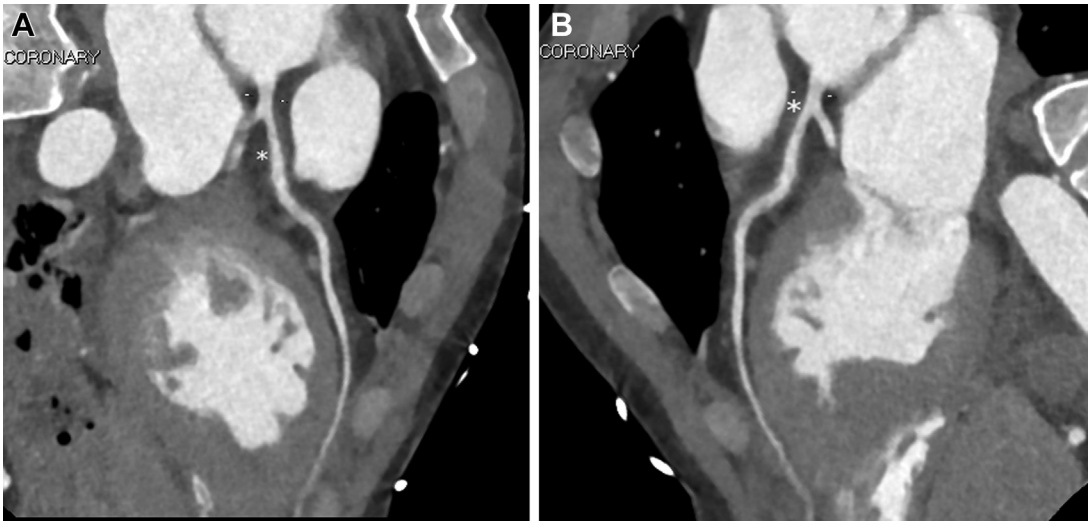
(A) Initial electrocardiogram and **(B)** repeat electrocardiogram in percutaneous coronary intervention–capable facility.

his left chest that was pleuritic in nature. A coronary CT angiogram showed that his LAD was completely patent, demonstrating resolution of the dissection flaps after 9 days of medical therapy (Figures 3A and 3B). Nonetheless, there were ground-glass opacities at the lung bases and patchy opacities in the lingula with a small left-sided pleural effusion. Transthoracic echocardiogram revealed a large mobile echodensity attached in the left ventricular apex consistent with thrombus. Aspirin was discontinued, and warfarin was started. Antibiotics and low-molecular-weight heparin were started as a bridge until the patient's international normalized ratio reached therapeutic range.

DISCUSSION

There are no society guidelines on the management of traumatic coronary artery dissection (TCAD), and most evidence is derived from case reports and case series. An extrapolation from spontaneous coronary artery dissection guidelines might be

FIGURE 3 Curved Multiplanar Reformatted Images of Left Anterior Descending Artery



(A) The left anterior descending artery is patent with minimal wall thickening. **(B)** *There is interstitial edema surrounding the proximal LAD that is also seen in the orthogonal view.

considered, recognizing that the pathophysiology, outcomes, and prognosis of TCAD could be different.

In a TCAD, the LAD is involved in ~76% of the cases due to its anatomic location, followed by the right coronary artery and the left circumflex artery (1). The clinical presentation can vary significantly depending on the severity of trauma. The classic chest pressure or tightness may appear several hours or even days after the trauma (2). This is an important feature that was present in our patient.

The dynamic ECG changes seen in this patient, starting with hyperacute T waves in the distribution of the affected vessel, followed by ST-segment elevations (**Figures 1 and 2**), have also been described by Leong and Brown (3). The mechanisms of dissection in TCAD include intimal tearing with propagation to the media, platelet activation and aggregation, thrombosis, and vasospasm (4). In terms of the mechanism of injury, TCAD can occur after a wide variety of traumatic mechanisms that involve blunt trauma to the chest, motor vehicle accidents with and without airbag deployment (5,6), and sports-related injuries. The management of this condition can range from conservative medical management in stable and asymptomatic patients, to PCI with balloon angioplasty, stent placement, and coronary artery bypass grafting (5).

The rapid recanalization of the LAD in our case may be due to spontaneous healing, early implementation of antiplatelet therapy, and anti-inflammatory effects of statins. To our knowledge, this case is the only one described in the literature with complete resolution of dissection with medical therapy alone for 9 days.

FOLLOW-UP

The patient followed up in our cardiology clinic and reported complete resolution of symptoms. He denied decreased exercise tolerance, nocturnal paroxysmal dyspnea, or edema. He quit smoking and has had no alcohol since the accident. His convalescent ECG showed normal sinus rhythm with T-wave inversions on V3 to V6 and a late transition of R-wave progression. A repeat transthoracic echocardiogram is planned with his next visit in 3 months.

CONCLUSIONS

Acute myocardial infarction after blunt chest trauma is rare, but it carries high mortality and morbidity. A heightened level of suspicion is mandatory for recognizing the condition early. Medical therapy alone may be a reasonable strategy in patients with electrical and hemodynamic stability who are free of chest pain. Coronary CT angiogram could represent the imaging modality of choice in patients with

traumatic coronary artery dissection if follow-up imaging to assess for coronary patency is needed.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS blunt chest trauma, cardiac contusion, catheterization

APPENDIX For supplemental videos, please see the online version of the paper.