

Traumatic Coronary Artery Dissection as A Potential Cause of Acute Myocardial Infraction in Motorcycle Accident

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Abstract

Background: Chest pain in blunt chest trauma can be caused by various intrathoracic injuries. Pneumothorax, hemothorax, and rib fractures are commonly seen in the emergency department. Although cardiac involvement is very rare, the probability should not be excluded.

Case Illustration: A 31-years-old male who complained of chest pain and diaphoresis was brought to the emergency department after a high-speed motorcycle collision. Chest X-ray revealed no abnormality but a 12-lead Electrocardiogram (ECG) demonstrated ST-segment elevation in lead I, AvL, V2-6, and atrial fibrillation. Because of the unusual presentation, the decision was to proceed with percutaneous coronary intervention (PCI). Coronary Angiography detected a thrombus at proximal LAD and spiral dissection at mid LAD (TIMI 2 Flow). After the procedure, he was transferred to the High Care Unit.

Conclusion: Following blunt chest trauma, chest pain in the setting of a vehicle collision can be caused by dissection of the coronary artery. Prompt cardiac workup (ECG, cardiac enzyme, and echocardiography) must be done in a highly suspected patient.

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(Indonesian J Cardiol. 2022;43:23-29)

Keywords: Blunt Chest Trauma, Traumatic Coronary Artery Dissection, Atrial Fibrillation, Motorcycle Collision.

Background

Chest pain in blunt chest trauma can be caused by various intrathoracic injuries. Pneumothorax, hemothorax, and rib fractures are commonly seen in the emergency department. Although cardiac involvement is very rare, it shall not be excluded. Traumatic coronary artery dissection can be one of the rare causes of chest pain in the setting of trauma. It is an uncommon event and its prevalence or incidence was merely described in case reports of around 0.1%.¹ In this case, we report a rare event of coronary artery dissection with secondary acute myocardial infarction after a motorcycle accident.

Case Illustrations

A 31-years-old male who complained of chest pain and diaphoresis was brought to the emergency department after a high-speed motorcycle collision one hour earlier. His past medical history was unremarkable. At presentation, he was hemodynamically stable with an irregular heartbeat. Physical examination revealed normal chest wall excursion, normal vesicular breath sounds of both lungs, and small bruises on the upper part of the sternum. Chest X-ray revealed no abnormality but a 12-lead Electrocardiogram (ECG) demonstrated ST-segment elevation in lead I, AvL, V2-6, and atrial fibrillation (**Figure 1**). Troponin I was found to be elevated at 1,05 ng/ml. Bedside echocardiography confirmed typical anterior wall hypokinesia and reduced ejection fraction (42%) (**Figure 2**).

Because of the unusual presentation (Young male [31-year-old] with normal Body Mass Index ; There weren't any history of hypertension, Diabetes, dyslipidemia, family history of premature CAD. The symptoms was developed after trauma), the decision was to proceed with percutaneous coronary intervention (PCI). Before the procedure, he was given ticagrelor and nitrate. Atrial fibrillation (presumably new-onset) was terminated by intravenous amiodarone. Coronary Angiography (CAG) detected a thrombus at proximal Left Anterior Descending artery (LAD) and spiral dissection at mid LAD (TIMI 2 Flow) (**Figure 3**). Cannulation of the left coronary artery was attempted with JL 3.5/6F guiding catheter. After that,

Sion Blue Wire was used but failed to cross the lesion at mid LAD (entry into false lumen). We stopped the procedure. There were risk of extending of dissection or coronary perforation if we continue the procedure. After the procedure, he was transferred to the High Care Unit. He was then treated conservatively on aspirin, ticagrelor, enoxaparin, pantoprazole, atorvastatin, morphine, bisoprolol, ramipril, and nitrate. Follow-up echocardiography on the 4th day week after admission showed 4 mm pericardial effusion, particularly in the anterior area. On the 5th day of hospitalization, he was asymptomatic with ECG showed resolution of ST-segment (> 50%) with well-formed Q waves (**Figure 4**) and minimal pericardial effusion based on echocardiography. He was discharged with additional colchicine plus ibuprofen for 2 weeks. After one month the patient was clinically stable and no remarkable complaint with LVEF of 45% and resolved pericardial effusion.

Discussion

TAcute myocardial infarction as a result of coronary artery dissection is caused by intimal disruption which produces lumen narrowing and the formation of an intramural hematoma. This condition is quite uncommon and usually caused by atherosclerosis, systemic arteriopathies, iatrogenic, and trauma.¹ In this patient, intimal tearing due to shearing of the coronary vessel wall is probably related to the acceleration/deceleration forces at a relatively weak junction of the coronary vessel. This mechanism is thought of as the underlying pathophysiology of traumatic coronary artery dissection in the setting of blunt chest trauma.² This condition is different from Spontaneous Coronary Artery Dissection (SCAD) which is non-iatrogenic and non-traumatic, that is relatively rare in males (0.07:0.6 compared to female). Underlying arteriopathy in SCAD poses the coronary arterial wall vulnerable to spontaneous tear. Furthermore, extreme physical and emotional stresses were potential triggers or precipitants which cause artery wall dissection in SCAD.¹

Traumatic coronary artery dissection's prevalence or incidence was merely described in case reports. Based on a recent case series, there were only 24 patients in 22 case reports over 10 years.² Recent literature describes that traumatic coronary artery dissection can occur

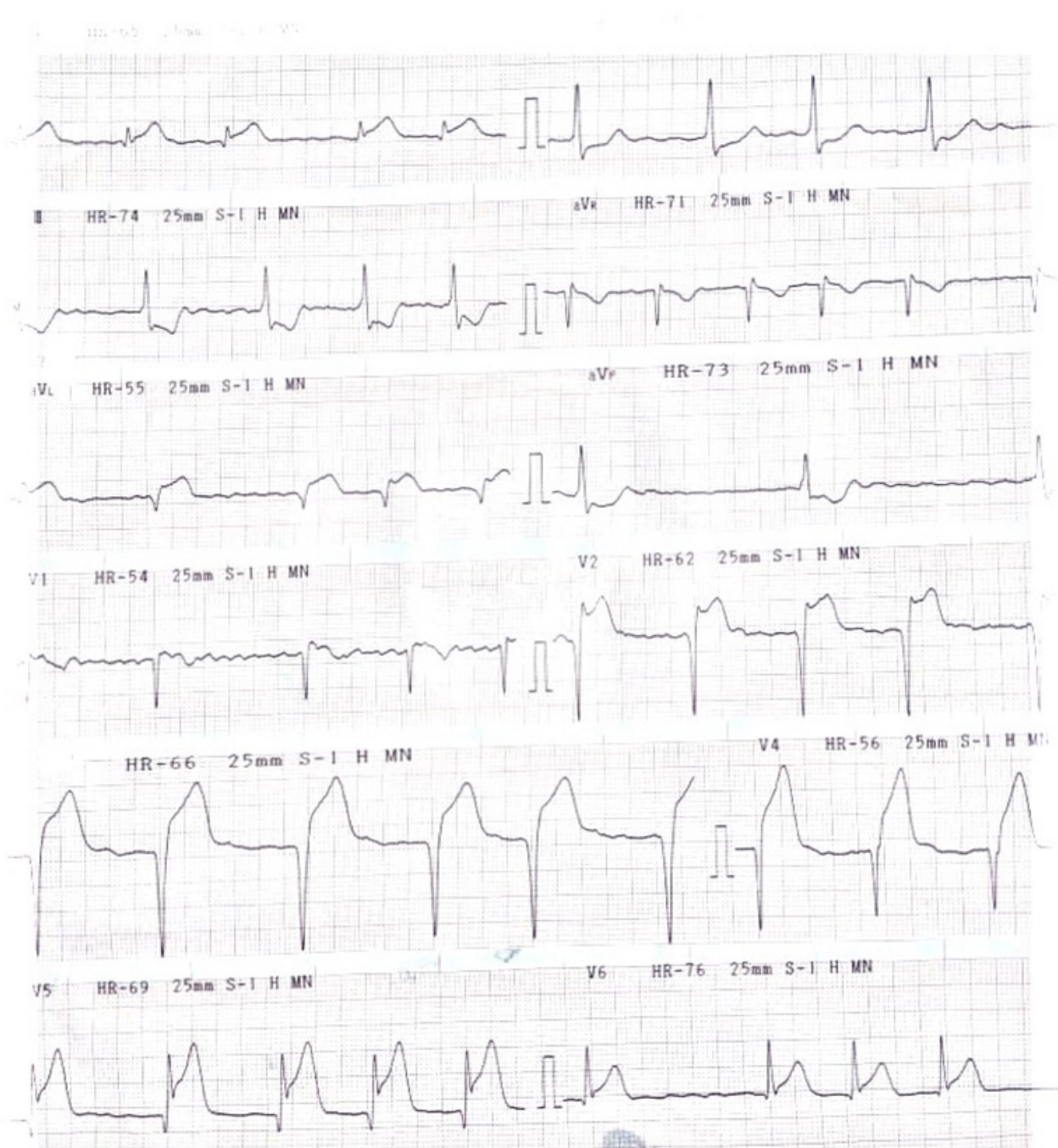


Figure 1. 12-Lead ECG at Presentation.

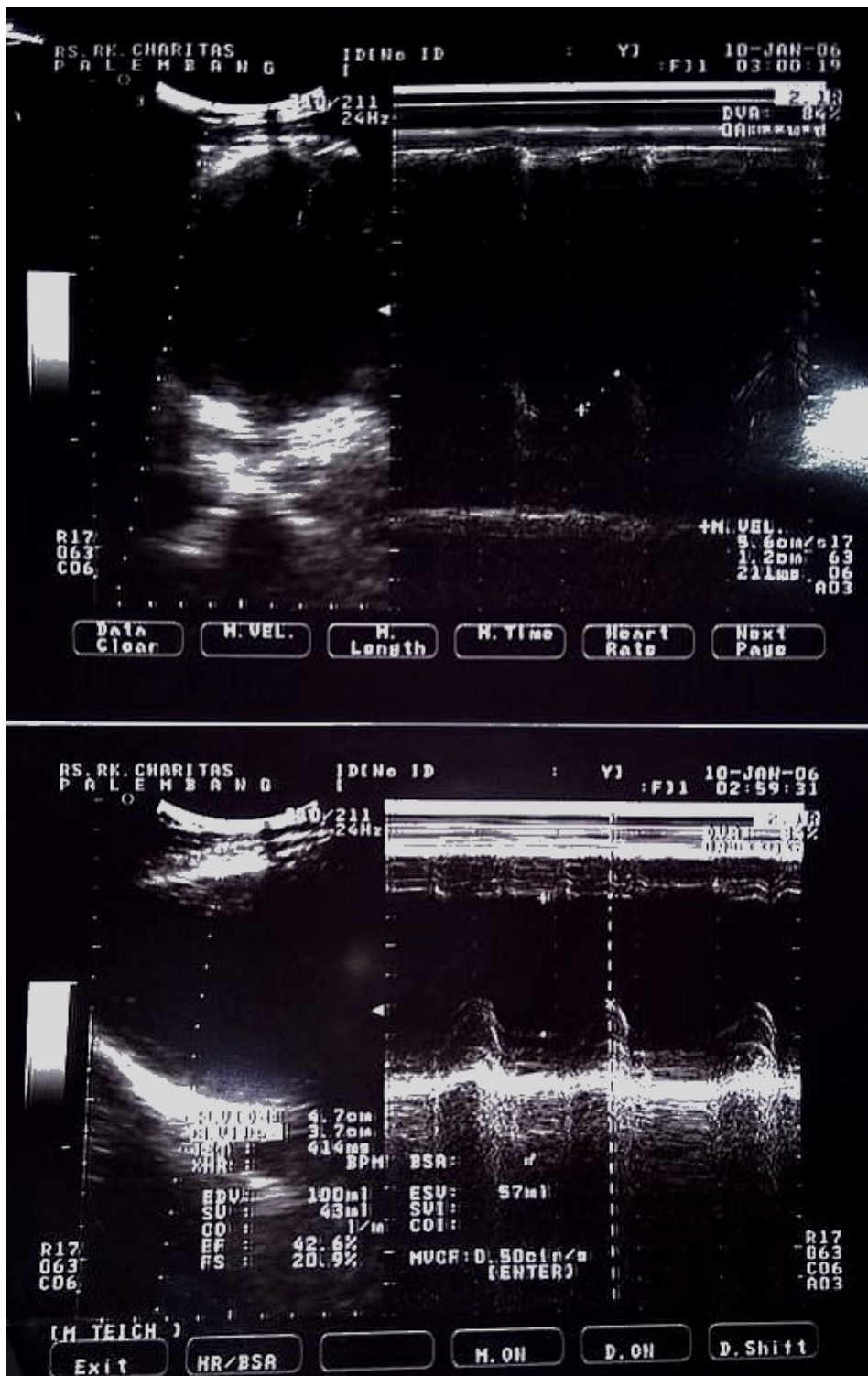


Figure 2. Bedside Echocardiography at Presentation.

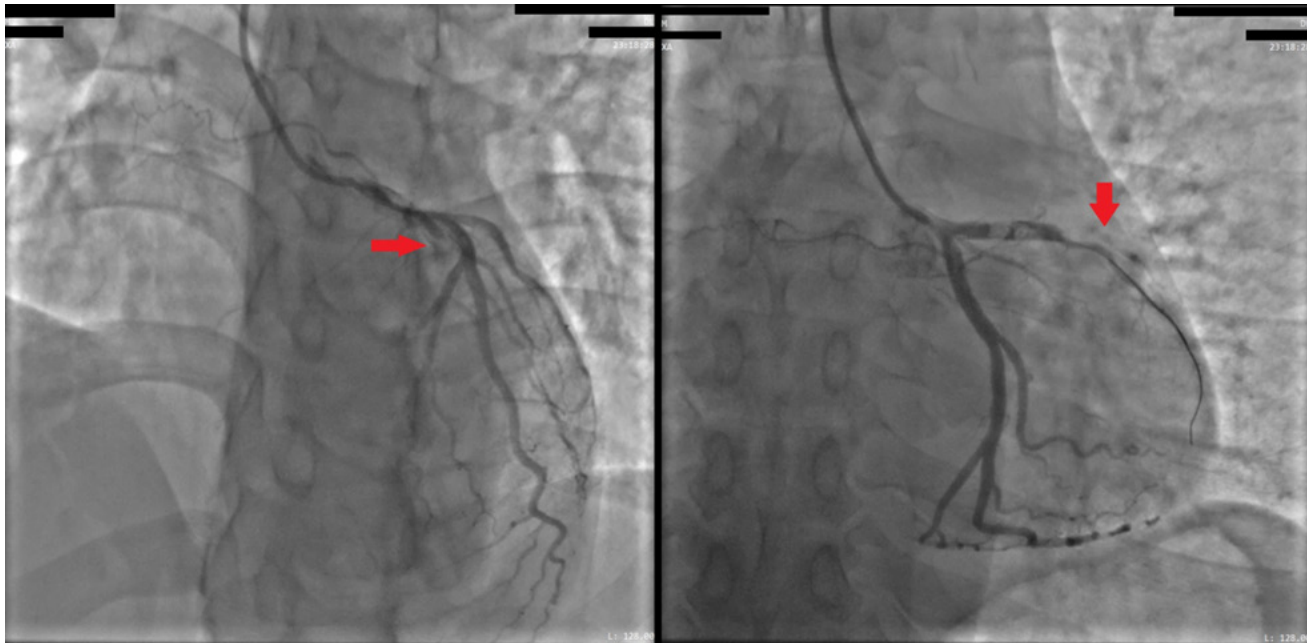


Figure 3. CAG Reveals Spiral Dissection at Mid LAD (Red Arrow).

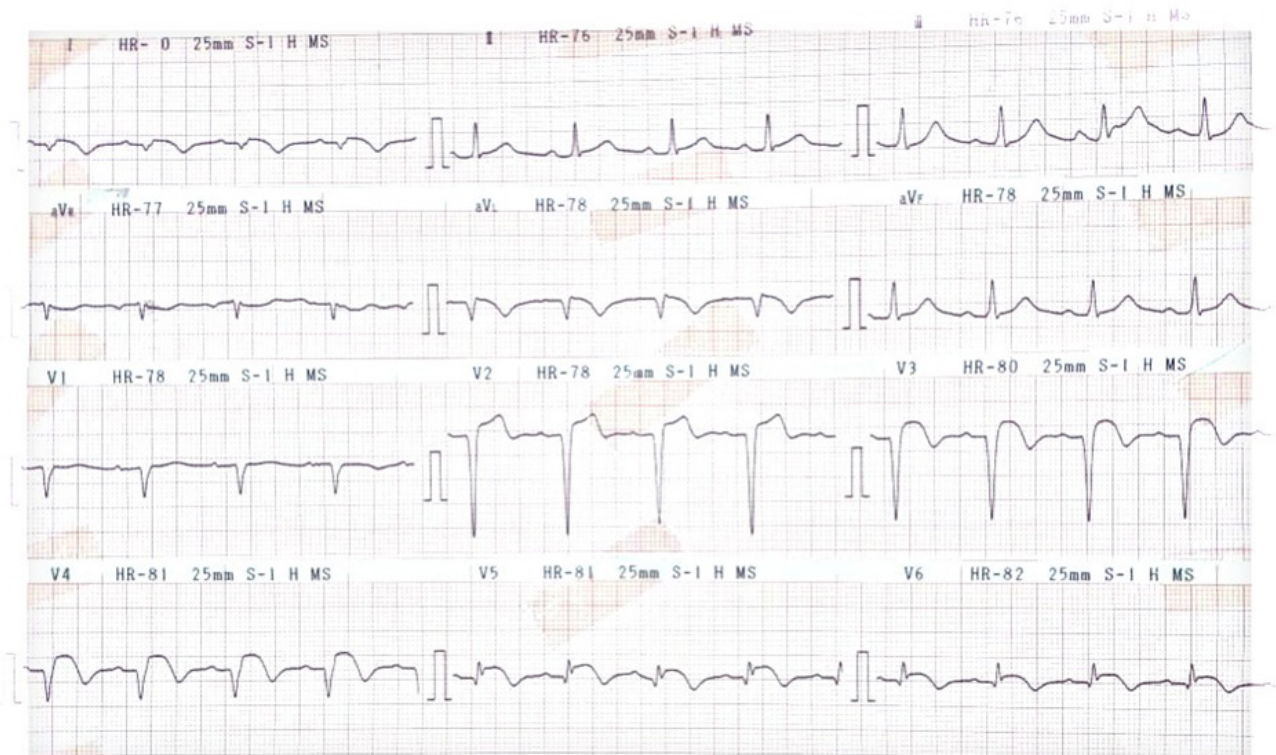


Figure 4. 12-Lead ECG on the 5th Day of Hospitalization.

after a direct blow to the chest wall in the setting of both vehicle collision^{2,3,4} and sports-related injury.⁵ The most commonly affected vessel is the Left Anterior Descending (LAD) artery (76 % of cases) because its anatomic location is directly exposed to blunt chest trauma.⁶

Chest pain is frequently observed in coronary artery dissection.¹ If it occurs after blunt chest trauma, other organs in the thoracic cavity would be a culprit. A 12-lead ECG, cardiac enzymes, and/or echocardiography at admission are usually used to screen for the probability of cardiac involvement. If the patient is highly suspected, coronary angiography can establish the diagnosis of coronary artery dissection. Optical Coherence Tomography (OCT) and Intravascular Ultrasound (IVUS) can also help to point toward dissection in the vessel's segment and confirm the stent position where necessary.⁵ The ECG can also help to confirm what type of arrhythmia is associated with current cardiac injury.⁷

Cardiac arrhythmias as a complication of blunt chest trauma have been reported in a case-control study. Younger patients (<50 years old) had a fourfold increase in the risk of arrhythmias.⁷ The most common arrhythmias reported is atrial fibrillation. It is usually seen as a result of a cardiac injury.² Atrial fibrillation which spontaneously resolved has also been reported in association with chest trauma in adult and youth.^{8,9} In our case, atrial fibrillation was successfully converted to sinus rhythm by intravenous amiodarone.

Pericardial effusion can result from inflammation of the pericardium (pericarditis) in response to illness or injury. Pericardial effusion can also occur when the flow of pericardial fluid is blocked or when blood collects within the pericardium, such as from chest trauma. Depending on how quickly pericardial effusion develops, the pericardium can stretch somewhat to accommodate the excess fluid to a certain limit. If the fluid keeps accumulating it would lead to cardiac tamponade.¹⁰ Our patient was clinically stable without signs of tamponade and was successfully treated for two weeks with medication, i.e. colchicine, an ancient drug with unique anti-inflammatory effects and a good long-term safety profile.² In this case, pericardial effusion may be due to injury response of blunt chest trauma (late presentation), STEMI complication, or both.

Aggressive management is required after coronary artery dissection is detected. Treatment options for

this condition include PCI, coronary artery bypass grafting, and conservative management.² Recent cases report high mortality from coronary artery dissection following a vehicle collision despite aggressive PCI.^{3,4} In this case, there wasn't any history of chest pain before the motorcycle accident. The patient has a low risk of atherosclerosis (young male with normal Body Mass Index (BMI) and without any history of hypertension, diabetes, dyslipidemia, or family history of premature CAD). However, to establish a definite diagnosis of traumatic coronary artery dissection, we decided to proceed with PCI. But due to the unfavorable anatomy of the lesion (the dissection flap prevented the wire to cross the lesion because of wire entry into the false lumen), we deferred the procedure and manage the patients conservatively with close monitoring of both hemodynamics and symptoms. The evidence is still lacking on which approach (revascularization vs conservative therapy) is preferable for traumatic coronary artery dissection. As in SCAD, the symptoms are mainly caused by dissection flap obstructing and reducing the flow of coronary vessels. Based on a retrospective study by Tweet et al (2014) on SCAD, high uneventful rates in-hospital course (90%) was reported in the conservative group, meanwhile a high failure rate (53%) was noted in the PCI group. There were also no differences in revascularization vs conservative therapy regarding recurrent SCAD and 5-year rates of target vessel revascularization. In this study, failure of PCI because of wire entry into false lumen was 7 out of 23.¹¹ Our patient was discharged from the hospital on the fifth day in stable condition with no significant symptom. Eventually, coronary CT angiogram can be considered as the imaging modality of choice to follow up patients with traumatic coronary dissection and re-evaluate coronary patency.¹²

Conclusions

Following blunt chest trauma, chest pain in the setting of a vehicle collision can be caused by dissection of the coronary artery. Prompt cardiac workup (ECG, cardiac enzyme, and echocardiography) must be done in a highly suspected patient. Immediate coronary angiography is critical to determine the diagnosis and treatment for a favorable outcome in patients with traumatic coronary artery dissection.

Conflict of Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

Acknowledgment

We express our gratitude to RK Charitas Hospital Palembang and those who have been very supportive in the publication process of this case report.

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