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MINI-FOCUS ISSUE: CHEST WOUNDS

INTERMEDIATE

CASE REPORT: CLINICAL CASE

An Uncommon Cause of ST-Segment Elevation Myocardial Infarction

Intramural Coronary Artery Hematoma After Blunt Chest Trauma

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ABSTRACT

Intramural coronary hematoma (IMCH) is a rare cause of acute myocardial infarction (MI). We aim to review the current knowledge and share our experience with the diagnosis and management of a patient presenting with traumatic IMCH leading to an acute ST-segment elevation MI. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:2304–9)

HISTORY OF PRESENTATION

The patient was a 71-year-old man with chest pain (CP) of 9 h of evolution. Four days prior he had a motor vehicle accident where he was propelled from his motorcycle after crashing into a car at 35 mph resulting in blunt chest trauma (BCT). Evaluation at an outside institution included a whole-body computed tomography scan that revealed no major organ damages and he was discharged on tramadol with instructions to rest.

Since the accident, he reported having pleuritic-like CP that was gradually improving. However, 9 h prior to presentation, the CP became sustained and progressively worse in intensity, which prompted him to seek medical attention.

Upon arrival to our emergency department, he was hemodynamically stable (blood pressure 137/85 mm Hg, heart rate 88 beats/min), afebrile (36.9°C), and not hypoxic (oxygen saturation 97%). His chest wall had various ecchymoses and was tender to palpation, but this pain was somewhat different from the one that brought him to the emergency department. His lungs were clear and no heart murmurs were detected. His extremities were warm with symmetric pulses and no edema.

LEARNING OBJECTIVES

- IMCH is subset of coronary artery dissection and both are rare causes of MI.
- Intracoronary imaging is confirmatory and useful to guide PCI.
- Conservative management may be preferred unless ongoing ischemia, ventricular arrhythmia, or shock warrants PCI.
- Physicians caring for patients after BCT should be aware of the possibility of MI as a treatable associated complication if suspected and diagnosed early.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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PAST MEDICAL HISTORY

Medical history was pertinent for hypertension, dyslipidemia, and type 2 diabetes mellitus. He was a non-alcohol user and denied smoking or illicit drug use.

INVESTIGATIONS

His electrocardiogram (ECG) revealed ST-segment elevations (>1.5 mm) in leads V_2 to V_5 with Q-waves suggestive of extensive anterolateral ST-segment elevation myocardial infarction (STEMI) (**Figure 1**). High-sensitive troponins were in increasing trend (1,844 to 1,969 ng/l) as well as elevated pro-B-type natriuretic peptide (1,168 pg/ml) and D-dimers (8.95 μg/ml). Limited bedside echocardiogram revealed apical-anterior hypokinesis and no pericardial effusion. Routine laboratory tests and chest x-ray were unremarkable (**Figure 2**).

DIFFERENTIAL DIAGNOSIS

CP associated with ST-segment elevations after BCT is concerning for atherothrombosis-related myocardial infarction (MI), aortic dissection, cardiac contusion, or coronary artery dissection/ intramural coronary hematoma (IMCH).

MANAGEMENT

After BCT, discerning CP from trauma or ischemia and establishing the onset of a suspected STEMI can be challenging. Additionally, anticoagulant/antiplatelet agents may precipitate internal bleeding (1). However, based on an estimated 9-h duration of new sustained CP and ECG changes consistent with an acute STEMI, emergent coronary angiography was recommended. Loading dose of dual antiplatelet therapy (DAPT) and intravenous heparin were administered. Coronary angiography showed a subtotal occlusion of the mid left anterior descending artery (LAD) with slow Thrombolysis In Myocardial Infarction (TIMI) flow grade 1 (**Figure 3A**, Video 1). After advancing the guidewire freely into the apical LAD segment, the absence of antegrade flow prompted confirmation of intraluminal position with distal injection via a dual-lumen catheter, after which aspiration thrombectomy and intracoronary nitroglycerin administration were performed (door-to-device time: 72 min). Angiography then showed a persistent long segment of luminal narrowing with a smooth contour distal to the atherothrombotic-appearing culprit lesion, which raised suspicion of extrinsic coronary compression related to the recent BCT (**Figure 3B**). Intravascular ultrasound (IVUS) confirmed an extensive intramural hematoma with lumen compromise (**Figure 4**, Video 2).

IVUS-guided direct stenting was done using a 2.75 mm × 38 mm drug-eluting stent with care to position the distal stent edge well past the distal end of the IMCH, followed by deployment of a 3.0 mm × 16 mm drug-eluting stent proximally with minimal overlap. TIMI flow grade 3 (Video 3) was restored with resolution of symptoms and improved ST-segment changes (**Figures 3C and 4D**). Ascending aortogram showed no dissection or aneurysm (**Figure 5C**).

ABBREVIATIONS AND ACRONYMS

BCT = blunt chest trauma

CP = chest pain

DAPT = dual anti-platelet therapy

ECG = electrocardiogram

IMCH = intramural coronary hematoma

IVUS = intravascular ultrasound

LAD = left anterior descending artery

MI = myocardial infarction

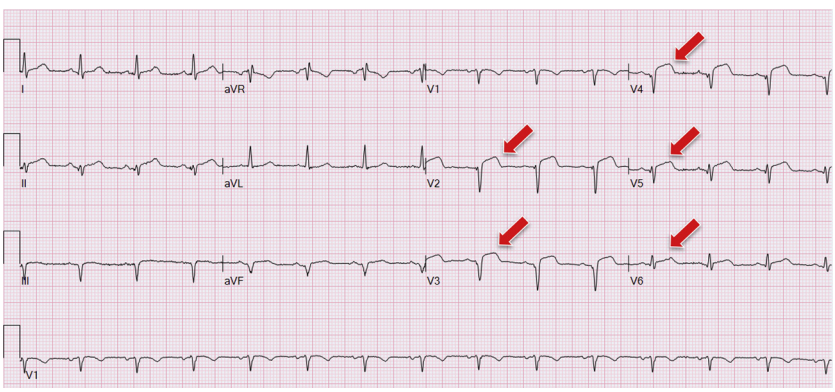
PCI = percutaneous coronary intervention

SCAD = spontaneous coronary artery dissection

STEMI = ST segment elevation myocardial infarction

TIMI = Thrombolysis In Myocardial Infarction

FIGURE 1 Initial Electrocardiogram



Initial electrocardiogram with sinus rhythm and ST-segment elevation **(red arrows)** in leads V_2 to V_6 with associated Q waves suggestive of anterolateral ST-segment elevation myocardial infarction.

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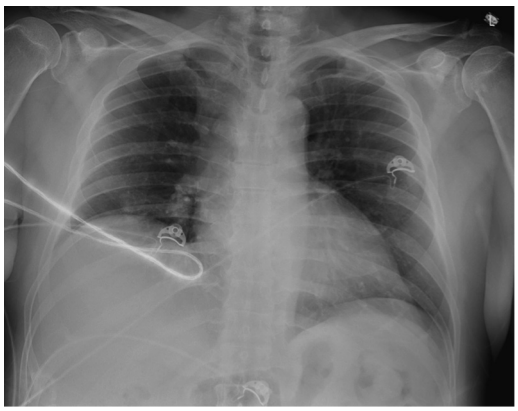
FIGURE 2 Chest X-Ray



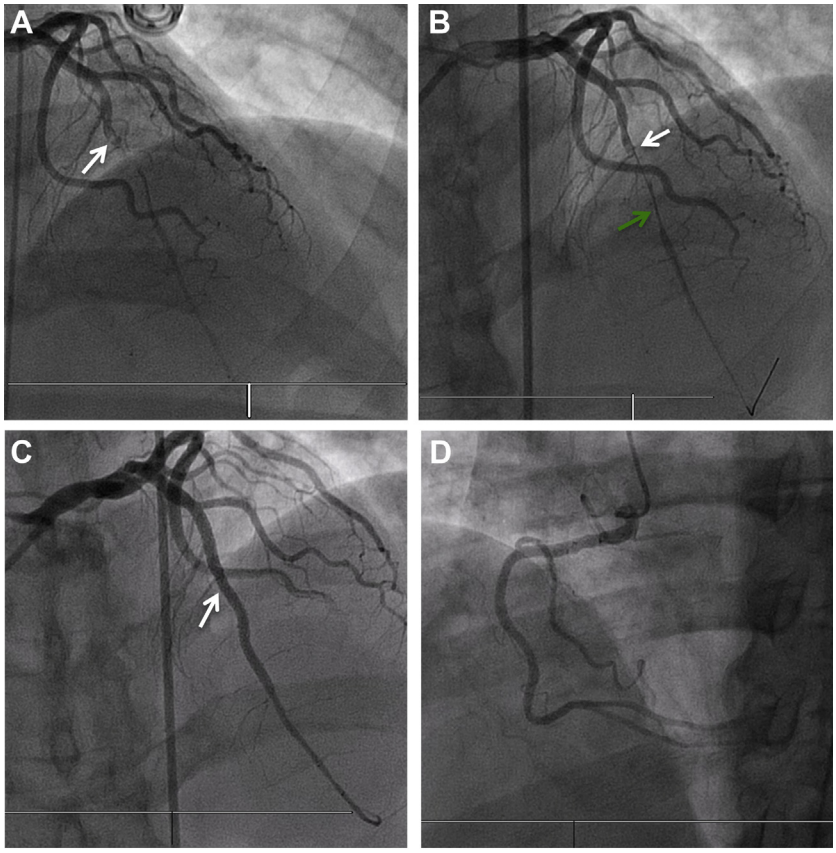
Chest X-ray without acute cardiopulmonary disease and no evident bone fractures.

DISCUSSION

IMCH is a subset of coronary dissection that occurs when blood accumulates in the media due to intimal tear or rupture of the vasa vasorum resulting in luminal narrowing that can lead to MI, arrhythmias, cardiogenic shock, and sudden cardiac death (2–4).

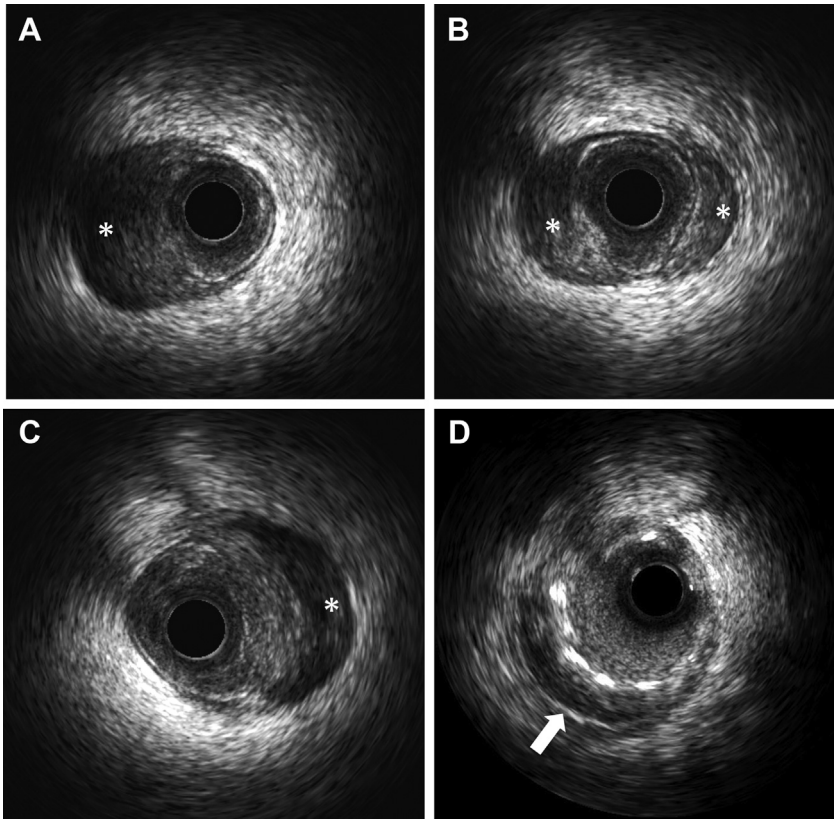
Spontaneous or trauma-related IMCH is a rare, under-recognized, and challenging diagnosis responsible for up to 1% to 4% of all MIs (2). It is more commonly seen as an iatrogenic complication in 6.7% of percutaneous coronary intervention (PCI) cases (3). Although scarcely reported, it has been described after motor vehicle accidents and with contact sports (1). Christenssen et al. (5) studied 77 patients with MI following BCT, and, in 70% of them, the infarction was caused by a coronary occlusion or dissection, with the LAD being the most commonly affected

FIGURE 3 Coronary Angiography



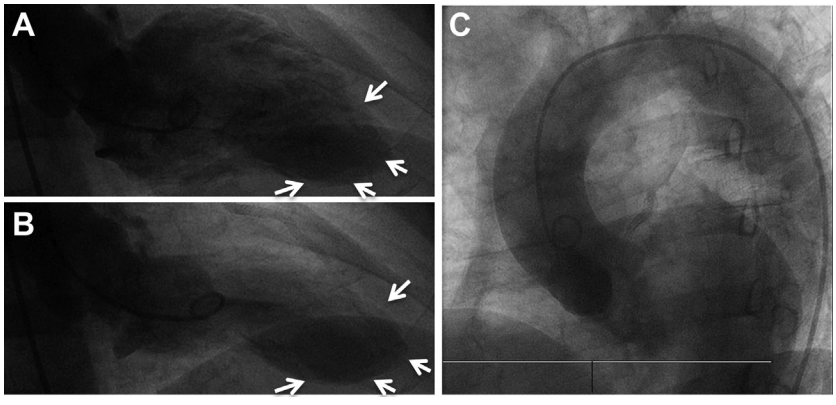
(A) Subtotal occlusion of mid left anterior descending artery with Thrombolysis In Myocardial Infarction flow grade 1 and filling defect suggestive of thrombus **(white arrow)** (Video 1). **(B)** After performing aspiration thrombectomy to the mid left anterior descending artery **(white arrow)** and providing 200 μg of intracoronary nitroglycerin, a long and smooth tubular lesion persisted **(green arrow)**, which raised suspicion for external compression given the history of recent blunt chest trauma. **(C)** Final result (Video 3) after intravascular ultrasound-guided stenting of the mid left anterior descending artery **(white arrow)**. **(D)** Normal right coronary artery.

FIGURE 4 Intravascular Ultrasound



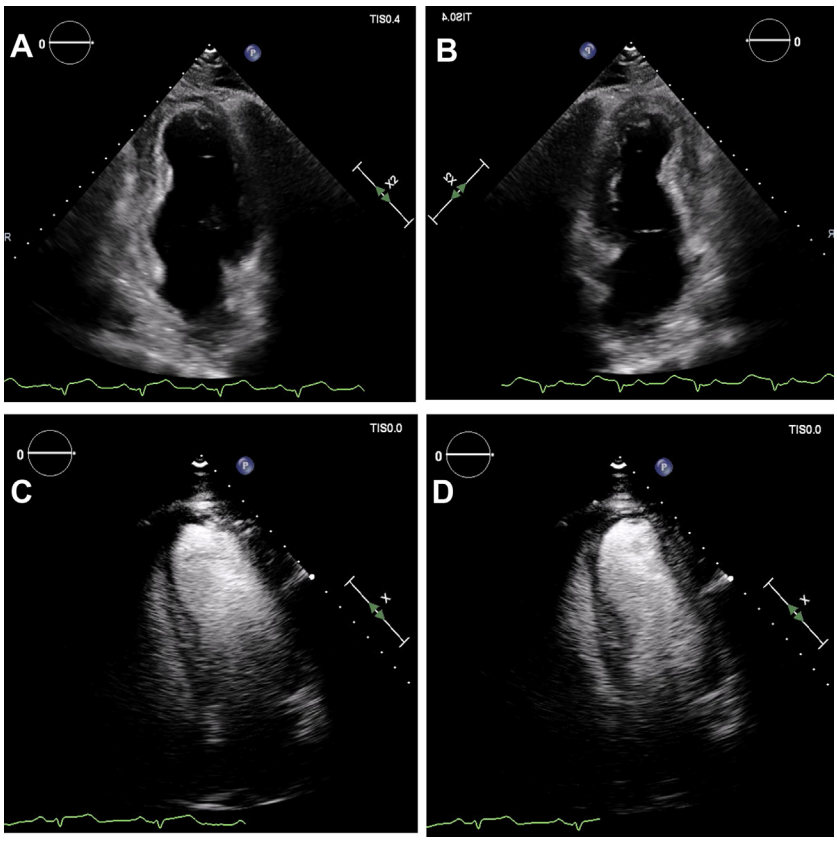
(A to C) Intravascular ultrasound frames showing extensive spiral intramural hematoma **(*)** of the mid to distal left anterior descending artery with lumen compromise (Video 2). **(D)** After Intravascular ultrasound-guided stenting of mid left anterior descending artery with compressed residual intramural hematoma **(arrow)**.

FIGURE 5 Ventricular and Aortic Angiography



Ventricular diastolic **(A)** and systolic **(B)** frames with evidence of distal inferoapical and anteroapical akinesis **(white arrows)** corresponding with the wrap-around left anterior descending artery territory and mildly reduced overall systolic function (ejection fraction 40% to 45%). **(C)** Ascending aorta angiography without evidence of dissection or aneurysm

FIGURE 6 Transthoracic Echocardiogram Post-Myocardial Infarction Day 1



Two-chamber diastolic **(A)** and systolic **(B)** frames showing severe hypokinesis of antero-apical, infero-apical, and mid anteroseptal walls. Four-chamber diastolic **(C)** and systolic **(D)** frames with intravenous contrast showing severe hypokinesis of the septal and lateral apical walls. No evidence of left ventricular thrombi, pericardial effusion, or major valvulopathies.

vessel (76%) followed by the right coronary, left main, and circumflex arteries. Therefore, ECG upon arrival following BCT is a level I recommendation for risk stratification.

When an acute coronary syndrome develops shortly after BCT, the possibility of traumatic coronary dissection/IMCH should be entertained and early coronary angiography should be considered. Although an angiographic appearance of a long lesion of smooth contour may raise suspicion for extrinsic compression from an IMCH as in our case, intracoronary imaging is required to confirm the diagnosis and make the distinction from a spiral coronary dissection or atherothrombosis.

Having made an anatomic diagnosis of spontaneous or traumatic coronary artery dissection or IMCH, the operator must decide whether immediate revascularization is warranted. As opposed to atherothrombosis-related MI for which early revascularization is recommended, retrospective studies with spontaneous coronary artery dissection (SCAD) seem to favor revascularization only if high-risk features such as ongoing/recurrent ischemia, hemodynamic instability, TIMI flow grade 0 to 1, or sustained ventricular arrhythmias are present (2,3). PCI remains the preferred revascularization strategy over surgery except in cases of unprotected left main or proximal 2-vessel SCAD (1,6). Nonetheless, PCI may be subject to worse outcomes in patients with IMCH/dissection if there is axial extension of the hematoma or iatrogenic dissection or inadvertent stenting of the false lumen (3,6). Accordingly, intracoronary imaging is mandatory to ensure intraluminal position and adequate coverage of the IMCH extension, with meticulous attention to stent positioning and sizing (2). Long-term DAPT should be provided after PCI as per American College of Cardiology/American Heart Association guidelines (6).

To the contrary, in stable patients with SCAD without high-risk features, a conservative approach is recommended with relook angiography at 1 month to assess for healing. A meta-analysis of 631 nonrandomized patients with SCAD showed that there is no significant statistical difference between revascularization versus medical therapy in terms of mortality, MI, and recurrent SCAD (7). Rogowski et al. (8) demonstrated angiographic resolution of SCAD in 29 of 30 (97%) patients after 1 month of conservative treatment with a 100% survival rate after a median follow-up of 4.5 years. Retrospective studies seem to favor an initial conservative approach as opposed to early revascularization in stable patients with SCAD without high-risk features, which may be extrapolated to those with traumatic IMCH-related MI because no large series informing of comparative treatment outcomes have been published (2–4)

In regard to medical therapy, long-term DAPT is of unclear benefit in those who did not undergo PCI; yet some recommend a short course of DAPT (1 to 3 months) while others only recommend aspirin for at least 1 year (6). Suitability for extended DAPT should be based on individual risk factors for bleeding. Furthermore, beta-blockers should be provided along with neurohormonal blockade if hypertension or systolic dysfunction is present (6).

FOLLOW-UP

Our patient had no bleeding events nor electrical or mechanical complications from MI. Echocardiogram in post-MI day 1 (**Figure 6**) revealed 40% to 45% ejection fraction. He was discharged home on the day 5 post-MI with guideline-directed medical therapy. Three months post-revascularization, his ejection fraction was 45% to 50% and he remained free of angina.

CONCLUSIONS

IMCH is a rare and potentially under-recognized cause of MI. When confronted with a patient with an acute STEMI, it may be reasonable to inquire about a history of BCT. In that clinical context, a low threshold to use intracoronary imaging to evaluate a culprit lesion may be advisable. In the presence of ongoing ischemia and need for immediate revascularization, intracoronary imaging can help guide percutaneous intervention.

AUTHOR DISCLOSURES

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KEY WORDS acute coronary syndrome, chest pain, dissection, intravascular ultrasound, myocardial infarction, percutaneous coronary intervention

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