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CASE REPORT
Heart failure

Traumatic ischaemic cardiomyopathy in a 27-year-old: a case report

Aishwarya E. Pastapur [1], Abbas Bitar[2], and Brett L. Wanamaker [2]*

[1]Department of Internal Medicine, University of Michigan, Ann Arbor, USA; and [2]Department of Internal Medicine, Division of Cardiovascular Medicine, University of Michigan, 1500 E Medical Center Dr, Ann Arbor, MI 48109, USA

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Background Coronary injury after blunt chest trauma is rare. This case illustrates the importance of evaluating for coronary injury after any episode of blunt chest wall trauma.

Case summary We review the case of a 27-year-old male who presented with acutely decompensated heart failure several months after a motor vehicle accident with chest wall impact from the steering wheel. Coronary angiography demonstrated an occluded left anterior descending artery, and he was found to have a severe ischaemic cardiomyopathy. After multiple hospital and intensive care unit admissions due to multi-organ dysfunction and debility, he was unable to tolerate any guideline-directed medical therapy. He was unable to be listed for heart transplantation due to his co-morbidities, multi-system sequelae of his heart failure, deconditioning, and recent substance use. He was ultimately discharged home with hospice.

Conclusion Coronary or other cardiac injuries should be considered in the evaluation of all patients after blunt chest wall trauma, regardless of prior risk factors for ischaemia.

Keywords Traumatic cardiac injury • Coronary artery injury • Case report • Ischaemic cardiomyopathy

ESC curriculum 3.4 Coronary angiography • 6.2 Heart failure with reduced ejection fraction • 6.5 Cardiomyopathy • 9.2 Trauma to the aorta or the heart

Learning points

Case: A patient presented with new cardiomyopathy secondary to traumatic coronary injury.

- To consider a broad differential in the evaluation of new-onset cardiomyopathy and heart failure, including causes such as trauma resulting in coronary vessel damage and ischaemia.
- To encourage physicians to expand cardiac evaluation after any chest wall trauma, regardless of patient history or risk factors.

Introduction

Cardiac injury following non-penetrating chest wall trauma is an important consideration in the acute evaluation of trauma patients. Cardiac arrhythmias and valvular injuries have been described following blunt chest wall trauma.[1–3] Coronary artery dissection and aneurysm formation have also been reported.[4–7] Cases of coronary artery occlusion or thrombosis are rare.[8,9] Here, we present the case of a young male patient who developed low-output heart failure with reduced ejection fraction after blunt chest wall trauma led to injury to his left anterior descending (LAD) coronary artery.

* Corresponding author. Tel: +734 232 4318, Fax: +734 647 9443, Email: bwanamak@med.umich.edu
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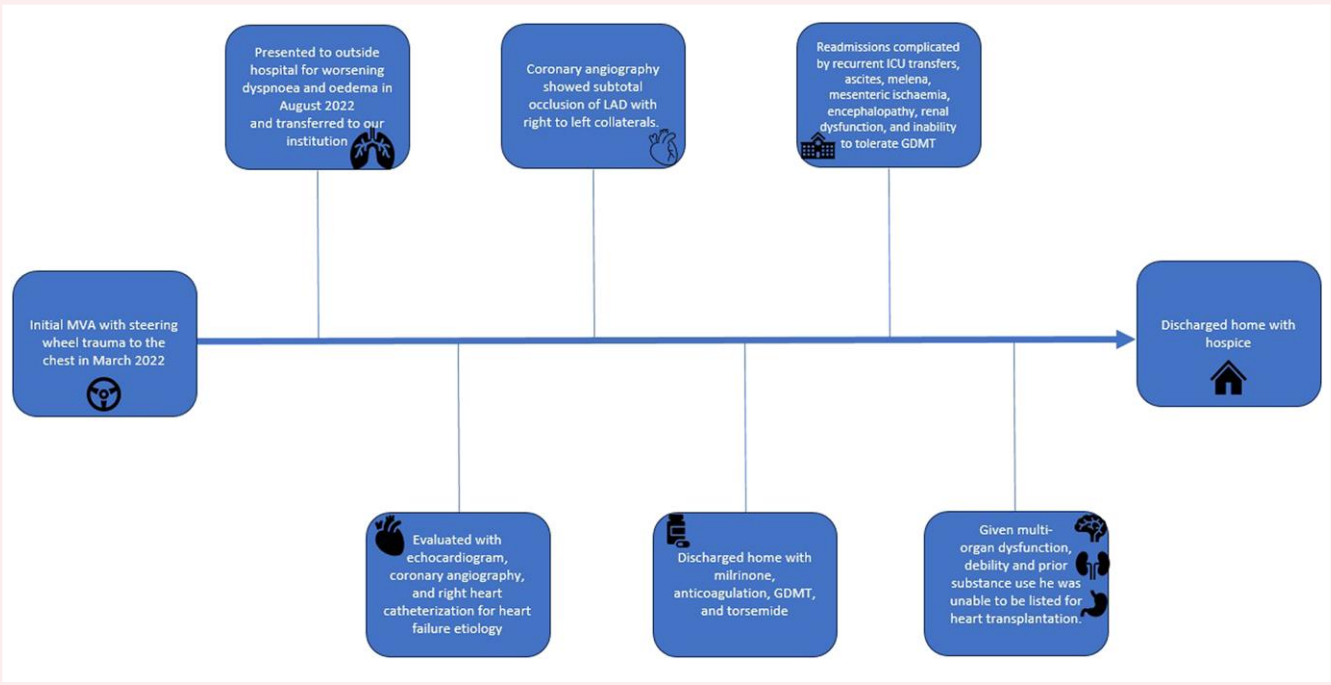
Summary figure



Case presentation

A 27-year-old man with a past medical history notable for a motor vehicle accident (MVA) 5 months prior presented with worsening exertional dyspnoea and lower extremity oedema that was slowly progressive since his MVA. He denied any preceding viral illness, sick contacts, or travel. Initial laboratory evaluation was notable for evidence of hepatic injury (aspartate aminotransferase 632 IU/L and alanine transferase 248 IU/L), normal troponin I levels (0.04 ng/mL), and an elevated B-type natriuretic peptide level at 1787 pg/mL. A transthoracic echocardiogram (TTE) showed normal left ventricle (LV) chamber size (LV end-diastolic diameter 5.6 cm) and severely decreased LV systolic function with an estimated ejection fraction of <20% and evidence of an apical thrombus. He was given intravenous diuretics with improvement in symptoms. Given concern for low-output heart failure, he was transferred to our institution for further workup and management.

On admission, the patient confirmed the above history, noting that, at the time of his MVA, he suffered significant chest wall trauma having struck the steering wheel during the accident, with ongoing chest discomfort he attributed to musculoskeletal pain. He did not seek further evaluation after the accident and his pain eventually resolved. In the following months, he noted worsening exertional dyspnoea, orthopnoea, intermittent chest tightness, and intermittent episodes of paroxysmal nocturnal dyspnoea.

His admission vital signs showed tachycardia to 109 b.p.m. and a blood pressure of 98/60 mm Hg. Physical exam was notable for jugular venous distension, abdominal distension with ascites, and pitting oedema of the bilateral lower extremities. A 12-lead electrocardiogram (ECG) demonstrated sinus tachycardia with low voltage, premature ventricular contractions, and a prior anterolateral infarct pattern (*Figure 1*).

His past medical history was notable for prior MVA and prior alcohol, tobacco, and marijuana use, all discontinued following his MVA.

The differential diagnosis included autoimmune or viral myocarditis, inherited or idiopathic cardiomyopathy, and stress cardiomyopathy amongst other diagnoses. Ischaemic cardiomyopathy was initially felt to be less likely based on the patient's age, though the presence of an anterolateral infarct pattern on ECG and an LV apical thrombus increased suspicion for a prior ischaemic insult.

A TTE was obtained and re-demonstrated severe biventricular systolic dysfunction in addition to an apical aneurysm vs. pseudo-aneurysm with evidence of thrombus (see Supplementary material online, *Video S1*). A subsequent cardiac magnetic resonance imaging scan confirmed severe biventricular systolic dysfunction with full-thickness late-gadolinium enhancement involving all apical LV segments, with apical aneurysm formation in addition to an associated layering thrombus (*Figures 2* and *3*).

Left coronary angiography revealed a subtotal occlusion of the mid-LAD with thrombosis in myocardial infarction 2 antegrade flow (see Supplementary material online, *Video S2*), which was also filled via right-to-left collaterals from the right coronary artery.

Right heart catheterization showed a mean right atrial pressure of 16 mm Hg, pulmonary arterial pressure of 54/31 mm Hg with a mean of 42 mm Hg, and pulmonary capillary wedge pressure of 29 mm Hg. Cardiac output was severely reduced with a cardiac index of 1.3 L/min/m^2 by Fick and 1.27 L/min/m^2 by thermodilution.

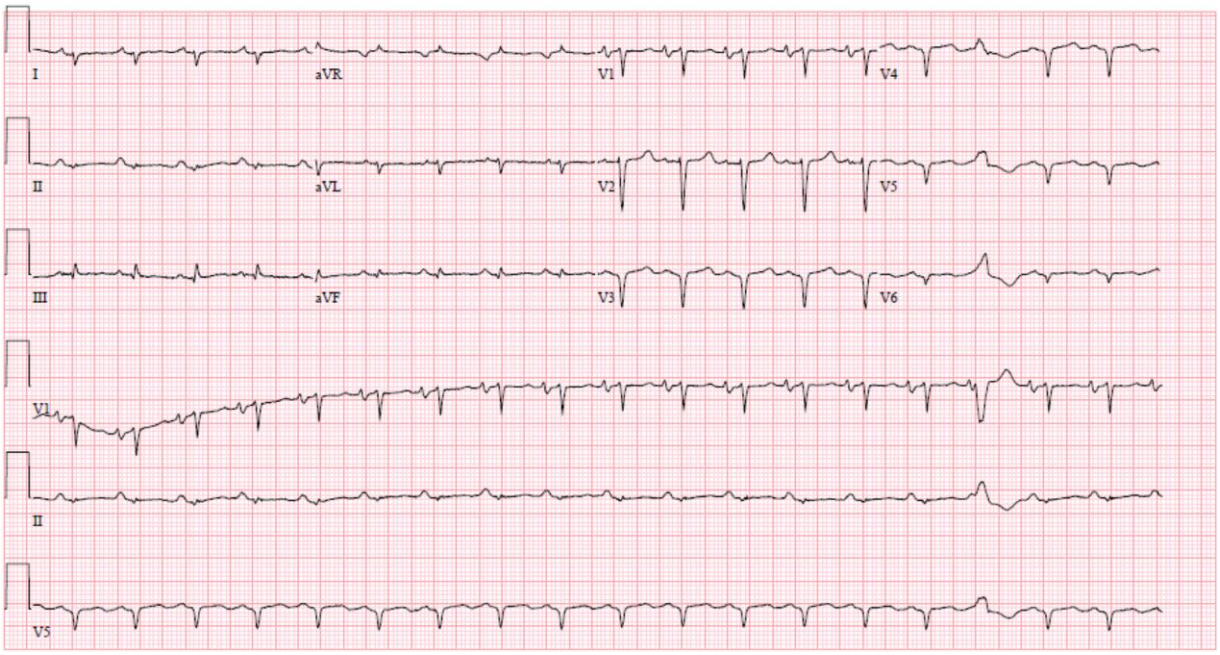


Figure 1 Twelve-lead electrocardiogram demonstrating sinus tachycardia with premature ventricular contractions, low voltage, and prior antero-lateral infarct pattern.

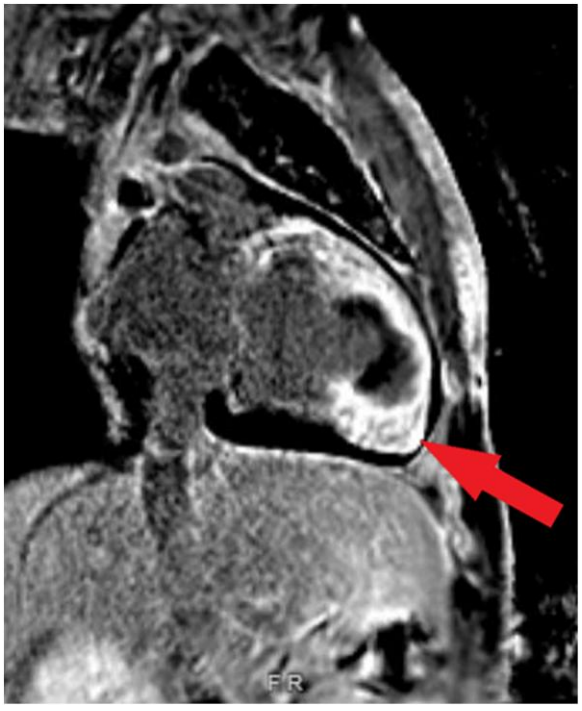


Figure 2 Cardiac magnetic resonance imaging two-chamber view demonstrating late-gadolinium enhancement involving all apical segments with aneurysm formation and associated layering thrombus.

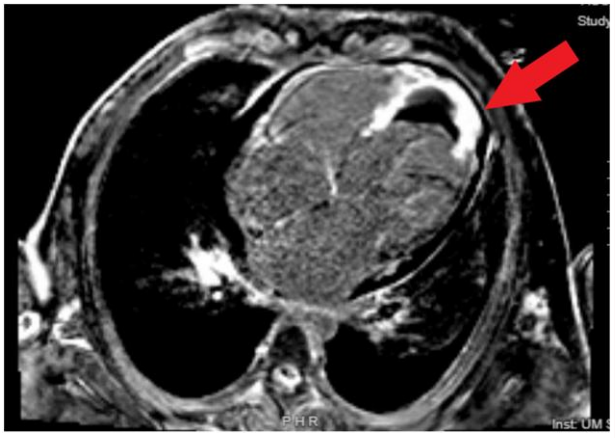


Figure 3 Cardiac magnetic resonance imaging four-chamber view demonstrating late-gadolinium enhancement involving all apical segments with aneurysm formation and associated layering thrombus.

The patient was diagnosed with acute low-output heart failure with reduced ejection fraction in the setting of a severe cardiomyopathy, thought to be due to an unrecognized traumatic coronary injury (sustained during prior MVA) and subsequent adverse LV remodelling.

The patient's volume status was managed with intravenous diuretics. He was unable to consistently tolerate low-dose renin–angiotensin–aldosterone system blockade or beta-blockade given his baseline

hypotension and low cardiac output. He was able to tolerate dapagliflozin and low-dose spironolactone, which he continued on discharge. Milrinone was eventually initiated to augment cardiac output, and a central line was placed for the continuation of therapy at home while the patient underwent further workup and consideration of advanced heart failure therapies. His LV apical thrombus was managed with continuous anticoagulation, and he was discharged on warfarin.

The patient's subsequent course was complicated by recurrent heart failure exacerbations with readmission to the cardiac intensive care unit, subsequent Coronavirus Disease 2019 (COVID-19) infection, and delirium. He was repeatedly evaluated by the multidisciplinary heart team for consideration of advanced heart failure therapies. Severe RV dysfunction precluded LV assist device placement. The patient had developed significant and progressive debility and severe malnutrition despite inotropes in addition to aggressive nutritional and other supportive care. There were also concerns regarding his social support and recent substance use. As such, he was not deemed to be a heart transplant candidate. He was discharged home with oral diuretics, dapagliflozin, and low-dose spironolactone as tolerated with close follow-up for labs, possible medication titration, and further management of advanced heart failure. He was evaluated for a second opinion at another major transplant referral centre and was not deemed to be a candidate for advanced therapies. He was unfortunately readmitted with acutely decompensated heart failure, this time complicated by recurrent ascites requiring paracenteses, mesenteric ischaemia, encephalopathy, continued dysphagia/malnutrition, and renal dysfunction. At the time of this manuscript, he had been discharged with home hospice.

Discussion

While coronary injury following blunt chest trauma is thought to be uncommon, it is a diagnosis of consequence that can affect relatively young patients without traditional cardiac risk factors.[10] Motor vehicle accident with steering wheel trauma is the most frequently reported mechanism, though sport-related injuries have also been described. The LAD is the most commonly reported culprit vessel, and injury may result in traumatic dissection, haematoma formation, or rupture of existing atherosclerotic plaques. Bedside ultrasound examination, cardiac biomarkers, and an ECG should be the standard of care for patients presenting with acute chest trauma regardless of symptoms on presentation. Any abnormalities should be pursued with further imaging such as TTE or computed tomography.[11]

Given the absence of other atherosclerotic diseases, our patient likely sustained a severe LAD injury at the time of his MVA 5 months prior with subsequent occlusion, myocardial infarction, and adverse LV remodelling resulting in low-output heart failure with reduced ejection fraction. This case highlights the need for cardiac evaluation after blunt chest wall trauma, particularly in the setting of ongoing chest discomfort. Clinicians should consider a coronary/ischaemic workup for patients with a history of trauma and cardiac dysfunction, regardless of age or the absence of traditional risk factors.

Lead author biography

Dr Aishwarya E. Pastapur is a resident in Internal Medicine at the University of Michigan with an interest in invasive cardiology. She has been involved with various projects, including those related to the utilization of wearable device data and the application of machine learning models in coronary angiography and the prediction of heart failure. In addition to clinical care and research, she has a passion for mentorship and education, which she hopes to continue in the academic environment.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Consent: The patient and family were made aware of this case report and consented to its publication in compliance with COPE guidelines.

Conflict of interest: None declared.

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author. All relevant data are presented within the manuscript.

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