An Unexpected Cause of Trauma-related Myocardial Infarction: Multimodality Assessment of Right Coronary Artery Dissection

Dear Editor,

Blunt cardiac injury refers to injury sustained due to blunt trauma to the heart. It encompasses a spectrum of pathologies ranging from myocardial contusion; myocardial, pericardial and valvular rupture/aneurysm to coronary artery injuries (dissection, thrombosis or rupture). The clinical manifestations range from clinically silent, transient arrhythmias, to acute myocardial infarction (AMI) and sudden cardiac death. The true incidence of blunt chest injury is unknown as reported rates vary greatly in the literature, ranging between 8% and 71%.[1]

Case Report

A 28-year-old male cyclist sustained blunt chest injury during a road traffic accident. Apart from transient loss of consciousness during the accident, he remained haemodynamically stable thereafter.

The electrocardiograph (ECG) (Fig. 1) showed Q waves and 2 mm ST elevation in leads II, III and aVF (inferior leads) with reciprocal ST depression in leads I and aVL (lateral leads), suspicious for an inferior myocardial infarction. Another ECG performed 30 minutes later showed persistent Q waves in the inferior leads but interval resolution of ST elevation in the inferior and lateral leads. A contrast-enhanced non-cardiac-gated thoracic computed tomography (CT) scan showed fractures of the upper ribs and right transverse process of the 7th cervical vertebra, small mediastinal haematoma, lung contusions and bilateral pneumothoraces. The troponin I increased from 10 ng/dL to >72,000 ng/dL (normal range 0-39 ng/dL). Subsequent serial ECGs revealed T wave inversion in leads III, aVF and V1, suggestive of an evolving subendocardial myocardial infarction.

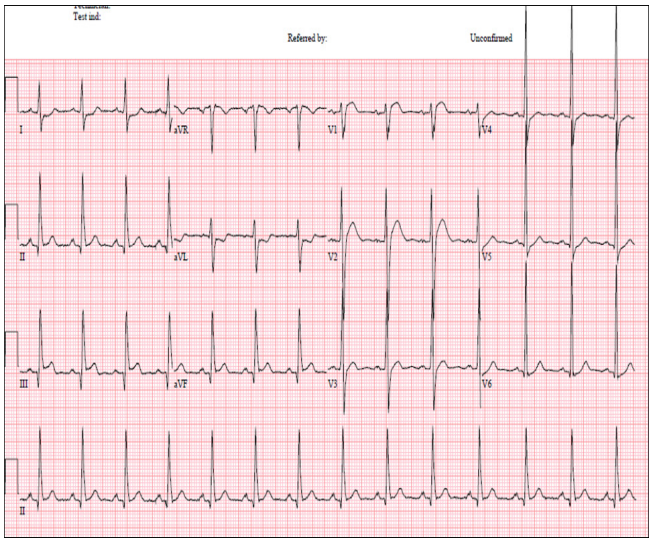

Fig. 1. A 12-lead ECG showed 2 mm ST elevation in leads II, III and aVF with reciprocal ST depression in leads I and aVL, suspicious for an inferior myocardial infarction.

Transthoracic echocardiogram showed inferoseptal and inferior regional wall motion abnormality. No pericardial effusion was detected (Fig. 2). Retrospectively, subendocardial hypoenhancement of the inferoseptal and inferior wall of the left ventricle was seen in the initial thoracic CT scan (Fig. 3). This was in keeping with myocardial infarction in the right coronary artery (RCA)

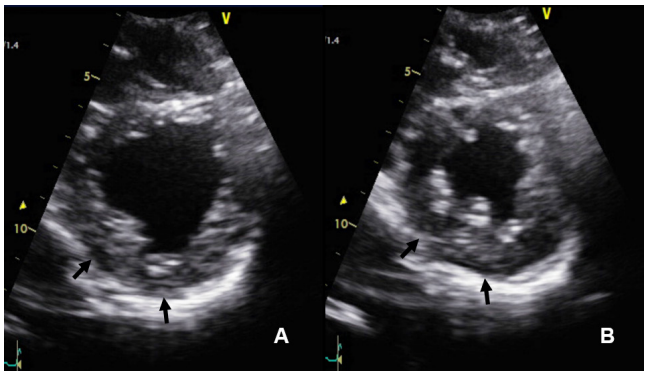

Fig. 2. Short axis images from echocardiogram in ventricular diastole (image A) and systole (image B) showed regional wall motion abnormality in the inferoseptal and inferior wall (arrows). No pericardial effusion was detected.

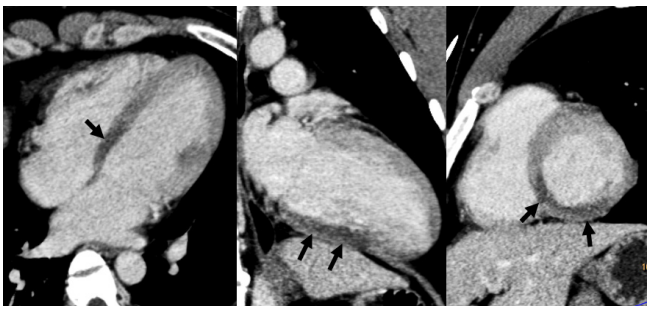

Fig. 3. Multiplanar reformats of the non-cardiac gated CT thorax images in the 4-chamber, 2-chamber and short axis views of the heart showed extensive subendocardial hypoenhancement involving the inferoseptal and inferior wall of the left ventricle (arrows), in keeping with myocardial infarction in the RCA territory.

territory. Since the patient remained well without any signs and symptoms to suggest an AMI, an initial diagnosis of cardiac contusion was made.

The patient was managed conservatively but 5 days following admission, the cardiovascular magnetic resonance imaging (CMR) (Fig. 4) showed features of acute myocardial infarction in the RCA territory. Late gadolinium enhancement (LGE) images showed LGE at the basal and mid-cavity inferoseptal and inferior left ventricular wall (RCA territory). The early gadolinium enhancement images showed subendocardial hypointense foci in the above myocardial segments, indicative of microvascular obstruction (no reflow phenomenon). There was moderate to severe hypokinesia in the same segments on the cine gradient echo images.

CT coronary angiogram (CTCA) (Fig. 5) showed proximal RCA dissection with the true lumen being compressed by the false lumen. As there was good distal runoff in the RCA and the patient remained asymptomatic, no invasive imaging or intervention was performed. The patient was treated medically with a plan to return for follow-up assessment and imaging.

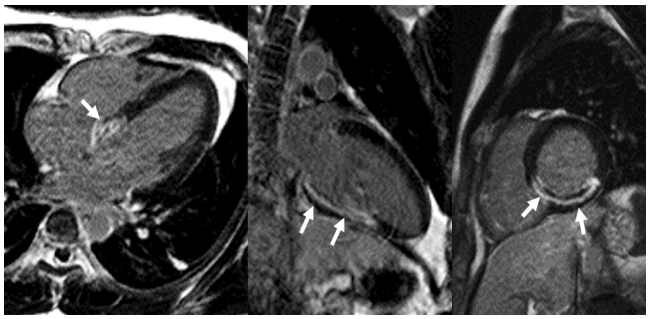

Fig. 4. Corresponding 4-chamber, 2-chamber and short axis late gadolinium CMR images showed LGE in the basal to mid cavity inferoseptal and inferior wall of the left ventricle (arrows), corresponding to the findings on the initial non-cardiac-gated thoracic CT scan. Subendocardial foci of hypointensity within the RCA territory myocardial infarction is in keeping with no reflow phenomenon, also known as microvascular obstruction.

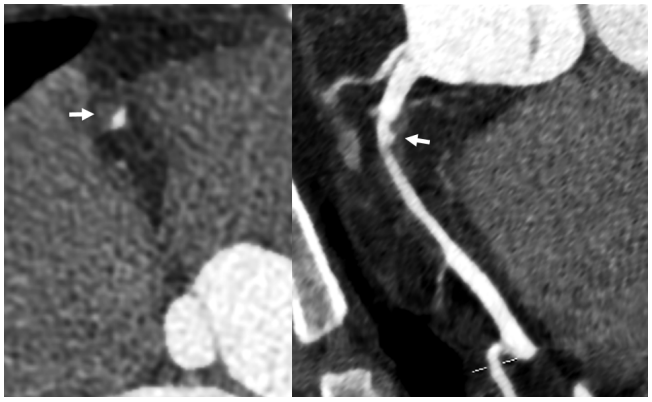

Fig. 5. Multiplanar reformatted images of the RCA from a 3rd generation dual-source CT coronary angiogram study 5 days after the trauma showed irregular severe luminal narrowing of the true lumen of the proximal RCA secondary to mass effect from the false lumen (arrows), in keeping with a dissection. A small outpouching of contrast proximally was suspicious for an intimal tear. There was good distal run-off with contrast present in the mid and distal RCA.

Discussion

The sequelae from blunt chest injury can vary from a simple arrhythmia to myocardial rupture. Coronary artery dissections are exceedingly rare in the clinical context of blunt chest injury. Autopsy studies of blunt chest trauma have revealed that injuries to the heart and coronary arteries are present in 20% and less than 2% of the study cohort respectively.[2] The mortality rate is high—ranging from 13.8% to 43.1%[3]—and is dependent on the severity of blunt chest injury assessed using the abbreviated injury score.

Traumatic coronary artery dissections are exceedingly rare. The most frequently injured vessel is the left anterior descending artery (71.4%) followed by the RCA (19%), left main coronary artery (6.4%) and left circumflex artery (3.2%).[4] The pathogenesis of acute AMI following trauma-induced coronary artery dissection is unclear, but shearing forces during the traumatic episode may produce a small intimal tear which subsequently initiates the process of thrombus formation.

Clinical manifestations of coronary dissection and cardiac contusion are variable and often overlap. It is often difficult to establish an accurate diagnosis because chest pain can be overshadowed by concomitant injuries. We recommend that patients with i) ECG changes suggestive of ongoing myocardial ischaemia, such as evolving and focal myocardial infarction, ii) unresolving or worsening chest pain, iii) persistent elevation or rising troponin I and; iv) worsening cardiogenic shock should undergo further non-invasive cardiac imaging such as CMR or CTCA.

There are no specific recommendations regarding the choice of advanced cardiac imaging modalities for assessing blunt chest injury currently.[5] With the widespread availability of multi-detector CT scanners, CTCA can now be easily performed for diagnostic imaging in patients with significant trauma and cardiac injury. It is fast, non-invasive and provides good quantitative and qualitative assessment of the coronary arteries and aortic root. CTCA can accurately identify the location and extent of coronary injury and differentiate between plaque rupture, thrombus, dissection or external compression.[6] This negates the potential risks of catheter-related injury, including propagation of the coronary artery dissection. Although CTCA exposes the patient to ionising radiation, the radiation dose can be as low as below 1 millisievert (mSv) (equivalent to less than 50 chest x-rays) using the latest CT scanners.[7] CMR is a useful complementary non-ionising imaging modality, which can distinguish myocardial infarction from myocardial contusion.

The management of coronary artery dissection from blunt chest injury remains controversial because of their rare occurrence. Percutaneous coronary intervention, coronary artery bypass grafting, thrombolysis and conservative medical treatment in the setting of AMI associated with blunt chest injury, have all been reported with good clinical outcomes.[8-9] Our patient was managed medically with good clinical recovery.

Conclusion

AMI secondary to coronary artery dissection is a rare complication from blunt chest injury, but it carries high morbidity and mortality. Vigilance and a high index of suspicion are necessary when managing patients with blunt chest injury. Timely intervention may be vital for myocardial recovery and prevention of further progression of coronary artery dissection. CTCA should be utilised as a form of non-invasive imaging during the investigation of blunt chest injury in the appropriate clinical context. We propose that in haemodynamically stable patients with a high clinical suspicion of cardiac injury, CTCA should be performed.

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