Case report

Acute chest pain of cardiovascular aetiology: a diagnostic dilemma

RAVI G. ASSOMULL1*, ANKUR GULATI1*, CHEUK F. CHAN1,2, NIZAR ISMAIL1, TRISTAN D.H. BROWN1, SADAF RAZA1, KAUSHIK GUHA1,2, NICHOLAS BUNCE3 and RAAD H. MOHIADDIN1,2

From 1Cardiovascular Magnetic Resonance Unit, Royal Brompton Hospital, London, 2National Heart and Lung Institute, Imperial College and 3Department of Cardiology, St George’s Hospital, London, UK

Address correspondence to Dr Ankur Gulati, CMR Unit, Royal Brompton Hospital, Sydney Street, London SW3 6NP, UK. email: a.gulati@rbht.nhs.uk
*These authors contributed equally to this work.

Case report

A 61-year-old gentleman was admitted to his local hospital with a 5-h history of central chest pain. He was an ex-smoker with no other cardiac risk factors or history of ischaemic heart disease. ECG on presentation demonstrated ST elevation in leads 1 and aVL consistent with acute myocardial infarction (MI) and he was therefore thrombolysed with tenecteplase. Following thrombolysis, his ST segment elevation resolved and his chest pain subsided completely. Peak CK was 1015 U/l (24–173 U/l) and troponin T 3.65 ug/l (0–0.10 ug/l). A routine PA chest radiograph revealed a widened superior mediastinum, raising the possibility of aortic dissection (Figure 1). An urgent contrast enhanced CT scan was therefore requested which demonstrated a type A aortic dissection with an aneurysmal ascending aorta (Figure 2). Despite the CT findings, the patient remained well following thrombolysis with no subsequent clinical sequelae of acute aortic dissection. Further investigation with transthoracic echocardiography revealed good left ventricular function with no pericardial effusion. There was mild central aortic regurgitation but the aortic annulus was not significantly dilated. Coronary angiography performed via right radial access demonstrated a proximal 60–70% left anterior descending (LAD) lesion and an occluded diagonal branch.

The patient was considered for aortic root replacement. However, it was felt that surgery should be deferred in the event that the patient’s

Figure 1. PA chest radiograph shows widening of the mediastinum and ectasia of the descending thoracic aorta. The lung fields are clear.
presentation was actually secondary to an acute MI which was unrelated to the dissection. On further direct questioning the patient admitted to a previous episode of severe chest pain 2 years earlier, although he did not seek medical attention at that time. In order to resolve this diagnostic uncertainty, a gadolinium enhanced cardiovascular magnetic resonance scan (CMR) was organized to further assess the dissection and detect any myocardial necrosis. This confirmed a Type A aortic dissection and aneurysmal ascending aorta with thrombus in the false lumen (Figure 3). No pericardial effusion was noted. The anterior wall was akinetic at mid-cavity level (Figure 4) with evidence of microvascular obstruction (MVO) and transmural late gadolinium enhancement (Figure 5). These findings were diagnostic of recent acute MI. Surgery was therefore deferred until 8 weeks later when the patient suc-

Figure 2. CT aorta demonstrates type A aortic dissection. In the dilated ascending aorta there is thrombus within the false lumen (asterisk). The dissection can be seen to extend into the descending thoracic aorta (arrow). T = true lumen.

Figure 3. (a) A balanced steady-state free-precession (bSSFP) MRI image of the aortic root and ascending aorta. There is a spiralling dissection flap which arises 15 mm above the sino-tubular junction and extends into the aortic arch (white arrows). The false lumen contains a large thrombus (asterisk). A jet of mild aortic regurgitation can also be observed (black arrow). (b) MR angiogram (MRA) shows the thrombus as a filling defect within the false lumen of the proximal ascending aorta (asterisk). The dissection flap in the aortic arch and proximal descending aorta is clearly seen (white arrows). (c) MRA reconstruction allows 3D visualization of the course of the dissection from its origin into the descending aorta (white arrows).
cessfully underwent elective aortic root replacement with a single graft to the LAD. He made an uncomplicated recovery.

**Discussion**

Acute chest pain has a variety of underlying causes. The commonest cardiac aetiology is acute coronary syndrome (ACS) for which antiplatelet agents, heparin, thrombolysis or percutaneous revascularization are the established initial treatments in most UK centres. Aortic dissection is another important aetiology, which may in turn be mistaken for and treated as ACS with potentially fatal consequences. Rarely, the two conditions may co-exist. In such patients who present with acute chest pain, the challenge for the clinician is to correctly identify which pathology is primarily responsible for the patient’s symptoms.

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**Figure 4.** bSSFP mid-ventricular short axis images at end-diastole (a) and end-systole (b). There is a localized akinetic segment in the anterior wall (arrowed).

**Figure 5.** T1-weighted early gadolinium contrast short-axis (a) and two-chamber (b) images reveal a well-defined hypointense segment of MVO (arrow head) in the anterior wall. The equivalent late gadolinium images (c and d) confirm MVO with peripheral transmural enhancement (arrows) consistent with myocardial infarction.
Our case highlights the application of CMR in making this important differentiation, thereby facilitating appropriate patient management. CMR offers several advantages which make it ideally suited for this role. First, CMR provides high spatial resolution, multi-planar imaging of cardiovascular anatomy without exposure to ionizing radiation or nephrotoxic contrast. This feature allows comprehensive interrogation of major arteries and minimizes the risk of missing small dissection flaps. MR angiography (MRA) can be used to construct detailed 3D representations of the great vessels which allow the course of dissection to be tracked contiguously and help facilitate surgical intervention. The presence of a pericardial effusion or thrombus in the false lumen are both easily visualized with CMR and can in turn be used to infer whether the dissection is acute or chronic.

In addition, CMR enables accurate, highly reproducible measurements regarding LV dimensions, performance and valvular function. The use of gadolinium contrast makes CMR a truly versatile imaging modality due to its unrivalled ability to facilitate tissue characterization. As a result, CMR can detect both acute and chronic myocardial infarcts with great accuracy and sensitivity. In this case, the use of gadolinium contrast imaging allowed visualization of MVO within the context of an acute infarct. Following gadolinium administration, the area of MVO is seen as a dark hypo-enhanced region (Figure 4) due to absence of contrast penetration into this area relative to normally perfused myocardium (early phase images) or surrounding bright infarcted tissue (late phase images). The presence of MVO is the hallmark of acute myocardial infarction.

In summary, this report highlights the potential value of CMR in discriminating between thoracic aortic dissection and acute myocardial infarction in patients presenting with chest pain. Our case was particularly unusual due to the independent co-existence of both pathologies. Despite this finding, CMR was able to establish the primary cause of our patient’s presentation so that treatment could be guided appropriately. For this reason we believe that where available, CMR may offer supplemental information to CT in the work-up of patients with chest pain of uncertain cardiovascular aetiology.

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References


