Left ventricular remodeling after acute myocardial infarction with microvascular obstruction

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Abstract
Accurate assessment of myocardial function and determinants of adverse functional outcome after acute myocardial infarction are essential in further therapeutic management of affected patients. Although echocardiography is most commonly used for this purpose, cardiovascular magnetic resonance (CMR) is emerging as an important diagnostic tool in the evaluation of ischemic heart disease. The advantage of CMR in comparison with other techniques is the ability to combine anatomical and functional information in a single non invasive examination, with superior spatial and temporal resolution, and without dependence on the patient’s acoustic window or the use of ionizing radiation. This case report demonstrates a comprehensive CMR evaluation of a patient with a large myocardial infarction, and its complications.

Keywords: Cardiovascular magnetic resonance, ischemic heart disease, left ventricular remodeling, microvascular obstruction, myocardial infarction

Case report
After a day at work, a 35-year-old man presented to the emergency department because of severe chest pain, with nausea and dyspnea. The symptoms began an hour after he had started work; however, he did not go to the emergency department until after his shift. He had smoked a pack of cigarettes a day since the age of 15 years and his family history was positive for cardiovascular disease. At the time of presentation, the patient still had chest pain and ST-segment elevation involving leads aVL and V1–V5, with reciprocal ST-segment depression in leads II, III, and aVF, which did not respond to vasodilative medication. He then was brought to the catheterization laboratory, where the left anterior descending artery was found to be proximally occluded. After primary percutaneous coronary intervention with stenting there was suboptimal epicardial flow (Thrombolysis In Myocardial Infarction [TIMI] grade 2), and the electrocardiogram revealed minimal ST-segment resolution after reperfusion. Laboratory testing showed a peak creatine kinase concentration of 11 515 U/L, with an MB fraction of more than 500 U/L. Six days after admission to hospital, the patient was referred to our imaging department for evaluation of ventricular function and the extent of infarction.

Cine imaging was used to assess cardiac function, revealing severely impaired left ventricular function (Figure 1a and c), with akinesia of the entire septum, anterior and anterolateral walls, and the apex, with preserved wall thickness. There was mitral valve regurgitation as a result of a dilated left ventricle, and a pericardial effusion. Assessment of the right ventricle also showed akinesia of the apex. In addition, there was a mass in the left ventricular apex suggesting a thrombus. Ten to 15 minutes after the administration of a gadolinium-based contrast agent, late contrast-enhanced images were acquired (Figure 1b), demonstrating a thrombus in the left
ventricular apex. There was transmural hyperenhancement of the akinetic segments of the left ventricle, with hypoenhanced areas in the infarct core, attributed to delayed wash-in of the contrast agent because of severe injury of the microvasculature. In addition, hyperenhancement of the distal right ventricular apex confirmed myocardial infarction, not stunning, to be the cause of the wall motion abnormalities. Before the patient was discharged from the hospital, a repeat CMR was performed, 16 days after the percutaneous coronary intervention. Cine imaging demonstrated further deterioration of left ventricular function and geometry, with wall thinning of the transmurally infarcted myocardium. Despite a low-flow state in the left ventricle, anticoagulation therapy had dissolved the thrombus.

Discussion

This case report illustrates that CMR offers a comprehensive evaluation of a patient after acute myocardial infarction in one single non invasive study. It provides information on the size and extent of myocardial infarction, its complications (eg, thrombus, mitral regurgitation) and other sequelae of infarction (eg, microvascular obstruction, right ventricular involvement), improving the risk stratification of these patients [1–3]. In this patient, large areas of microvascular obstruction were found; this was the result of impaired reperfusion at the myocardial tissue level, caused by mechanisms including the development of tissue edema, platelet plugging, neutrophil adhesion, myonecrosis, and intracapillary red blood cell stasis, also known as the “no-reflow” phenomenon [4]. Several studies have shown that microvascular obstruction is strongly associated with a greater incidence of left ventricular remodeling, congestive heart failure, and death [5,6]. In a comparative study, the prognostic value of microvascular obstruction detected by CMR was stronger than the commonly used criteria of microvascular injury (eg, TIMI flow, myocardial blush grade, ST-segment resolution) for the prediction of ventricular
function; in addition, it appeared to be more relevant than infarct size or the transmural extent of infarction [7]. Furthermore, typical findings of no-reflow measured by functional intracoronary Doppler flow (e.g., systolic retrograde flow, rapid deceleration of diastolic flow, reduced coronary flow velocity reserve) are associated with the anatomical extent and size of microvascular obstruction revealed by CMR [8]. Therefore, CMR should be considered in patients after acute myocardial infarction with severe impairment of left ventricular function, to predict functional outcome and prognosis, and to assess the presence and extent of suspected infarct-related complications.

REFERENCES


