Case report

Contrast-enhanced magnetic resonance imaging guided decision making after primary percutaneous coronary intervention for acute ST-elevation inferior myocardial infarction

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Abstract

Coronary occlusion of large epicardial branches leads to profound ischemia at the infarct core, resulting in simultaneous necrosis of myocytes and endothelial cells. This process leads to microvascular obstruction in the infarct core, described as the no-reflow region in basic studies and documented in humans by contrast-enhanced magnetic resonance imaging and ultrasound. After coronary occlusion, contrast-enhanced magnetic resonance identifies myocardial infarction as a hyperenhanced region containing a hypoenhanced core. There is growing interest in incorporating its assessment into the evaluation of acute myocardial infarction because it is the key in defining specific therapeutic strategies and in directing the interventional therapy. We report a rare case of right ventricular infarction where contrast-enhanced magnetic resonance produced detailed images of myocardial perfusion pattern and tissue damage and directed the treatment after acute myocardial infarction.

Keywords: Cardiac magnetic resonance; Right ventricular infarction; No-reflow phenomenon

1. Introduction

Microvascular obstruction within an area of acute myocardial infarction (AMI) indicates worse functional recovery and a higher risk of postinfarction complications. The magnitude and type of myocardial damage within the infarcted region have been recognized as important determinants of prognosis after AMI [1]. Electron microscopic studies of tissue within the no-reflow region reveal severe microvascular damage and obstruction by red and white blood cells and other necrotic debris. Microvascular obstruction at the infarct core has been demonstrated in humans [2] and represents a predictor of poor AMI recovery and postinfarction cardiovascular complications [3]. Contrast-enhanced magnetic resonance imaging (MRI) has been shown to index infarct size by probing water kinetics through shortened proton relaxation times induced by gadolinium atoms [4]. There is a growing interest in incorporating no-reflow phenomenon assessment into the evaluation of AMI because it is the key in defining specific therapeutic strategies and in directing the interventional therapy.

We report a rare case of right ventricular infarction with a large area of no-reflow at MRI.

2. Case report

A 54-year-old caucasian male with hypertension, smoking habit and a previous history of anterior myocardial infarction presented with an inferior ST-elevation AMI. Coronary angiography showed coronary artery ectasia with aneurysms, a total occlusion in the mid right coronary artery and a significant lesion of the first diagonal branch. Primary percutaneous transluminal coronary angioplasty was performed at the site of the culprit lesion within the first three hours after chest pain onset. The early result was an incomplete reperfusion (TIMI grade 2 flow). On the 4th hospital day the patient was sent to the Cardiothoracic Centre of Monaco for cardiovascular MRI scan (1.5 T Sonata, Siemens, Germany) in order to look for viability in the infarct zone and ischemia in the non-infarcted territory. Steady-state free procession sequence confirmed normal ventricular volumes and a moderately reduced ejection fraction (40%) with respectively left ventricular anterior and inferior wall motion abnormalities and right ventricular diaphragmatic wall motion abnormalities as assessed with echocardiography. At short T1 inversion recovery sequence the edema of the recent infarct area was visualized. Gadolinium early (2 min) and late (20 min) images recognized a large hyperenhanced area (transmural necrosis) involving the left ventricular inferoseptal wall and the right ventricular diaphragmatic wall surrounding a hypoenhanced region. This
phenomenon describes the compromised myocardial blood flow at the microvascular level (no-reflow phenomenon) with no evidence of viability (Figs. 1 and 2; early and late enhancement, respectively). The adenosine stress MRI revealed reversible ischemia in some of the territories supplied by the left anterior coronary artery and showed perinecrotic inferoseptal ischemia. The patient underwent four-vessel coronary artery bypass grafting.

3. Discussion

Early reperfusion by thrombolytic therapy or angioplasty is now widely used to limit infarct size, preserve ventricular function, and improve survival in patients with AMI [5]. Although restoration of blood flow to previously ischemic tissue does occur after reperfusion, the process is not homogeneous, and limited myocardial perfusion is observed in some parts of the injured territory that creates the no-reflow phenomenon [1]. Multiple factors, including embolization of thrombus and plaque, endothelial dysfunction, inflammation, edema, may all work in concert to generate microvascular obstruction after AMI [6]. Major determinants of the amount of no-reflow are the duration of occlusion, infarct size and also the duration of reperfusion [7]. Despite its prognostic importance, accurate assessment of microvascular reperfusion in AMI and its consequences is difficult. The ideal modality would combine in one single examination information on microvascular flow, along with defining regional wall motion abnormalities and areas of myocardial viability. A promising modality is represented by cardiac MRI [8]. The application of cardiac MRI is highly accurate in assessing ventricular function, and techniques incorporating the administration of gadolinium contrast with rapid acquisition of sequential images demonstrate myocardial perfusion. Furthermore, delayed hyperenhancement after contrast enables accurate delineation between infarcted and viable myocardium, allowing prediction of recovery of myocardial function [9]. After contrast injection, gadolinium extravasates into the interstitium of infarcted myocardium where contrast wash-out is delayed. This results in significantly greater T1 shortening of infarcted myocardium compared to normal tissue. On delayed enhancement MRI (i.e. 20 min after contrast administration), the area of infarction is thus visualized as a region of high signal intensity or hyperenhancement (Fig. 2). On early first pass imaging (i.e. the first few minutes after contrast injection), however, gadolinium is an intravascular agent (Fig. 1). When seen within an acutely reperfused infarct territory perfused by a patent epicardial artery, regions of hypoenhancement (low signal intensity) represent the no-reflow phenomenon or regions of microvascular obstruction.

4. Conclusion

Advances in imaging modalities have improved visualization of no-reflow, showing its frequency to be higher than was estimated by clinical judgment alone. A rare case of right ventricular infarction that guided the therapeutic approach by the MRI was described.
References


