# Role of cardiac MRI in acute myocardial infarction

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**Abstrak**

*Angka kematian pada pasien dengan infark miokardium akut (IMA) telah menurun secara signifikan oleh karena strategi terapi reperfusi saat ini. Terapi reperfusi dapat berakibat pada cedera reperfusi. Oleh karena itu, tatalaksana pada pasien ini menghadapi tantangan baru dalam hal diagnosis dan tatalaksana gagal jantung, identifikasi kondisi iskemia, estimasi keperluan antikoagulan, dan penilaian risiko kardiovaskular secara menyeluruh. Ilustrasi kasus ini akan menunjukkan pengaruh* magnetic resonance imaging *(MRI) kardiak dalam penilaian patofisiologi IMA pada era terapi reperfusi. MRI kardiak akan memberikan informasi berguna yang akan membantu para klinisi dalam tatalaksana dan pemilihan strategi terapi spesifik pada pasien IMA.* ***(Med J Indones. 2013;22:46-53)***

**Abstract**

Mortality in patients with acute myocardial infarction (AMI) has decreased significantly and appears to be the result of current reperfusion therapeutic strategies. Reperfusion itself may develop into reperfusion injury. Therefore, management of these patients poses several challenges, such as diagnosing and managing heart failure, identifying persistent or inducible ischaemia, estimating the need for anticoagulation, and assessing overall cardiovascular risk. This case presentation will demonstrate the impact of cardiac magnetic resonance imaging (MRI) in the assessment of the pathophysiology of AMI in the current reperfusion era. Cardiac MRI can provide a wide range of clinically useful information which will help clinicians to manage and choose specific therapeutic strategies for AMI patients. ***(Med J Indones. 2013;22:46-53)***

**Keywords:** *Acute myocardial infarction, cardiac magnetic resonance imaging, reperfusion injury*

Nowadays, at least 70% of patients hospitalized with acute myocardial infarction (AMI) survive the acute hospital phase.[1] Mortality has decreased significantly and appears to be the result of the decline in the incidence of ST-segment elevation myocardial infarction (STEMI) along with the absolute reduction of overall mortality due to the efficacy of current therapeutic strategies either by thrombolitic therapy or primary percutaneous coronary intervention (PCI).[2,3] Reperfusion itself, after prolonged coronary occlusion may develop into reperfusion injury associated with impairment of microcirculatory flow and possible further deterioration due to intramyocardial haemorrhage.[4] Therefore, management of these patients poses several challenges, such as diagnosing and managing heart failure, identifying persistent or inducible ischaemia, estimating the need for anticoagulation, and assessing overall cardiovascular risk. Cardiac imaging plays a prominent role in all of these tasks, especially cardiac magnetic resonance imaging (MRI).[5]

The pathological consequences of reperfusion strategies can be currently detected by cardiac MRI. Cardiac MRI can provide a wide range of information such as myocardial oedema (myocardium at risk), location of transmural necrosis, quantification of infarct size (IS) and microvascular obstruction leading also to intramyocardial haemorrage.[4] Moreover, cardiac MRI provides an accurate and reproducible modality for the assessment of global ventricular volumes and function.[6]

This case presentation will demonstrate the impact of cardiac MRI in the assessment of the pathophysiology of AMI in the current reperfusion era, focusing on clinical applications and future perspectives for therapeutic strategies.

## CASE ILLUSTRATION

Patient 29 years old male came to National Cardiovascular Center Harapan Kita on May, 10 2012 with extensive anterior STEMI 8 hours onset Killip I. Patient's BMI 17.2 kg/m$^2$, blood pressure 116/74 mmHg, heart rate 69 bpm with normal physical examination. He was an active smoker.

Electrocardiogram (ECG) revealed sinus rhythm with ST elevation and Q waves at V1-V4, aVL leads (Figure 1). Chest X-Ray showed no sign of cardiomegaly and congestion. Patient was given double anti-platelet, nitrates, and eptifibatide. He underwent primary PCI, since the coronary angiography showed thrombus at left anterior descending (LAD) with thrombolysis in myocardial infarction (TIMI) 3 flow (Figure 2), total occlusion at distal diagonal 2 and others within normal limit so primary PCI wasn't performed.

Laboratory results revealed CKMB 337 U/L, troponin T 2.95 ng/mL, total cholesterol 157 mg/dL,

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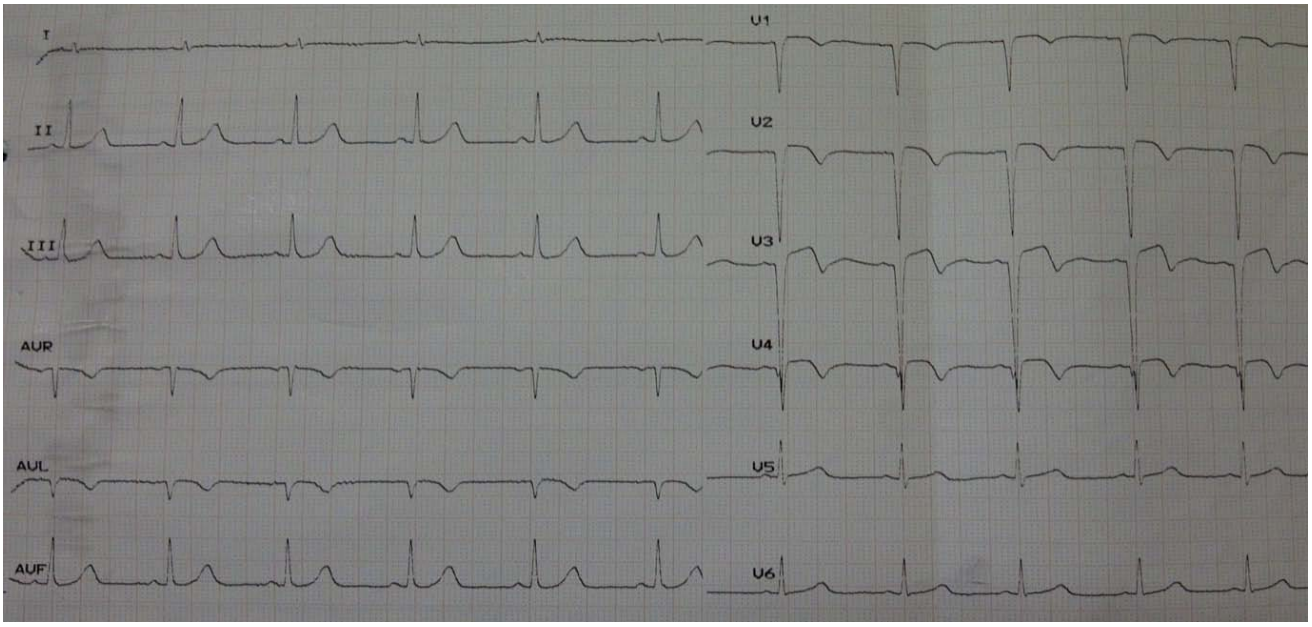


*Figure 1. Electrocardiogram (ECG)*

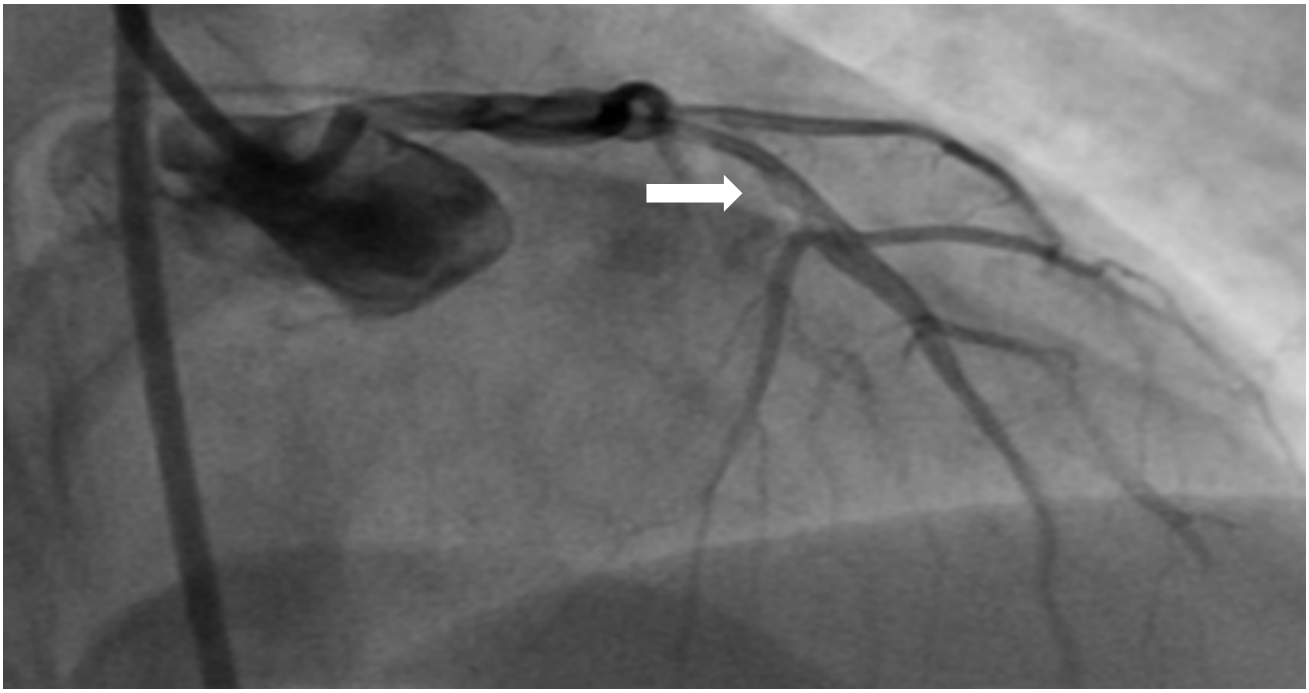

*Figure 2. Thrombus at left anterior descending (LAD)*

HDL-C 34 mg/dL, LDL-C 102 mg/dL, triglyceride 140 mg/dL, normal glucose tolerance test and kidney function. Echocardiography showed EDD 46 mm, ESD 33 mm, LVEF 39% (Simpson) and TAPSE 1.9 cm. Further examination by cardiac MRI (Figure 3-5) showed LVEF 48%, RVEF 55% with rest subendocardial defect at left anterior descending (LAD) area, intramural haemorrhagic at anterior region, and subendocardium scar formation at LAD area (non viable with LGE > 50% transmurality). Patient was discharged in stable condition on May, 14 2012 and was given double anti-platelet, simvastatin, nitrates, carvedilol, and ramipril.

Further evaluation using single photon emission computer tomography (SPECT) (Figure 6) showed partial reversible defect at basal anteroseptal, anterior, apicoseptal, and mid anterolateral. Fixed defect at mid

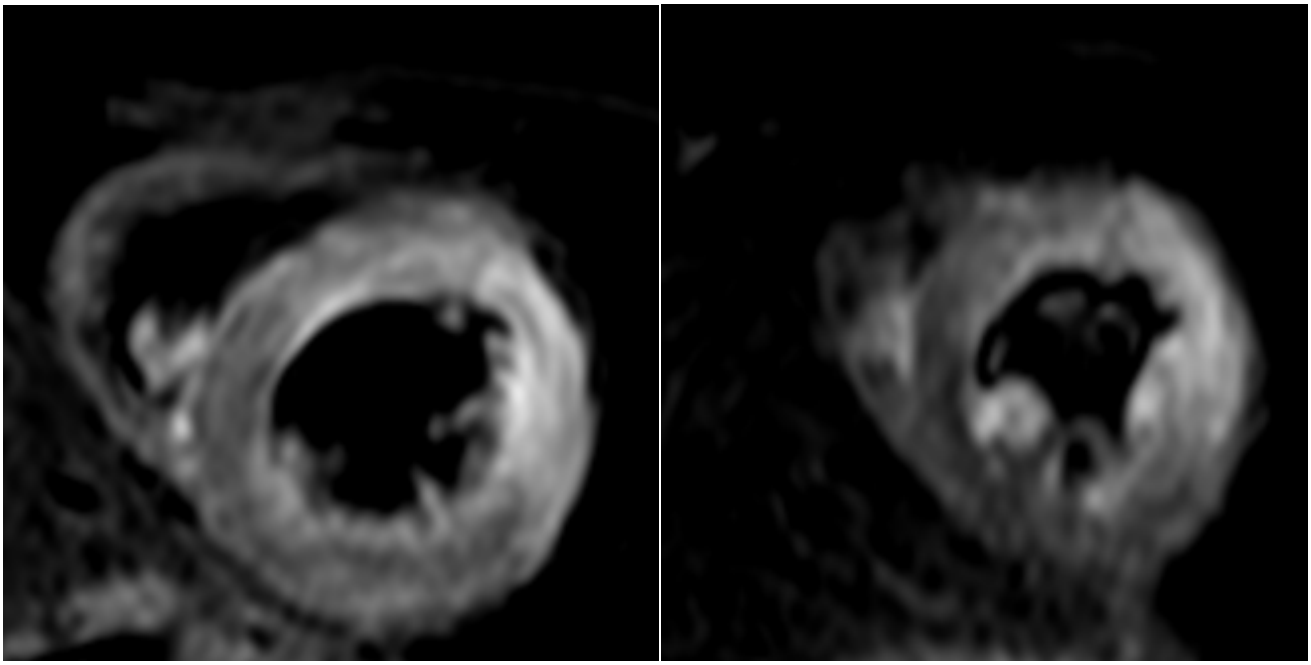

*Figure 3. T2-weighted showing myocardial oedema 'myocardium at risk'*

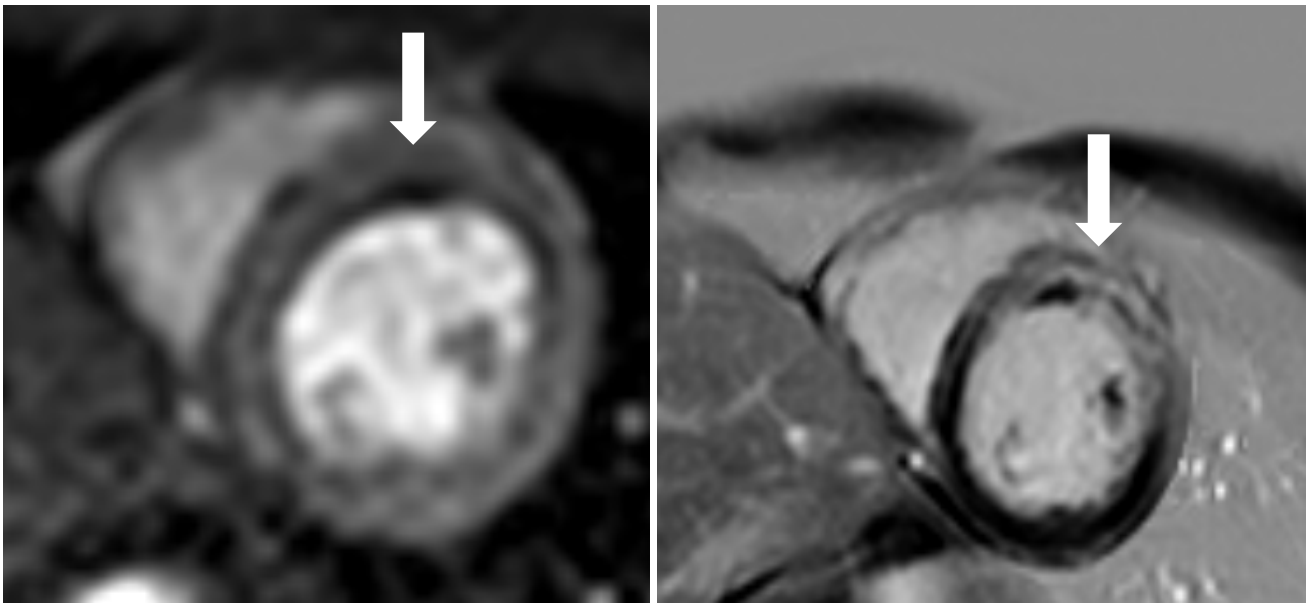

*Figure 4. Hypoenhancement in first pass perfussion and positive late gadolinium enhancement (LGE)*

anteroseptal, anterior, apex, and apicolateral. Perfussion defect size 40% at stress and 45% at rest. Rest LVEF 52% and stress LVEF 56%.

## DISCUSSION

### Myocardial ischemia

Cardiac MRI provides the assessment of function, perfusion, and tissue characterization in a highly reproducible manner during a single examination even in patients with acoustic window limitations.[7] Cine MRI for evaluation of cardiac volumes, mass, and systolic function is considered a gold standard compared with other imaging modalities.[6] The steady-state free precession (SSFP) sequences for cine images have replaced the older turbo gradient echo due to increased natural contrast between blood and endocardial border.[8,9] Regional myocardial function including wall thickening, evaluation and measures of myocardial strain may also be perfomed.[10]

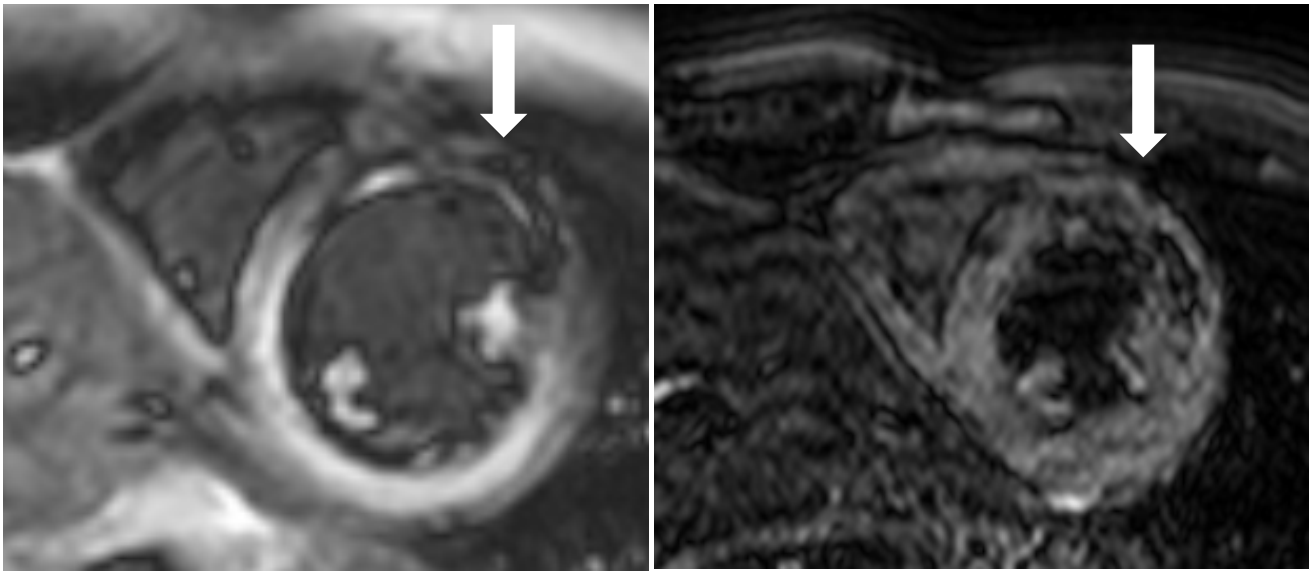

*Figure 5. Intramyocardial haemorrhage*

Myocardial oedema in the acute phase of myocardial infarction can be visualized as a bright signal on T2-weighted images, defining 'myocardium at risk'[11] (Figure 3). The major advantages of this technique are to distinguish chronic from acute infarction and to quantify the proportion of myocardial salvaged by comparing T2-weighted oedematous size and late enhancement images.[12,13]

Late gadolinium enhancement (LGE) images are T1-weighted inversion recovery sequences acquired about 10 min after intravenous administration of gadolinium. Gadolinium enhances its distribution volume in certain conditions such as necrotic or fibrotic myocardium (hyperenhancement).[3] The pattern of LGE is useful to differentiate post-infarction necrosis (subendocardial or transmural LGE) from fibrosis in nonischaemic dilated cardiomyopathies (mid-wall LGE, subepicardial LGE), or myocarditis (subepicardial or focal LGE) (Figure 4-right). There was subendocardial LGE in this patient suggested the result of post infarction necrosis. Delayed post-contrast sequences are currently used also to evaluate persistent microvascular dysfunction/damage: in the context of white LGE regions may coexist dark hypoenhanced areas, traditionally referred to as microvascular obstruction (Figure 5).[3,14]

In early phase of a coronary occlusion, the discrepancy between myocardial oxygen supply and demand lead to myocardial ischaemia and if persists, myocardial injury becomes irreversible and the necrosis extends from the subendocardium towards the subepicardium.[15] The final IS depends mainly on the extent of the so-called 'risk area', defined as the myocardial area related to an occluded coronary artery with complete absence of blood flow, either antegrade or collateral. Up until now, myocardium at risk has been measured by SPECT using technetium-based tracer.[3]

The most important application of MRI is the evaluation of 'salvaged myocardium'. Friedrich et al have compared T2-weighted images with LGE to visualize reversible and irreversible myocardial injury.[13] Thereafter, many clinical studies have evaluated the potential of MRI to assess myocardial oedema in comparison with well-established techniques.[16-19] The concept of reduction of total viable myocardium amount in proportion to the delay on time-to-reperfusion is demonstrated in vivo, the salvaged myocardium (the difference between area of increase T2 signal and area of LGE) is markedly reduced when reperfusion occurs > 90 min after coronary occlusion in contrast with patients with ≤ 90 min of delay.[20] We also discovered the same condition in this patient, since the patient came in 8 hours onset of AMI.

**Reperfusion injury**

Reperfusion injury consists of no-reflow phenomenon, microvascular obstruction, and haemorrhage. The assessment of microvascular damage had been performed using MRI first-pass perfusion and the delayed post-contrast sequences.[3] Currently, it is generally accepted that delayed hypoenhancement is less sensitive than first pass (Figure 4-left) and the persistence of a hypoenhancement on delayed contrast sequences (10–20 min after injection) seems to characterize a persistent microvascular damage.[14]

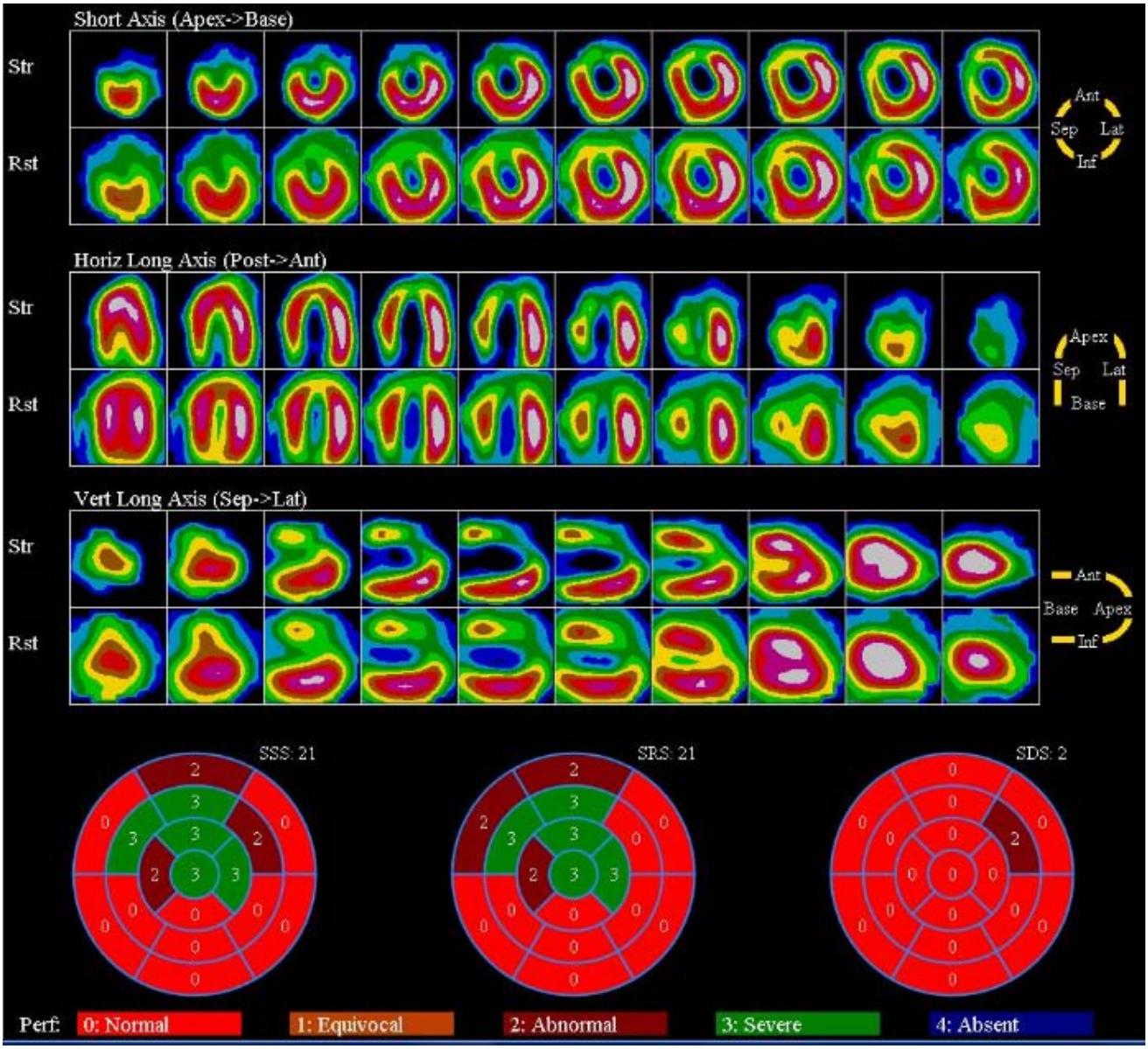


*Figure 6. Single photon emission computer tomography (SPECT)*

The typical presence of early systolic retrograde flow associated with no-reflow on intracoronary flow measurements was associated with the presence of microvascular obstruction assessed on LGE images.[21] Cardiac MRI features of reperfusion injury have been compared also with angiographic perfusion parameters like TIMI flow and myocardial blush grade (MBG). A study by Appelbaum et al[22] in 21 patients who underwent successful primary PCI and MRI, evidence of impaired perfusion at first pass was present in 90% of cases with post-PCI myocardial perfusion grade (0/1/2), but only in 18.2% with normal one. Porto et al[23] found a linear correlation in 27 patients with AMI between decreasing MBG and MRI signs of vascular obstruction.

Cardiac MRI features of no-reflow have prognostic significance in terms of clinical outcome and changes in LV volumes.[14,24] The 'no-reflow phenomenon' refers to absent distal myocardial reperfusion after a prolonged period of ischaemia, despite the culprit coronary artery's successful recanalization, and likely secondary to both luminal obstruction and external compression by oedema and haemorrhage.[25] The haemorrhagic AMI reflects the metamorphosis of ischaemic infarcts in the reperfusion era and are commonly observed after prolonged ischaemia once myocardial cell necrosis is well established.[26] This phenomenon was also found in this patient since he came with 8 hours onset of AMI.

Microvascular cell damage causes leakage of blood out of the injured vessel and the subsequent healing

process is characterized by haemoglobin degradation in which dark areas on post-contrast sequences indicate not only the presence of microvascular obstruction, but also of intramyocardial haemorrhage.[27] The extent of the hemorrhagic area correlates with the size of 'dark zones' on LGE sequences.[28,29] Patients with haemorrhagic AMI have shown a lower pre-PCI TIMI flow; moreover the area at risk, IS, and ratio of IS to area at risk were significantly larger.[4] Furthermore, the size of 'dark zones' on post-contrast images is larger and interestingly present in all patients with haemorrhagic AMI. The hypoenhancement on T2-weighted images, suggesting intramyocardial haemorrhage (Figure 5-right), is present in the majority of patients with dark zones on LGE (Figure 5-left) and also closely related to markers of IS and function.[30] Though TIMI flow 3 was seen in this patient during coronary angiography, there was no prove that reperfusion injury had not happened yet until we performed cardiac MRI. So, we should be prepared for the outcome of these microvascular cell damages.

**Viability**

The major determinant of final transmural necrosis and microvascular damage is the duration of ischaemia, as demonstrated in vivo by MRI.[31,32] The LGE represents a permanent memory of myocardial injury even after the acute phase of infarction.[33] IS on MRI appeared to diminish in size on follow-up.[14,34] Baks et al[35] evaluating the effects of primary PCI on early and late IS described a 31% decrease in LGE between 5 days and 5 months. Cardiac MRI in STEMI patients within 12 hours of primary PCI and at 6 months showed a reduction in LGE volume from 22 to 16%.[36]

IS within 1 week from AMI was directly related to LV remodelling and was a stronger predictor of future events than measures of LV systolic performance.[36-38] The occurrence of LV dysfunction at 6 months increased with greater LGE: a cut-off of ≥ 23% LGE measured on hyperacute MRI showed the best accuracy for late LV dysfunction (sensitivity 89%, specificity 74%).[36] In the assessment of myocardial viability in AMI patients, when the extent of LGE is < 50% the likelihood for functional recovery is efficient.[39] This patient had 34% LGE so we should manage the possibility of LV dysfunction in the near future and the extent of LGE was > 50%, therefore the likelihood for functional recovery was decreased.

The main advantage of MRI LGE is its spatial resolution of 1–2 mm, contrary to about 10 mm with SPECT scans.[40] Therefore, MRI can identify subendocardial necrosis when perfusion by SPECT appears unaltered.[41,42] SPECT detection of LGE-evidenced infarcts varied according to transmural extent: among segments with near transmural LGE (involving > 75% wall thickness) (Figure 4-right), all show evidence of infarct by SPECT, on the contrary segments with subendocardial LGE (involving < 50% wall thickness), SPECT detected infarct only in 53%. Evaluating patients early after AMI, cardiac MRI was significantly more sensitive than SPECT for the detection of small infarcts and infarction in non-anterior region.[42] SPECT showed the similar findings as MRI in this patient because the LGE in MRI was > 50% transmurality.

In conclusion, cardiac MRI can provide a wide range of clinically useful information in AMI by detecting not only location of transmural necrosis, infarct size, and myocardial oedema, but also showing in vivo important microvascular pathophysiological processes associated with AMI in the reperfusion era, such as intramyocardial haemorrhage and no-reflow. These impacts will help clinicians to manage and choose specific therapeutic strategies for AMI patients.

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