

Cardiovascular magnetic resonance imaging assessment of outcomes in acute myocardial infarction

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uniquely characterizes myocardial and microvascular injury in acute myocardial infarction (AMI), providing powerful surrogate markers of outcomes. The last 10 years have seen an exponential increase in AMI studies utilizing CMR based endpoints. This article provides a contemporary, comprehensive review of the powerful role of CMR imaging in the assessment of outcomes in AMI. The theory, assessment techniques, chronology, importance in predicting left ventricular function and remodelling, and prognostic value of each CMR surrogate marker is described in detail. Major studies illustrating the importance of the markers are summarized, providing an up to date review of the literature base in CMR imaging in AMI.

Key words: Myocardial infarction; Infarct; Cardiovascular magnetic resonance; Left ventricular remodelling; Prognosis

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Core tip: Cardiovascular magnetic resonance (CMR) imaging uniquely characterizes myocardial and microvascular injury in acute myocardial infarction (AMI). Contrast-enhanced CMR offers robust, validated and reproducible surrogate markers, providing an accurate representation of pathophysiology, assessment of myocardial function and injury, and predictive value for medium to long-term LV function, remodelling and prognosis following primary percutaneous coronary intervention for STEMI. These qualities significantly increase the statistical power of studies using CMR endpoints and has resulted in an exponential increase in AMI studies utilizing CMR based endpoints. An understanding of the role of CMR in the assessment of outcomes in AMI is of key importance not only to interventional and imaging cardiologists, but to the cardiology community as a whole.

Abstract

Cardiovascular magnetic resonance (CMR) imaging

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INTRODUCTION

Cardiovascular magnetic resonance (CMR) imaging uniquely characterises myocardial and microvascular injury in acute myocardial infarction (AMI), providing powerful surrogate markers of outcomes. The last 10 years have seen an exponential increase in studies utilising CMR based endpoints in patients with AMI undergoing primary percutaneous intervention. This article provides a contemporary, comprehensive review of the powerful role of CMR imaging in the assessment of outcomes in AMI. The theory, assessment techniques, chronology, importance in predicting left ventricular function and remodelling, and prognostic value of each CMR surrogate marker is described in detail. Major studies illustrating the importance of the markers are summarised, providing an up to date review of the literature base in CMR imaging in AMI.

MARKERS OF OUTCOMES FOLLOWING PRIMARY PERCUTANEOUS CORONARY INTERVENTION IN AMI

Prognostic studies using clinical outcomes, in particular mortality require large sample sizes. Surrogate biomarkers of outcome are directly measured alternative endpoints used as a substitute for biological processes and clinical outcomes^[1,2]. CMR imaging uniquely characterises myocardial and microvascular injury in AMI due to its accuracy, reliability and validity (Figure 1)^[2-4]. This significantly increases the statistical power of studies, allowing sample size requirements to be reduced. CMR data are strong surrogate markers of outcome following primary percutaneous coronary intervention (PPCI) in acute ST-segment elevation MI.

LV EJECTION FRACTION AND VOLUMES IN AMI

Background

In the medium-term following STEMI, LV end-diastolic volume (LVEDV) increases, LV end-systolic volume (LVESV) decreases^[5-7] and there can be compensatory hypertrophy of remote myocardium^[8,9] in order to preserve stroke volume and ejection fraction (LVEF). Adverse remodelling results from an inability of the heart to maintain geometry post MI in the context of large infarcts and increased wall stresses^[10,11]. An increase in LVEDVI > 20%^[12,13] and increase in LVESVI > 15%^[14] at follow-up are the most commonly used criteria for adverse remodelling.

CMR assessment of LV volumes and ejection fraction

CMR is the gold standard modality for the assessment of ventricular function and volumes. It has higher spatial

resolution than single-photon emission computed tomography (SPECT) (approximately 1.8 mm × 1.8 mm × 8 mm vs 10 mm × 10 mm × 10 mm)^[15], and suffers from little subjectivity or reliance on patient body habitus^[16].

Volumes and mass are assessed on analysis of a 3D cine stack of short-axis biventricular contiguous slices. Modern cine sequences use breath-hold, electrocardiographic-gated, segmented steady-state free precession (SSFP) to produce high spatial resolution images with excellent myocardium-blood contrast. Regional systolic function can alternatively be assessed using wall motion scoring^[17].

CMR studies have demonstrated that recovery of LVEF occurs relatively early post STEMI. Ripa showed that improvement in LVEF and systolic wall thickening occurred by 1 mo, with no further change at 6 mo^[5]. The majority of improvement in LVEF occurred between day 2 and 1 wk in the study by Mather^[18], with a final increase by 3 mo. Beek showed that 55% of segments with initially impaired systolic wall thickness improved at 13-wk^[19]. Ganame *et al*^[20] and Dall'Armellina *et al*^[21] however showed that LVEF underwent no significant change by 6 and 12 mo post PPCI respectively. This may be because their subjects sustained less myocardial damage, represented by relatively preserved LVEF and thus lower potential for improvement^[21].

Volumetric changes occur more slowly. Ripa *et al*^[5] showed a continued increase in LVEDV and reduction in LVESV until 6 mo. Engblom *et al*^[7] demonstrated similar sequelae to 12-mo. Ganame showed progressive significant changes in LVEDV and LVESV and resulting LV sphericity at all timepoints to 12 mo^[20]. These studies have important implications for optimising timing of follow-up CMR studies assessing remodelling.

The degree of impairment of LVEF and changes in volume depend on a number of CMR-based markers including infarct size (IS)^[22], microvascular obstruction (MVO)^[23,24], intramyocardial haemorrhage (IMH)^[25] and myocardial salvage [non-infarcted proportion of ischaemic area at risk (AAR)]^[26,27]. Anterior STEMI results in larger IS and lower LVEF due to the greater ischaemic AAR^[28].

Prognostic importance of LVEF and volumes in AMI

Norris *et al*^[29] and White *et al*^[30] first illustrated the prognostic importance of LVEF (strongest independent predictor of survival at 3.5 years) and LVESV (only independent predictor of long-term mortality at 6 years) respectively, using invasive ventriculography. Burns first demonstrated the prognostic importance of LVEF and LV volumes and their strong correlation with each other, using radionuclide analysis^[31].

A large evidence base has emerged for the prognostic impact of impaired systolic function based on reduced CMR-derived LVEF (Table 1).

In addition to LVEF-based global systolic function, Bodi demonstrated that the number of dysfunctional segments on CMR at 1-wk post STEMI was an independent predictor of combined MACE at a median follow-up of 553 d^[38]. The evidence base for the prognostic importance of LV volumes is largely historical, based on large echocardiographic

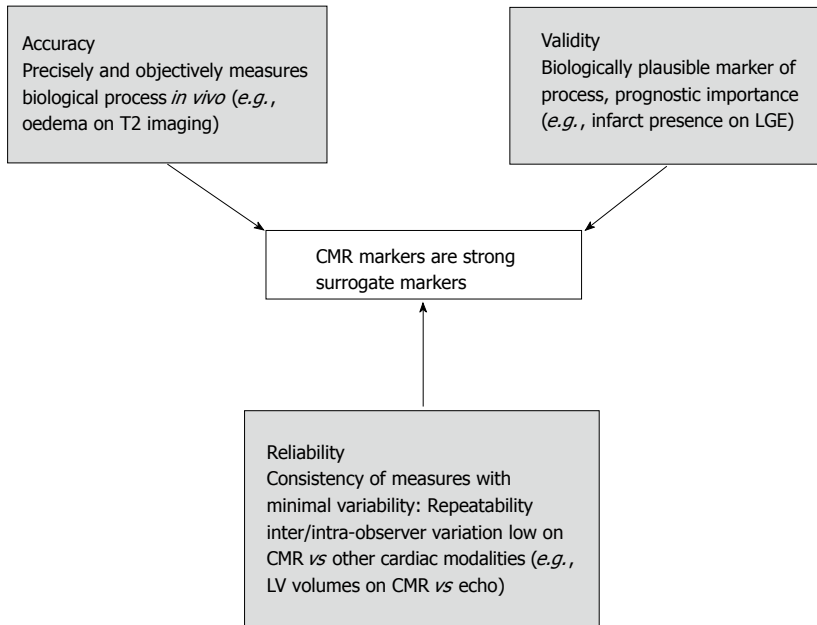


Figure 1 Cardiovascular magnetic resonance markers are ideal surrogate biomarkers for the assessment of revascularisation in acute myocardial infarction^[2-4]. CMR: Cardiovascular magnetic resonance; AMI: Acute myocardial infarction; LGE: Late gadolinium enhancement.

Table 1 Cardiovascular magnetic resonance studies illustrating the prognostic importance of left ventricular ejection fraction in acute myocardial infarction

Ref.	Year	n	CMR time	Main findings	Follow-up
El Aidi <i>et al</i> ^[32]	2014	25497	N/A	Meta analysis of prognostic value of CMR surrogate markers. LVEF was only IP for MACE (HR 1.05 per -5%)	N/A
Husser <i>et al</i> ^[33]	2012	304	7 d	LVEF was IP for MACE (HR 0.95 for each +1% LVEF)	140 wk
Eitel <i>et al</i> ^[34]	2011	208	3 d	LVEF was IP for MACE (HR 0.95 for each +1% LVEF)	18.5 mo
Amabile <i>et al</i> ^[35]	2010	114	6 d	LVEF was IP for MACE (HR 0.96 for each +1% LVEF)	12 mo
de Waha <i>et al</i> ^[36]	2010	438	3 d	LVEF was IP for MACE (OR 1.63) and all-cause mortality (OR 2.51)	19 mo
Cochet <i>et al</i> ^[37]	2009	127	3-7 d	LVEF of < 40% was IP for MACE (OR 1.20)	12 mo
Hombach <i>et al</i> ^[6]	2005	110	6 d	LVEF was IP for 9 mo MACE (<i>P</i> = 0.006)	225 d

CMR time: Mean/median time of CMR post acute STEMI; MACE: Major adverse cardiovascular events; IP: Independent predictor; LVEF: Left ventricular ejection fraction; CMR: Cardiovascular magnetic resonance; N/A: Not available.

Table 2 Studies illustrating the prognostic importance of left ventricular volumes and adverse left ventricular remodelling in acute myocardial infarction

Ref.	Year	n	Modality	Main findings	Follow-up
Ahn <i>et al</i> ^[13]	2013	135	Echo	Adverse LV remodelling (> 20% inc. LVEDV) at 6 mo was IP 3 yr MACE. MACE rate approximately 25% in patients with adverse LV remodelling <i>vs</i> approximately 6% in non-remodelled patients	981 d
Hombach <i>et al</i> ^[6]	2005	110	CMR	Baseline LVEDV was IP for MACE (<i>P</i> = 0.038)	225 d
St John Sutton <i>et al</i> ^[39]	2003	512	Echo	Percentage change in LV area (surrogate for LV volume) between baseline echo and follow-up at 12 mo was IP for ventricular ectopy and VT	24 mo
Bolognese <i>et al</i> ^[12]	2002	284	Echo	Baseline LVESV was IP for cardiac death and MACE. Components of MACE higher in patients with adverse remodelling (> 20% inc. LVEDV: Mortality 14% <i>vs</i> 5%, MACE 18% <i>vs</i> 10%)	5 yr
Otterstad <i>et al</i> ^[40]	2001	712	Echo	Increase in LVESV between acute scan at 7 d and echo at 3 mo strongest IP for MACE	24 mo
St John Sutton <i>et al</i> ^[41]	1994	512	Echo	LV end-diastolic area (RR 1.1) and LV end-systolic area (RR 1.1) on baseline echo, and % change in LV area at 12 mo echo (RR 1.55) were strongest IPs for MACE	12 mo
White <i>et al</i> ^[30]	1987	605	LV gram	LVESV of LV gram at 4 wk was strongest IP of long-term mortality (<i>P</i> < 0.0001)	78 mo

MACE: Major adverse cardiovascular events; IP: Independent predictor; LVEDV: Left ventricular end-diastolic volume; LVESV: Left ventricular end-systolic volume; Modality: Modality of LV volume assessment (CMR: Cardiovascular MRI; Echo: Echocardiography; LV gram: LV contrast angiography).

and radionuclide studies, demonstrating the negative prognostic impact of ventricular dilatation and remodelling as summarised in Table 2.

Negative LV remodelling has demonstrated prognostic importance in two studies, based on the cut-off of LVEDVI dilation of > 20% at 6-mo follow-up^[12,13].

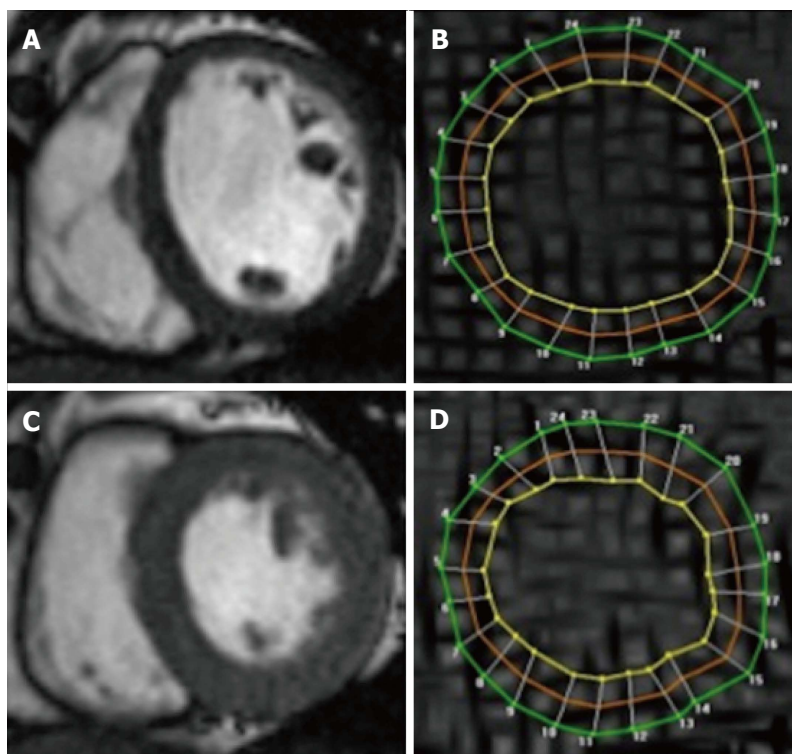


Figure 2 Cardiovascular magnetic resonance assessment of strain using tissue tagging. Cine SSFP images in end-diastole (A) and end-systole (C), with corresponding Spatial Modulation of Motion (SPAMM) tagged images (B and D). Grid lines (tags) are visible and contours drawn at 3 myocardial levels [green (epicardial), red (mid myocardial), yellow (endocardial)] allow tracking of myocardial motion and strain (circumferential), here using Harmonic Phase Analysis.

Recently, left ventricular global performance index has been proposed as a CMR marker of cardiac performance, incorporating LVEF, LV volumes and mass. It has been assessed in one study in STEMI and correlated strongly with IS, MSI, MVO and IMH extent, and had incremental prognostic value to LVEF in predicting 12-mo MACE^[42]. Further work is needed to investigate its prognostic value in STEMI.

MYOCARDIAL STRAIN IN AMI

CMR-measured myocardial strain (tissue deformity) is the gold standard non-invasive measure of systolic and diastolic myocardial function^[43]. Circumferential strain (Ecc) describes shortening of fibres (contraction) in a short-axis plane tangential to the epicardium; longitudinal strain (E_{ll}) describes shortening in the long axis, and radial strain (Err) describes lengthening (thickening) of fibres towards the centre of the ventricle. Torsion is wringing of the ventricle caused by clockwise rotation at the base, and anticlockwise at the apex.

Strain offers greater accuracy in detecting myocardial dysfunction compared with global (LVEF) and regional (visual wall-motion scoring, segmental wall thickening)^[44] measures.

CMR assessment of myocardial strain

In 1989, Axel *et al*^[45] developed a T1 spoiled gradient echo sequence, creating “tags” formed by saturation of thin myocardial lines running in perpendicular directions in-plane to form a myocardial grid. These lines act as tissue markers, tracking myocardial deformation as shown in Figure 2. Peak systolic strain and peak diastolic strain

rate (relaxation rate of strain) provide very sensitive measures of systolic and diastolic function respectively. Its accuracy has been validated on comparison with sonomicrometry^[46,47]. Harmonic Phase Analysis (HARP) is currently the most widely used CMR strain method^[48].

Feature tracking (FT) has been introduced as an alternative method to tagging for assessing strain on CMR. FT tracks anatomical features of interest along contour lines on routinely acquired SSFP cine images analogous to echocardiographic Speckle Tracking, obviating the need for additional tagging sequences^[49]. FT-derived strain has been compared to tagging in acute STEMI and shown greater feasibility, accuracy and observer agreement^[50] and remains an exciting prospect.

CMR LV strain as a predictor of LV function and remodelling in AMI

Strain could improve our understanding of the mechanics underlying LV dysfunction associated with prognostic CMR surrogate markers of myocardial damage in STEMI (e.g., MVO, IMH, oedema).

Systolic function is also in remote (non-infarcted) segments, and LV mechanics outside of the infarct zone are also affected during infarction and contribute to remodelling^[44,51,52]. MVO had the highest predictive value for persistent dysfunction on circumferential strain at 7-mo post STEMI and may result in systolic dysfunction due to direct mechanical effects (myocardial stiffness)^[53]. Baseline segmental circumferential strain was the strongest predictor of segmental functional recovery at 3-mo in a model containing infarct transmural and MVO^[54]. FT-derived global circumferential strain assessed acutely post PPCI was recently shown to correlated strongly with

Table 3 Studies illustrating the prognostic importance of left ventricular strain in acute myocardial infarction

Ref.	Year	n	Modality	Main findings	Follow-up
Ersbøll <i>et al</i> ^[56]	2014	1048	TTE	(E-prime divided by peak early diastolic strain rate) strongest IP of MACE and death	29 mo
Ersbøll <i>et al</i> ^[57]	2013	849	TTE	GLS was IP of MACE	30 mo
Hung <i>et al</i> ^[58]	2010	610	TTE	GLS and strain-rate, and GCS and strain-rate IPs for MACE in model with WMS, LVEF	25 mo
Antoni <i>et al</i> ^[59]	2010	659	TTE	GLS (HR 1.2) was IP of mortality. LVEF, wall-motion score and Tissue Doppler mitral valve inflow not	21 mo

TTE: Transthoracic echocardiography; GLS: Global longitudinal strain; MACE: Major adverse cardiovascular events; IP: Independent predictor; HR: Hazard ratio; LVEF: Left ventricular ejection fraction.

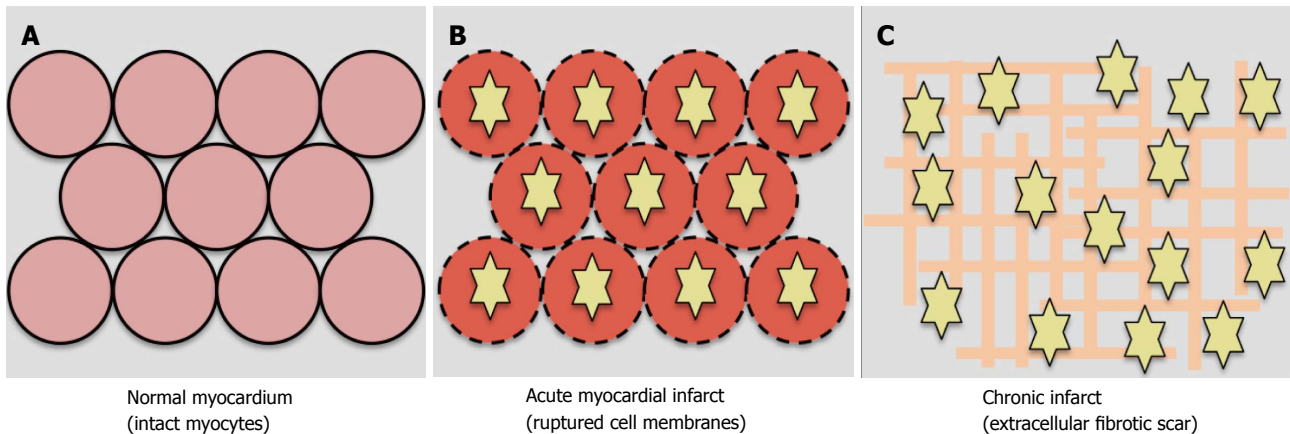


Figure 3 Mechanism of late gadolinium enhancement. Gadolinium is extracellular. A: In normal myocardium, gadolinium washes out approximately 10 min post administration and there is no late gadolinium enhancement (LGE); B: In acute infarct, gadolinium (yellow stars) enters ruptured cell membranes and causes LGE; C: In chronic infarct, LGE results from increased extracellular space due to fibrotic scar deposition.

acute IS on late gadolinium enhancement (LGE) imaging ($r = 0.75$) and final LVEF at 6 mo ($r = -0.71$). Global circumferential strain was a stronger predictor of functional recovery (LVEF > 50%) at 6 mo than global longitudinal strain, age, diabetes and baseline LVEF, and was of similar predictive value to acute IS [AUC 0.86 (Ecc) vs 0.92 (IS)]^[55].

Prognostic importance of LV strain in AMI

The evidence base for the prognostic importance of LV strain post STEMI is currently based on echocardiographic studies demonstrating that global longitudinal predicts medium and long-term using Speckle Tracking analysis as summarised in Table 3.

INFARCT SIZE IN AMI

Background

The "ischaemic cascade" is the sequence of pathophysiological effects developing immediately following coronary occlusion. Aerobic respiration loses efficiency resulting in cellular oedema. With increasing ischaemic time, cell membranes rupture. Following healing, necrotic cells are replaced by extracellular collagen deposition (scar). The acute and chronic phases are characterised by increased myocardial extracellular volume^[60-62].

CMR assessment of IS in AMI

Gadolinium contrast agents are large extracellular

molecules (Figure 3). Infarct can be visualised on T1-weighted imaging approximately 10 min after intravenous contrast administration, known as LGE imaging.

In acute infarct, LGE results from gadolinium entering ruptured cell membranes. In chronic infarction, LGE results from increased extracellular space due to collagen deposition and prolonged washout due to reduced capillary density within myocardium^[60,63]. Gadolinium shortens T1, causing infarcted myocardium to appear bright, and normal myocardium to appear black (Figure 4)^[63,64]. Normal myocardium is progressively nulled using the appropriate inversion time to provide optimal contrast between infarct and normal myocardium.

Typically, a high spatial resolution of approximately 1.4 mm × 1.6 mm × 6-8 mm is achieved^[15]. IS is typically expressed as a percentage of total LV mass. Delineation of infarct can be performed visually (manual quantification)^[6,9,22], however most groups use semi-automated methods to reduce observer variability. These include enhancing myocardium exceeding a pre-defined signal intensity (SI) threshold, typically > 2-6 standard deviations above that of remote (non-infarcted) myocardium^[2,65]. Currently, the semi-automated full-width at half-maximum (FWHM) method is commonly used^[66-70], defining infarct as myocardium with SI > 50% of the peak SI in the infarct core. Amado demonstrated that FWHM had the highest interobserver agreement and closest correlation with TTC-stained infarct in a dog model of acute infarction ($r^2 = 0.94$), compared with standard

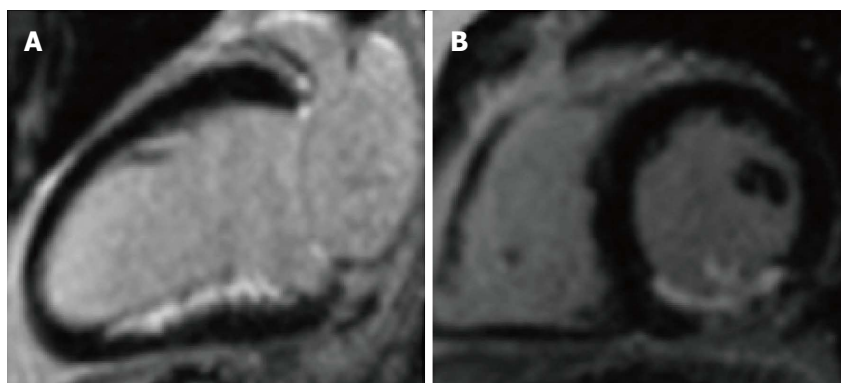


Figure 4 Late gadolinium enhancement of acute infarct. Infarct appears white (enhanced) in the inferior wall, with unaffected myocardium black (nulled). A: 2-chamber long-axis view; B: Short-axis view, mid ventricular level. The posteromedial papillary muscle is also infarcted in the short-axis view.

Table 4 Temporal changes in cardiovascular magnetic resonance-derived infarct size in acute myocardial infarction

Ref.	Year	n	CMR times post STEMI	Relative LGE IS reduction	LGE method	Main findings
Carrick <i>et al</i> ^[74]	2016	30	8 h → 3 d → 10 d → 7 mo	26%	Automated	Significant decrease d3 to d10 (20% ± 13% to 14% ± 10% LV mass). No change at 7 mo
Dall'Armellina <i>et al</i> ^[21]	2011	30	2 d → 6 mo	22%	> 2SD	IS reduced at times from 27% ± 15% LV mass 24 h post PPCI, to 21% ± 11% at 6 mo
Mather <i>et al</i> ^[18]	2011	48	2 d → 1 wk → 30 d → 3 mo	37%	> 2SD	27% IS drop between d2 and d7 post PPCI, no change at 3 mo
Ganame <i>et al</i> ^[20]	2011	58	3 d → 4 mo → 12 mo	45%	Manual	33% decrease IS d3 and 4 mo then no further decrease at 12 mo
Ibrahim <i>et al</i> ^[9]	2010	17	1 d → 1 wk → 1 mo → 6 mo	37%	Manual	34% reduction in IS from d2 to 1 wk, then no further change at 1 and 6 mo
Engblom <i>et al</i> ^[7]	2009	22	1 d → 1 wk → 12 mo	40%	Automated	28% reduction in IS between d1 and 1 wk
Ripa <i>et al</i> ^[5]	2007	58	2 d → 1 mo → 6 mo	30%	Manual	14% % reduction in IS from d2 to 1 mo
Hombach <i>et al</i> ^[6]	2005	110	6 d → 9 mo	28%	Manual	28% reduction in IS from d6 to 9 mo

LGE method: SD: Standard deviations; Total LGE IS Overest: Relative overestimation of final IS (last timepoint) on acute CMR; CMR: Cardiovascular magnetic resonance; LGE: Late gadolinium enhancement; IS: Infarct size; PPCI: Primary percutaneous coronary intervention.

deviation methods^[66]. This may be because FWHM is less prone to IS overestimation in the presence of oedema, and partial volume effects giving rise to intermediate signal intensities^[18,71]. Comparing techniques in STEMI patients showed that FWHM quantification had the lowest intraobserver and interobserver variability, and greatest agreement with LVEF^[72].

CMR measurement of IS on LGE is well validated^[63,64]. Kim demonstrated that IS in dog myocardium on *ex-vivo* CMR corresponded closely with IS derived from tetrazolium (TTC) staining ($r = 0.99$)^[15,64]. LGE has higher sensitivity for infarct detection compared with SPECT. In an experimental model of MI, CMR LGE detected 92% of all segments with subendocardial infarction (< 50% transmural) compared with only 28% with SPECT^[15]. In patients with MI, SPECT only detects approximately 50% of the infarcts seen on LGE. The superior sensitivity is due to the increased spatial resolution and reproducibility of CMR^[60].

Since gadolinium is distributed throughout the extracellular space, gadolinium contrast agents are not specific to necrosis. Acutely, the area of LGE detects not only necrotic cells but also the increased (oedematous) interstitium surrounding viable cells, and thus can over-

estimate true IS. Studies of IS chronology in humans corroborate this (Table 4). Indeed, severely dysfunctional segments with minimal myocardial salvage early post STEMI can show significant functional improvement at follow-up^[73].

The majority of IS reduction occurs relatively early post STEMI, particularly by 1 wk. Indeed IS assessed at 1 wk has been shown to closely correlate with final IS^[7,9,18]. Overestimation of necrosis by LGE-derived IS early post STEMI is due to a combination of oedema, infarct resorption and partial volume effects. Oedema results in an overestimation of LGE IS due to increased extracellular water content and thus volume of distribution of contrast agent^[66,75].

Infarct resorption results from the healing process where collagenous scar tissue is produced to provide stability and tensile strength to necrotic myocardium^[7,11]. This was confirmed in a canine model where a 3.4-fold decrease in infarct volume was seen between day 3 and 8-wk post infarct on *ex-vivo* LGE and TTC-stained slices^[64]. The degree of infarct resorption has been shown to be proportional to initial IS ($r = 0.65$) and presence of LV remodelling ($r = 0.41$)^[10]. The greater degree of infarct resorption relative to total myocardial

Table 5 Cardiovascular magnetic resonance studies illustrating importance of segmental late gadolinium enhancement extent and functional recovery in acute myocardial infarction

Ref.	Year	n	LGE method	Cutoff (LGE)	Main findings	Time of CMR 1	Time of CMR 2
Khan <i>et al</i> ^[85]	2016		FWHM	50% SEE	SEE strong predictor of segmental functional improvement (AUC 0.840) and normalisation (AUC 0.887)	2 d	9 mo
Wong <i>et al</i> ^[54]	2014	45	FWHM	50% SEE	Inverse relationship between TEE and likelihood of functional recovery on WMS at 24 wk (area under curve 0.68)	8 d	13 wk
Natale <i>et al</i> ^[86]	2011	46	2SD	50% TEE	Inverse relationship TEE and likelihood of functional recovery on SWT (93% sens, 75% spec)	5 d	20 wk
Engblom <i>et al</i> ^[7]	2008	22	Manual	50% TEE	Inverse relationship between TEE and functional recovery on WMS	7 d	24 wk
Shapiro <i>et al</i> ^[87]	2007	17	Manual	50% SEE	Inverse relationship between TEE and likelihood of functional recovery on WMS at 26 wk. Odds-ratio of functional recovery 0.2 with each SEE quartile	6 d	26 wk
Kitagawa <i>et al</i> ^[88]	2007	18	2SD	50% TEE	Inverse relationship between TEE and functional recovery. 31% segments > 50% TEE still improved	5 d	39 wk
Janssen <i>et al</i> ^[89]	2006	67	Manual	50% TEE	Inverse relationship between TEE and functional recovery on WMS at 12w (51%-75%: 39% segments improved, 76%+: 21% improved)	4 d	12 wk
Motoyasu <i>et al</i> ^[90]	2004	23	2SD	50% TEE	Inverse relationship between SEE and functional recovery on SWT	25 d	24 wk
Beek <i>et al</i> ^[19]	2003	30	6SD	50% SEE	Inverse relationship between SEE and functional recovery on WMS	7 d	13 wk

WMS: Wall motion scoring; SWT: Systolic wall thickening; TEE: Transmural extent of enhancement; SEE: Segmental extent of enhancement; SD: Standard deviations.

Table 6 Cardiovascular magnetic resonance studies illustrating importance of infarct size on left ventricular function and remodelling in acute myocardial infarction

Ref.	Year	n	LGE method	Main findings	Time post STEMI of predictive CMR	Follow-up
Ahn <i>et al</i> ^[13]	2013	135	Manual	IS strongest IP of LVR in model with LVEF and MI location	7 d	6 mo (echocardiogram)
Husser <i>et al</i> ^[33]	2012	304	> 2SD	IS IP of LVR in model incl. LVEF, IS, LV vols, MVO	6 d	189 d
Monmeneu <i>et al</i> ^[91]	2012	118	> 2SD	No. segments > 50% transmural IP for LVR	6 d	6 mo
Ezekowicz <i>et al</i> ^[92]	2010	64	Manual	IS strongest IP of LVEF in model with MVO, troponins	7 d	3 mo
Ganame <i>et al</i> ^[25]	2009	98	Manual	IS strongest IP of LVR (>> MVO, AAR, Troponin-I)	2 d	6 mo
Bodi <i>et al</i> ^[93]	2009	214	> 2SD	Extent of transmural necrosis (no. segments > 50% TEE) strongest IP for LV recovery (+ > 5% LVEF)	7 d	6 mo
Wu <i>et al</i> ^[94]	2008	122	Manual	IS extent only IP for LVEF and LVR	2 d	4 mo
Hombach <i>et al</i> ^[6]	2005	110	Manual	IS extent IP of LVR in model with MVO, % transmural	6 d	225 d

IS: Infarct size; IP: Independent predictor; LVR: LV remodelling; LVEDVI: Left-ventricular end-diastolic volume index; LVEDVI: Left-ventricular end-systolic volume index; LVEF: Left ventricular ejection fraction; MVO: Microvascular obstruction; SD: Standard deviation.

mass and volume results in an inability to maintain LV geometry in light of mechanical stresses post STEMI, resulting in adverse LV remodelling and sphericity^[10,76].

Factors known to affect IS include AAR extent^[77-79]; collateral flow to the AAR^[79,80]; MVO^[81]; time to reperfusion^[82] and hyperglycaemia^[83].

CMR IS as a predictor of LV function and remodelling in AMI

Segmental function: Kim illustrated in stable patients awaiting revascularisation, that LGE transmural strongly predicted recovery of systolic function in dysfunctional segments. Only 2% of segments with > 75% transmural improved after revascularisation^[84]. Segmental extent of LGE has also been shown to negatively predict functional

recovery in dysfunctional segments following PPCI for acute STEMI, as summarised in Table 5.

Global function: IS is a powerful independent predictor of global LV function and adverse LV remodelling in the medium to long-term post STEMI as summarised in Table 6.

Prognostic importance of CMR-derived IS in AMI

The goal of STEMI management is early reperfusion in order to minimise IS and thus maximise myocardial salvage^[95]. There is a strong evidence base for the prognostic importance of CMR-derived IS post STEMI, as summarised in Table 7. IS strongly predicts medium to long-term clinical outcomes.

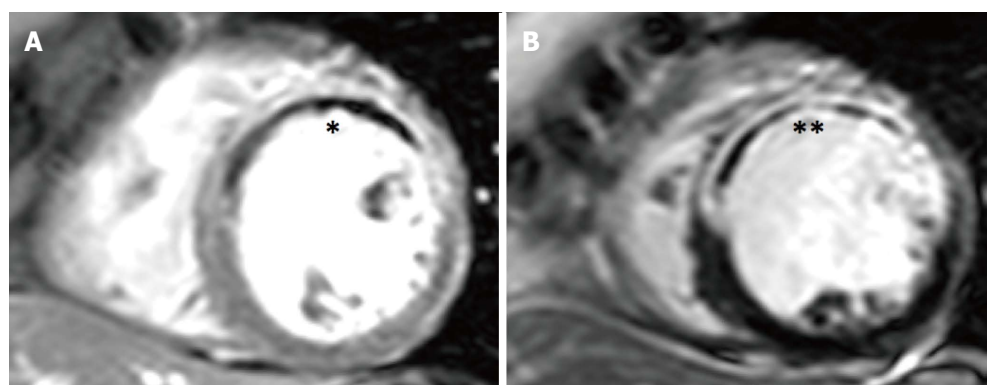


Figure 5 Early and late microvascular obstruction on cardiovascular magnetic resonance. A: Early gadolinium imaging at 1-min post contrast with hypoperfusion in anteroapical, anterior and anterolateral segments, consistent with early MVO (E-MVO, *); B: Corresponding late gadolinium image showing transmural infarction with a hypointense late MVO core (L-MVO, **) co-localising with E-MVO. MVO: Microvascular obstruction.

Table 7 Cardiovascular magnetic resonance studies illustrating the prognostic importance of infarct in acute myocardial infarction

Ref.	Year	n	LGE method	Main findings	CMR timepoint	Follow-up
Husser <i>et al</i> ^[96]	2013	250	> 2SD	Extent of transmural infarction (no. of segments > 50% transmural) only IP for MACE at 6 mo	7 d	163 wk
Izquierdo <i>et al</i> ^[97]	2013	440	> 2SD	IS was IP for AACEs (arrhythmic cardiac events: Sudden death, VT, VF, ICD shock) in model including LVEF, hypertension	7 d	123 wk
Eitel <i>et al</i> ^[34]	2011	208	> 5SD	IS was IP of MACE at 19 mo in model including MVO, LVEF, MSI, Killip, TIMI post-PPCI	3 d	18.5 mo
Miszalski-Jamka <i>et al</i> ^[98]	2010	77	Manual	LV transmural index IP (HR 1.03) and IS (HR 1.03) IPs for MACE in a model containing RVEF and RV IS	“3-5 d”	1150 d
Larose <i>et al</i> ^[67]	2010	103	FWHM	IS strongest IP for MACE (HR 1.36) in model containing LVEF, CK. LGE > 23% had HR 6.1 for MACE	4.5 h	2 yr
Bodi <i>et al</i> ^[38]	2009	214	> 2SD	Extent of transmural infarction (no. of segments > 50% transmural) IP for MACE (HR 1.35 if > 5 segs)	7 d	553 d
Wu <i>et al</i> ^[99]	2008	122	Manual	IS only IP of 2 yr MACE in model containing LVEF, LVESVI	2 d	538 d

LGE: Late gadolinium enhancement; FWHM: Full-width half-maximum; SD: Standard deviations; MACE: Major adverse cardiovascular events; LVEF: Left ventricular ejection fraction; PPCI: Primary percutaneous coronary intervention; LGE method (LGE quantification method): SD: Standard deviations; FWHM: Full-width half-maximum.

MVO IN AMI

Background

Despite prompt IRA recanalization, perfusion of the microcirculatory bed does not always ensue. Histopathological studies have demonstrated that the infarct core (endocardial) perishes first as necrosis spreads transmurally towards the epicardium. This is known as the “wavefront theory”^[100]. At the infarct core, necrosis occurs rapidly with myocardial and capillary endothelial cells perishing simultaneously. Capillaries can become obstructed by cellular debris, resulting in non-perfusion of the infarct core, despite IRA patency^[101]. This is known as MVO and can be indicated at angiography, as “no reflow”^[101].

CMR assessment of MVO in AMI

Three CMR methods demonstrate MVO (Figure 5). MVO extent is typically expressed as a percentage of LV mass: (1) Qualitative first-pass rest perfusion. A modified version involves quantification of myocardial blood flow (SI-time curve) and time to 50% of maximal SI^[102,103]; (2) Hypoperfusion on inversion recovery images between 1-3

min post contrast. A fixed inversion time of approximately 440 ms nulls MVO and retains intermediate signal in normal myocardium. This is known as “early MVO (E-MVO)”^[28,104]; and (3) Hypointensity within infarct core on LGE due to absence contrast perfusion, known as “late MVO (L-MVO)”. L-MVO occurs in upto 60% of patients on CMR within the first week post STEMI^[5,6,18,20]. This is the preferred method of MVO demonstration in contemporary clinical practice and research.

L-MVO extent is maximal at 48 h post infarct^[8,18], and then decreases. It exists for at least 1 wk, and for up to 1 mo^[8,18] and then resolves in the medium-term in humans (Table 8). Animal models corroborate these findings^[105,106].

The extent of MVO on CMR has been shown to correlate with IS^[82,94,107,108], oedema, IMH, TIMI-flow pre PCI^[35,109] and time to reperfusion^[35,82,110].

CMR MVO as a predictor of LV function and remodelling in AMI

L-MVO is a strong independent predictor of medium-term LV function and adverse remodelling (Table 9). It

Table 8 Temporal changes in cardiovascular magnetic resonance late microvascular obstruction in acute myocardial infarction

Ref.	Year	n	CMR timepoints	LGE method	Main findings
Carrick <i>et al</i> ^[74]	2016	30	8 h → 3 d → 10 d → 7 mo	Auto	L-MVO in 20%, peaked early at 8 h and stable at d3. Decreased by d10, absent at 7 mo
Mather <i>et al</i> ^[18]	2011	48	2 d → 1 wk → 30 d → 3 mo	> 2SD	L-MVO in 60%, peak at d2. Decrease at subsequent points. L-MVO absent at 3 mo
Ganame <i>et al</i> ^[20]	2011	58	3 d → 4 mo → 12 mo	Manual	L-MVO in 64%. L-MVO absent at 4 mo
Ripa <i>et al</i> ^[5]	2007	58	2 d → 6 mo	Manual	L-MVO in 42%. L-MVO absent at 6 mo
Hombach <i>et al</i> ^[6]	2005	110	6 d → 9 mo	Manual	46% had L-MVO (2.8% LV mass, 16% of IS) on acute CMR. L-MVO absent at 6 mo

MVO: Microvascular obstruction; LGE method: SD: Standard deviations; IS: Infarct size; LV: Left ventricle; CMR: Cardiovascular magnetic resonance.

Table 9 Cardiovascular magnetic resonance studies illustrating the importance of late microvascular obstruction on left ventricular function and remodelling in acute myocardial infarction

Ref.	Year	n	LGE method	Main findings	Time post STEMI of predictive CMR	Follow-up
Kidambi <i>et al</i> ^[115]	2013	39	> 2SD	L-MVO only IP of impaired infarct strain. Model with IS, TIMI flow, diabetes, transmural	3 d	3 mo
Wong <i>et al</i> ^[103]	2012	40	Manual	L-MVO extent only IP for LVEF at 3 mo in model including E-MVO, IS and myocardial blood flow on perfusion	3 d	3 mo
Ezekowitz <i>et al</i> ^[92]	2010	64	Manual	L-MVO extent was IP of LVEF in model with IS and NT-proBNP	7 d	4 mo
Weir <i>et al</i> ^[112]	2010	100	Manual	L-MVO extent was only IP of LVR in model with TIMI post PCI, E-MVO, IS	4 d	6 mo
Ganame <i>et al</i> ^[25]	2009	98	Manual	L-MVO extent was IP of LVR in model with IS, troponin-I, TTR	2 d	6 mo
Nijveldt <i>et al</i> ^[111]	2008	60	Manual	L-MVO presence strongest IP of LVEF change and LVR in model with TTR, IS, LVEF, E-MVO	5 d	4 mo
Hombach <i>et al</i> ^[6]	2005	110	Manual	L-MVO extent IP for LVR in model with baseline IS, infarct transmural	6 d	225 d

MVO: Microvascular obstruction; IS: Infarct size; IP: Independent predictor; TTR: Time to revascularisation; LVR: Left ventricular remodelling; LVEF: Left ventricular ejection fraction; LVEDVI: Left-ventricular end diastolic volume index; LVESVI: Left-ventricular end systolic volume index.

is likely that this is because L-MVO reflects more severe microvascular and myocardial damage than E-MVO^[28,36]. In most studies demonstrating the independent predictive value of L-MVO on LV function and remodelling, E-MVO was not a predictor^[103,111,112]. L-MVO was a predictor independent of baseline IS^[6,20,92,111-113]. Monocyte recruitment, crucial in cellular debris removal and scar formation, is impaired in areas of L-MVO in rat myocardium and may contribute to the adverse remodelling^[114].

Prognostic importance of CMR MVO in AMI

An increasing evidence base demonstrates the strong medium-term prognostic value of L-MVO following STEMI, independent of IS and LVEF^[6,36,37,116] (Table 10). The 2 studies featuring both L-MVO and E-MVO showed that L-MVO was a stronger prognostic indicator^[36,37]. Regenfus *et al*^[117] demonstrated that L-MVO was the strongest IP of long-term combined MACE at 6 years follow-up in a model including CMR-assessed LVEF and IS (HR 3.9), providing incremental prognostic value over traditional CMR markers of myocardial damage. A meta-analysis^[118] (8 studies, $n = 1025$) demonstrated that L-MVO presence was the strongest independent predictor of medium-term combined MACE (HR 3.7) and

cardiovascular death (HR 13.2) at 2 years independent of IS and LV volumes.

The strong adverse prognostic value of L-MVO may be due to its negative effects on LV function, wall thickness and stiffness, and remodelling, and subsequent risk of heart failure and arrhythmias^[6,20,92,111-113].

IMH IN AMI

Background

IMH is a reperfusion injury occurring when restored blood flow into damaged capillaries extravasates erythrocytes into myocardium^[121,122]. CMR-derived IMH was first described in reperfused canine myocardium on *ex-vivo* T2-weighted spin-echo (T2w-TSE) imaging with excellent agreement with histology ($r = 0.96$ for IMH extent)^[123].

CMR assessment of IMH in AMI

Paramagnetic haemoglobin breakdown products shorten T2 relaxation times^[123,124]. IMH is seen as hypointense zones within hyperintense oedematous myocardium on T2w-TSE sequences. It shows good histological correlation in canine myocardium (*ex-vivo* MRI, $r = 0.96$)^[123] and in an human autopsy case series (*in-vivo* MRI, $r = 0.97$)^[124].

Table 10 Cardiovascular magnetic resonance studies illustrating the prognostic importance of late microvascular obstruction in acute myocardial infarction

Ref.	Year	n	LGE method	Main findings	Time of prognostic CMR post STEMI	Follow-up
Regenfus <i>et al</i> ^[117]	2015	249	Manual	L-MVO extent strongest IP for MACE in model including IS, LVEF, TIMI pre and post PPCI and no. diseased vessels	3.7 d	72 mo
Eitel <i>et al</i> ^[119]	2014	738	> 5SD	Largest multicentre study of L-MVO in PPCI. L-MVO > 1.4% LVM and TIMI risk score only IPs of combined MACE. Adding L-MVO to model with clinical predictors, LVEF and IS increased c-statistic	7 d	6 mo
de Waha <i>et al</i> ^[120]	2012	438	Manual	L-MVO extent IP for combined MACE in model including IS, LV volumes (only other IP was LVEF). L-MVO/IS strongest IP in model including L-MVO extent, LVEF, IS, LV volumes	3 d	19 mo
de Waha <i>et al</i> ^[36]	2010	438	Manual	Presence and extent of L-MVO were strongest IPs for MACE and mortality in models with IS, LVEF, ST-res, TIMI-flow post PCI. E-MVO was not an IP	3 d	19 mo
Cochet <i>et al</i> ^[37]	2009	184	Manual	L-MVO strongest IP for MACE, in models including GRACE score, IS, LVEF. L-MVO stronger IP than E-MVO (OR 8.7 vs 2.5)	"3-7 d"	12 mo
Bruder <i>et al</i> ^[116]	2008	143	Manual	Only extent of L-MVO > 0.5% LV mass was IP for MACE; model included IS, LVEF, age, DM, sex	4.5 d	12 mo
Hombach <i>et al</i> ^[6]	2005	110	Manual	L-MVO IP for MACE ($P = 0.04$) in model including LV end-diastolic volume and LVEF	6 d	268 d

MVO: Microvascular obstruction; LVEF: Left ventricular ejection fraction; IS: Infarct size; PPCI: Percutaneous coronary intervention; MACE: Major adverse cardiovascular events; IP: Independent predictor.

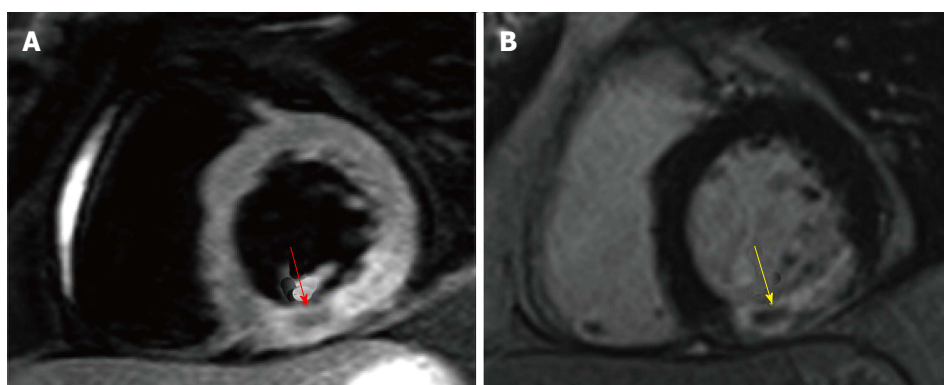


Figure 6 Intramyocardial haemorrhage on cardiovascular magnetic resonance. A: T2-weighted spin-echo image with hypointensity corresponding with IMH within the hyperintense oedematous region in the inferior wall (red arrow); B: Corresponding LGE image showing co-localisation of IMH and L-MVO (yellow arrow). IMH: Intramyocardial haemorrhage; LGE: Late gadolinium enhancement; MVO: Microvascular obstruction.

IMH occurs exclusively in areas of L-MVO (r^2 for co-localisation approximately 0.9) (Figure 6)^[25,33,125,126].

Newer sequences based on direct quantification of T2 and T2*^[74,126-129] allow IMH to be quantified without the limitations of T2w-TSE imaging. Initial studies have been promising and shown that these sequences are reproducible and appear more sensitive and accurate than T2w-TSE for IMH detection^[126,130,131]. O'Regan *et al*^[126] showed that T2* had 100% sensitivity for IMH detection compared to 90% for T2w-TSE, where the "gold standard" was co-localisation with L-MVO. In canines, T2* in haemorrhagic infarcts closely correlates with iron levels on spectrometry, and T2*-detected IMH co-localises with iron deposition on Perl's staining^[132] and extravasated erythrocytes on Haematoxylin-Eosin staining^[128]. In pigs, regions of IMH on T2* imaging showed vessel degeneration and iron deposition^[8].

There is a paucity of data on temporal changes in CMR-detected IMH. Mather *et al*^[18] showed that IMH on

T2w-TSE was present in 33% of patients, with maximal extent at 48 h post PPCI and progressively resolution by 3 mo. Carrick *et al*^[74] recently demonstrated that the incidence and extent of IMH on T2* increased between 8 h and 3 d post PPCI. Its extent was significantly lower at 10 d and was seen in only 13% of patients at 7 mo. The authors also found that MVO was present in all patients with IMH, and its extent peaked earlier at 8 h suggesting that IMH is an ensuing reperfusion injury in regions of MVO.

CMR IMH as a predictor of LV function and remodelling in AMI

There is a small evidence base demonstrating that IMH is a strong univariate predictor of medium-term impaired LV function and remodelling, however multivariate analysis reveals mixed results, with some studies suggesting no incremental predictive value of IMH over MVO and IS (Table 11).

Table 11 Cardiovascular magnetic resonance studies illustrating the importance of intramyocardial haemorrhage on left ventricular function and remodelling in acute myocardial infarction

Ref.	Year	n	IMH CMR method	Main findings	CMR time post MI	Mean/median F/U CMR
Carrick <i>et al</i> ^[74]	2016	245	T2*	IMH strongest IP for LVR. IMH associated with lower LVEF and greater volumes	3 d	7 mo
Kidambi <i>et al</i> ^[115]	2013	39	T2w-TSE and T2*	IMH associated with attenuation of follow-up infarct strain	3 d	3 mo
Husser <i>et al</i> ^[33]	2012	304	T2w-TSE	IMH strongest IP for LVR in model with LVEF, IS, LV vol, L-MVO	6 d	189 d
Mather <i>et al</i> ^[131]	2011	48	T2w-TSE and T2*	IMH strongest IP of LVR in model with IS, LVEF, LVESV, E-MVO, MSI	2 d	3 mo
Beek <i>et al</i> ^[24]	2010	45	T2w-TSE	IMH was a univariate predictor of LVEF. However no prognostic significance beyond baseline LVEF and MVO in predicting final LVEF	5 d	4 mo
Bekkers <i>et al</i> ^[121]	2010	90	T2w-TSE	Acute MSI and LVEF increase at follow-up lowest if IMH present. But IMH no prognostic significance beyond MVO in predicting LVEF	5 d	103 d
O'Regan <i>et al</i> ^[126]	2010	50	T2*	IMH presence univariate predictor of LVEF and LV volumes. However only IS independently predicted LVEF	3 d	N/A
Ganame <i>et al</i> ^[25]	2009	98	T2w-TSE	IMH extent strongest IP of LVR in model with IS, E-MVO, Troponin-I, AAR, TTR, IS	2 d	4 mo

IS: Infarct size; IP: Independent predictor; LVR: Left ventricular remodelling; MVO: Microvascular obstruction; LVEF: Left ventricular ejection fraction; LVESVI: Left ventricular end systolic volume index; T2w-TSE: T2-weighted turbo spin-echo; AAR: Area at risk; MSI: Myocardial salvage index; N/A: Not applicable.

Prognostic importance of CMR IMH in AMI

Multivariate analyses including IMH as a prognostic indicator also show mixed results. Amabile *et al*^[133] demonstrated that IMH on T2w-TSE at 4 d post STEMI was the strongest independent predictor of MACE at 1-year (HR 2.8) in a model including LVEF, ST-resolution and L-MVO. Husser *et al*^[33] showed that only LVEF and IMH extent on T2w-TSE independently predicted MACE at 140 wk follow-up in a model containing LV volumes, AAR, IS and L-MVO. However IMH and MVO extent showed strong correlation ($r = 0.95$) and adding T2w imaging to a model containing LGE and cine imaging did not improve the predictive power for MACE, supporting a strong concordance of IMH and MVO. Eitel *et al*^[125] demonstrated that IMH presence on T2w-TSE and LVEF < 53% were the only CMR independent predictors of MACE at 6 mo in a model with lone MVO. Carrick *et al*^[74] recently demonstrated that IMH on T2* mapping was the strongest independent predictor of cardiac death and heart failure hospitalisation at 830 d follow-up. In their multivariate model, L-MVO was not a predictor suggesting that IMH reflects extreme microvascular injury.

ISCHAEMIC AAR AND MYOCARDIAL SALVAGE IN AMI

Background

Oedema is seen in acute cardiac inflammation. In STEMI, it signifies reversible myocardial injury in the ischaemic cascade. The area of oedematous myocardium defines the ischaemic AAR supplied by the occluded IRA^[61,134].

CMR assessment of AAR and MSI in AMI

The T2 (transverse) relaxation time is increased by

regional water content^[135]. T2w-TSE sequences illustrate oedema as hyperintensity^[134] and are currently the mainstay of CMR oedema imaging. Most commonly used is the black-blood T2-weighted short-tau inversion-recovery sequence (T2w-STIR). This uses two initial inversion pulses to null moving blood. This is followed by a third inversion pulse, which nulls tissues with short T1 times (fat) to provide high contrast between blood (nulled) and myocardium^[134,136]. T2w imaging of myocardial oedema is well-validated in animal studies assessing myocardial water volume on histological assessment^[137] and fluorescent microspheres^[77]. T2w oedema assessment is well-validated with SPECT^[138-140] and angiographic markers of AAR (BARI^[141], APPROACHp^[142] scoring). AAR on T2w can be assessed accurately for upto 1-wk post-PPCI unlike SPECT, which requires radionuclide administration during coronary occlusion and has higher spatial resolution and thus ability to detect subendocardial injury^[138].

However T2w-TSE imaging has inherent disadvantages that can compromise image quality and oedema detection. Upto 30% of datasets are non-analysable in studies^[24,143,144]. New T2w sequences have been studied, with encouraging results (Figure 7).

The aim of prompt reperfusion is to limit IS by minimizing the conversion of reversibly injured myocardial cells (AAR) into necrotic, infarcted tissue (IS)^[95,156]. Anterior STEMI typically results in larger IS due to the larger coronary bed supplied by the left anterior descending artery^[14,80,82]. Hence a more accurate assessment of revascularisation strategies can be provided by adjusting IS for the AAR. The resulting myocardial salvage index (MSI) defines the proportion of reversibly injured tissue (AAR) that does not progress to infarction (IS, Equation 1, Figure 8). MSI is expressed as percentage of the initial AAR [0% is no salvage, 100% is complete salvage (aborted

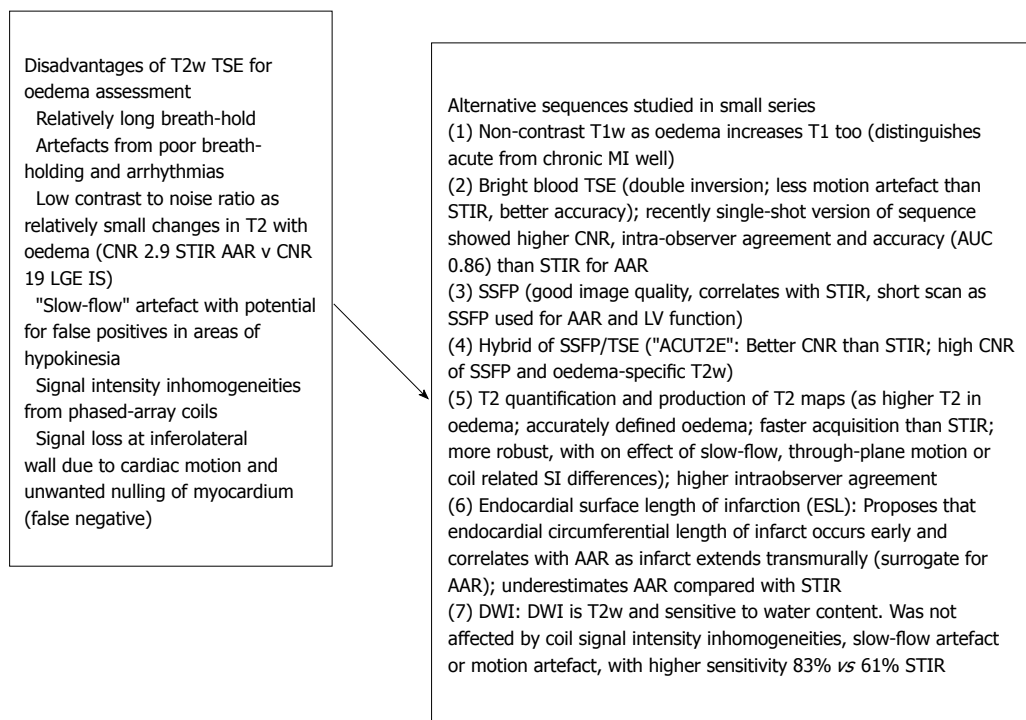


Figure 7 Alternative sequences to dark-blood T2-weighted turbo spin-echo for visualising oedema. Left: Inherent disadvantages of T2w-TSE^[134,144-147]; Right: Sequences compared with T2w-TSE: (1)^[145], (2)^[141,142,144,148], (3)^[149,150], (4)^[144], (5)^[151,152], (6)^[153,154], (7)^[155]. T2w-TSE: T2-weighted turbo spin-echo; DWI: Diffusion-weighted imaging; AAR: Area at risk.

STEMI)]^[157].

Equation 1: Myocardial salvage index (MSI, %) = $100 \times [(AAR-IS)/(AAR)]$.

Desch showed excellent intraobserver and inter-observer agreement for MSI assessment using T2w-STIR and LGE (coefficients of variation approximately 5.0%) and excellent test-retest reproducibility in a study of 20 acute STEMI patients^[158].

Other determinants of AAR include TTR^[91,130,159-162], extent of collateralised IRA territory flow^[5,80,159,163], TIMI-flow pre PPCI, LAD IRA and diabetes^[91].

Studies of the chronology of oedema suggest that it occurs very early in the ischaemic cascade. Abdel-Aty confirmed the presence of transmural oedema in canines on *in-vivo* T2w imaging at 28 min post LAD occlusion at which point LGE and troponin release were absent, indicating reversible injury^[164]. Fernández-Jiménez *et al.*^[165] however recently demonstrated a bimodal pattern of AAR extent in pigs with T2-mapping CMR and histological water quantification. They showed peak values at 2 h thought to be a direct result of reperfusion, followed by a return to baseline at 2 d and then progressive increase towards peak values at 7 d, with the latter peak felt due to water replacement of cleared cellular debris. Studies of temporal changes in AAR and MSI in humans are summarised in Table 12. Correct timing of oedema imaging is crucial in accurate calculation of AAR and MSI.

The near-resolution of oedema by 6 mo^[5,18,21,91,138] allows distinction between acute and chronic infarcts when combined with LGE imaging.

CMR MSI as a predictor of LV function and remodelling in AMI

Myocardial salvage is a strong univariate predictor of medium-term LV function^[14,166,167] and adverse LV remodelling post STEMI^[14,27,91,161]. Multivariate analysis demonstrates mixed results. MSI independently predicted LV remodelling in the work of Mather^[131] (Table 13). However MSI was not a predictor once IS was added into multivariate models in studies by Monmeneu^[91] and Masci^[14]. This, in conjunction with the correlation between MSI and IS, and AAR and IS^[26] questions whether MSI and IS are truly independent of each other in predicting LV remodelling and prognosis post STEMI. It could be argued that since MSI adjusts IS for the extent of AAR, it may have less inherent variability than IS. Since up to 30% of AAR datasets have been deemed non-diagnostic in previous studies^[24,143,144], this may impact on the robustness of MSI quantification whereas IS datasets are exceptionally rarely excluded based on image quality. It is not clear currently whether IS or MSI is the better measure of revascularisation success post PPCI.

Prognostic importance of CMR MSI in AMI

Historically, the prognostic value of MSI was demonstrated using SPECT. Ndrepa first showed that MSI was the strongest independent predictor of 6-mo mortality^[168]. MSI was an independent prognostic indicator in the medium term post STEMI in two studies. Although both studies were from the same patient cohort, they have both been

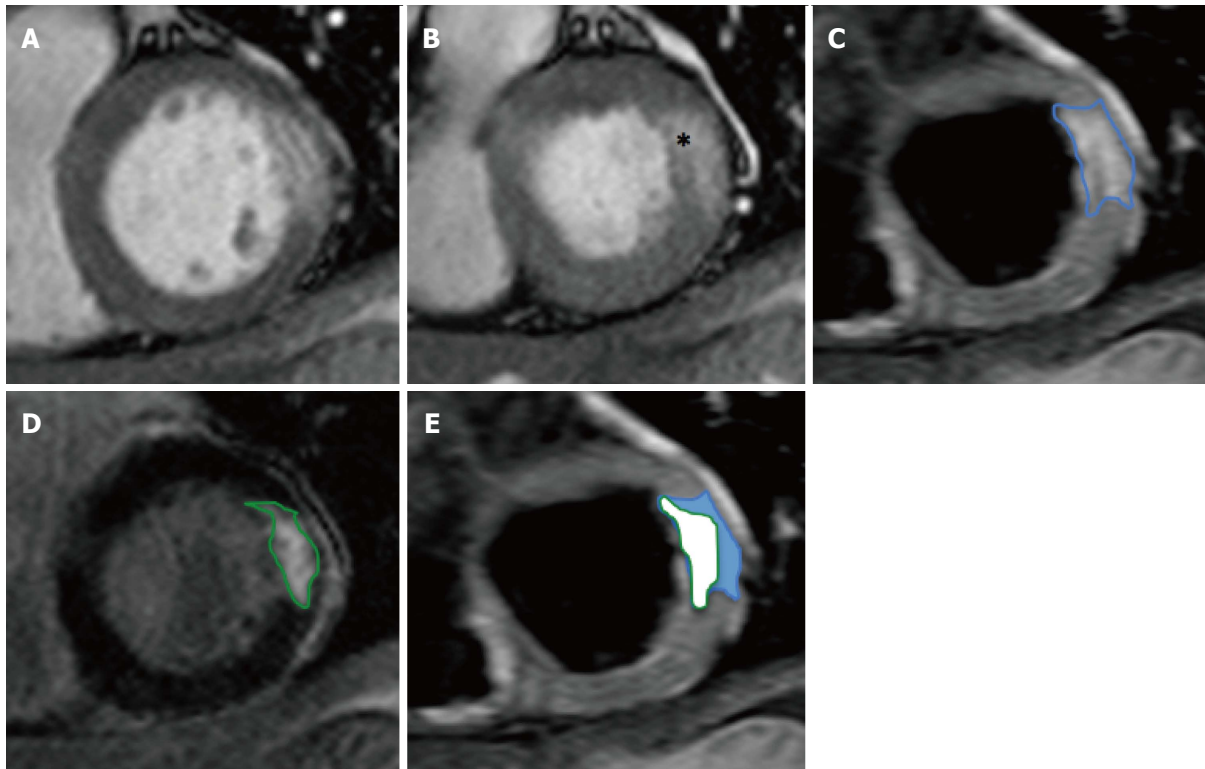


Figure 8 Calculation of salvaged myocardium. A: SSFP end-diastolic cine image; B: SSFP end-systolic cine image showing hypokinetic basal anterolateral segment (*); C: T2w-STIR image showing oedema (AAR) in anterolateral wall consistent with circumflex artery occlusion; D: Corresponding LGE image with near-transmural infarction; E: Calculation of salvaged myocardium in blue. SSFP: Steady-state free precession; T2w-STIR: T2-weighted short-tau inversion-recovery sequence; LGE: Late gadolinium enhancement.

Table 12 Temporal changes in cardiovascular magnetic resonance-derived area at risk and myocardial salvage index in acute myocardial infarction

Ref.	Year	n	CMR timepoints post STEMI	AAR, IS method	Main findings
Mather <i>et al</i> ^[18]	2011	48	2 d → 1 wk → 30 d → 3 mo	> 2SD STIR, > 2SD LGE	AAR reduction at successive timepoints, 1-3 mo (-75%). No change MSI at d2 or 1 wk as IS and AAR decreased proportionally
Dall'Armellina <i>et al</i> ^[21]	2011	30	2 d → 1 wk → 2 wk → 6 mo	> 2SD T2p-BB, > 2SD LGE	100% had oedema at d2. AAR stable over 1st week (37% vs 39% LVM). Decreased by 2 wk and nearly resolved at 6 mo
Carlsson <i>et al</i> ^[38]	2009	16	1 d → 1 wk → 6 wk → 6 mo	Manual STIR, and LGE	AAR at all timepoints. AAR stable in 1st week, correlated with 1 wk SPECT. Decrease by 1 mo (10% LVM), nearly gone by 6 mo
Ripa <i>et al</i> ^[5]	2007	58	2 d → 1 mo → 6 mo	Manual STIR and LGE	All had oedema at d2. AAR decreased at all time points. No data on MSI in this study

AAR: Area at risk; MSI: Myocardial salvage index; AAR, LGE method: SD: Standard deviations; STIR: T2-weighted short-tau inversion recovery imaging; T2p-SS-BB: T2-prepared single-shot bright-blood; 3T: 3.0 tesla field strength; IS: Infarct size.

included in Table 14 due to their differing primary findings.

T1, T2 AND T2* QUANTIFICATION AND MAPPING IN AMI

The current mainstay of LGE and T2w techniques for the detection of infarct and oedema rely on semi-quantitative threshold-based quantification methods using arbitrary SI cut-offs compared to user-defined regions of interest, automated algorithms or are based on manual planimetry. There is currently no consensus on the optimal quantification method for IS or AAR using these

sequences. This can lead to subjectivity and dependence upon optimal nulling of normal myocardium and thus potential for error. In addition, commonly used T2w-TSE sequences suffer from non-diagnostic image quality in upto 30% of patients^[24,143,144].

T1, T2 and T2* quantification present an exciting and complementary approach to LGE and T2w imaging. Developed by Messroghli *et al*^[169] in 2003, their use in MI research has accelerated over the last 5 years. They allow not only the location and extent of infarction, oedema, MVO and IMH to be determined from subsequent parametric myocardial maps, but also the severity of these pathologies to be assessed through the magnitude

Table 13 Cardiovascular magnetic resonance studies showing the importance of myocardial salvage index on left ventricular function and remodelling in acute myocardial infarction

Ref.	Year	<i>n</i>	AAR, IS method	Main findings	CMR timepoint post STEMI	Follow-up
Mather <i>et al</i> ^[131]	2011	48	> 2SD STIR, > 2SD LGE	MSI was IP for LVR (OR 0.95) in model including LV volumes, LVEF, IS, IMH, MVO	2 d	3 mo
Monmeneu <i>et al</i> ^[91]	2012	118	> 2SD STIR, > 2SD LGE	MSI univariate predictor of LVR and final LVEF. However not IP of LVR in model with LVESVI, IS, no. transmural segs	6 d	6 mo
Masci <i>et al</i> ^[14]	2011	260	> 2SD STIR, > 5SD LGE	MSI strong univariate predictor of LVR and final LVEF. However not IP in model including IS, MVO	1 wk	4 mo
Masci <i>et al</i> ^[26]	2010	137	> 2SD STIR, > 5SD LGE	MSI strongest IP for LVR However IS and MSI ($r = -0.72$) and IS and AAR ($r = 0.85$) correlated	1 wk	4 mo

IS: Infarct size; IP: Independent predictor; LVR: Left ventricular remodelling; MVO: Microvascular obstruction; LVEF: Left ventricular ejection fraction; LVESVI: Left-ventricular end systolic volume index; STIR: T2-weighted short-tau inversion-recovery; LGE: Late gadolinium enhancement.

Table 14 Cardiovascular magnetic resonance studies illustrating the prognostic importance of myocardial salvage index in acute myocardial infarction

Ref.	Year	<i>n</i>	AAR, IS method	Main findings	CMR timepoint post STEMI	Follow-up
Eitel <i>et al</i> ^[34]	2011	208	> 2SD -STIR, > 5SD LGE	MSI was only CMR-based IP of mortality in model with age, IS, MVO, LVEF, TIMI- post PPCI, diabetes, age (IS not IP). MSI not IP of MACE (only IS, LVEF, age were)	3 d	19 mo
Eitel <i>et al</i> ^[161]	2010	208	> 2SD STIR, > 5SD LGE	MSI was only IP for MACE and mortality in model including LVEF, MVO, IS, ST-resolution and TIMI-grade post PCI	3 d	6 mo

IS: Infarct size; PCI: Percutaneous coronary intervention; MACE: Major adverse cardiovascular events; IP: Independent predictor; MVO: Microvascular obstruction; LVEF: Left ventricular ejection fraction.

of values obtained^[170,171]. These methods are not reliant on reference regions of interest and do not suffer from T2w-TSE artefacts.

T1 mapping (longitudinal relaxation)

T1 relaxation curves allow calculation of the T1 time (time taken for recovery of 63% of longitudinal magnetization). The currently used curve-fitting sequences used include MOLLI (Modified Look-Locker Inversion Recovery), ShMOLLI (Shortened MOLLI), SASHA (SATuration recovery single-SHot Acquisition) and SAPHIRE (SATuration Pulse Prepared Heart rate independent Inversion REcovery)^[172]. Infarcted and oedematous myocardium demonstrate prolonged pre-contrast T1 values and reduced post-contrast T1 values compared with normal myocardium, allowing infarct visualisation and quantification^[169,170,173,174]. Messroghli showed that this technique had high test-retest reproducibility^[175], was stable within the range of heart rates commonly seen in clinical practice and showed comparable sensitivity for IS quantification compared with LGE^[169,173,176]. T1 values within the infarct core were recently shown to demonstrate a strong inverse correlation with L-MVO extent, incidence of LV remodelling and all-cause mortality at 2.5 years^[177].

T2 mapping (transverse relaxation)

T2w images are generated using a T2-SSFP sequence with log-transformed curve-fitting T2 quantification, with

different T2 preparation (TE) times. T2 mapping has shown excellent reproducibility and no effect of slow-flow, through-plane movement, SI loss, or effects of coil SI inhomogeneities^[151,178]. T2 mapping accurately assessed oedema in 96% of patients (good image quality in 100%), whereas T2w-STIR detected oedema in only 67% of patients (15% non-diagnostic 15%)^[151]. High observer agreement and close agreement between T1 ($r^2 = 0.94$) and T2 maps ($r^2 = 0.96$), and fluorescent microspheres for AAR detection was seen in canine myocardium^[179].

T2* mapping (transverse relaxation in presence of field inhomogeneities)

T2* mapping allows visualisation and quantification of IMH due to the presence of paramagnetic haemoglobin breakdown products. A cut-off value of < 20 ms has been used to define the presence of IMH^[180]. Although the evidence base for T2* mapping in assessing IMH is currently limited, O'Regan demonstrated that it has greater sensitivity than T2w-STIR imaging (100% vs 90%) for IMH. Kali showed good correlation between *in-vivo* T2* and histological assessment of IMH and iron levels in canine myocardium^[127,128]. T2* mapping may improve the specificity of IMH detected on CMR^[131].

T1, T2 and T2* surrogate markers hold promise for improving the accuracy of detection of infarct, oedema and IMH respectively, and further improving statistical power of STEMI studies. However, due to the importance

Table 15 Cardiovascular magnetic resonance studies illustrating the prognostic importance of right ventricular infarction in acute myocardial infarction

Ref.	Year	<i>n</i>	RV LGE analysis method	Main findings	CMR timepoint post STEMI	Follow-up
Jensen <i>et al</i> ^[184]	2010	50	Manual	RVI only IP of MACE in model with age, sex, LVEF, LV IS	3 d	32 mo
Miszalski-Jamka <i>et al</i> ^[198]	2010	99	Manual	RVEF (HR 1.46) and RVI extent (HR 1.50) IP for MACE	"3-5 d"	1150 d
Grothoff <i>et al</i> ^[187]	2012	450	Manual	RVI was IP of MACE (HR 6.70)	"1-4 d"	20 mo

MACE: Major adverse cardiovascular events; IP: Independent predictor; HR: Hazard ratio; RV: Right ventricle; LVEF: Left ventricular ejection fraction; LGE: Late gadolinium enhancement; IS: Infarct size; RVI: Right ventricular infarction.

Table 16 Key studies illustrating the independent predictive value of cardiovascular magnetic resonance markers for left ventricular remodelling

CMR marker	Ref.	Year	<i>n</i>	CMR quantification	Main findings	Acute CMR time	Follow-up CMR time
IS	Husser <i>et al</i> ^[133]	2012	304	2SD	IS extent IP for LVR in model with LVEF, IS, LV volumes, MVO	6 d	189 d
IS	Monmeneu <i>et al</i> ^[91]	2012	118	2SD	Number of segments > 50% transmural IP for LVR	6 d	6 mo
IS	Wu <i>et al</i> ^[94]	2008	122	Manual	IS extent at 2 d only IP for LVEF and LVR	2 d	4 mo
IS	Hombach <i>et al</i> ^[6]	2005	110	Manual	IS extent at 6 d was an IP for LVR in model with MVO, % transmural	6 d	225 d
L-MVO	Weir <i>et al</i> ^[112]	2010	100	Manual	L-MVO extent was only IP of LVR in model with TIMI post PCI, E-MVO, IS	4 d	6 mo
L-MVO	Hombach <i>et al</i> ^[6]	2005	110	Manual	L-MVO extent IP of LVR in model with baseline IS, infarct transmural	6 d	225 d
IMH	Carrick <i>et al</i> ^[74]	2016	245	T2*	IMH strongest IP of LVR in model with patient/angio characteristics, LVEDVI	3 d	7 mo
IMH	Husser <i>et al</i> ^[133]	2012	304	T2w-TSE	IMH strongest IP for LVR in model with LVEF, IS, LV volumes, L-MVO	6 d	189 d
MSI	Monmeneu <i>et al</i> ^[91]	2012	118	2SD LGR/STIR	MSI univariate but not IP of LVR in model with IS, LVESVI, segments > 50%	6 d	6 mo
MSI	Masci <i>et al</i> ^[14]	2011	260	2SD STIR, 5SD LGE	MSI univariate predictor of LVR and final LVEF. However not IP of either	1 wk	4 mo
MSI	Masci <i>et al</i> ^[26]	2010	137	> SD STIR, 5SD LGE	MSI strongest IP for LVR. However IS and MSI and IS and AAR correlated	1 wk	4 mo
T1	Carrick <i>et al</i> ^[177]	2016	300	T1 map, 2SD STIR, 5SD LGE	Infarct core native T1 inverse relationship with LVR (OR 0.91 per -10 ms T1)	2 d	6 mo

Criteria: Individual studies with $n \geq 100$ and follow-up CMR ≥ 3 mo post-PPCI. IS: Infarct size; L-MVO: Late microvascular obstruction; IMH: Intramyocardial haemorrhage; MSI: Myocardial salvage index; SD: Standard deviations; STIR: T2-weighted short-tau inversion recovery; LGE: Late gadolinium enhancement; IP: Independent predictor; LV: Left ventricular; LVEF: Left ventricular ejection fraction; AAR: Area at risk; LVEDVI: Left ventricular end-diastolic volume; CMR: Cardiovascular magnetic resonance.

of protocol standardisation, these techniques are rarely used in multicentre studies at present.

RIGHT VENTRICULAR INVOLVEMENT IN AMI

CMR assessment of right ventricular infarction in AMI

CMR is the gold standard imaging modality for the assessment of right ventricular (RV) volumes, function, oedema^[181] and infarction (RVI)^[182]. CMR identifies RVI with greater sensitivity than echocardiography, ECG (V4_R ST-segment elevation) and clinical examination^[183,184] and demonstrates RV L-MVO^[185,186]. There is good interobserver and intraobserver agreement for the identification of RV oedema ($\kappa = 0.62$, $\kappa = 0.62$, respectively) and very good agreement for RVI ($\kappa = 0.70$, $\kappa = 0.70$, respectively)^[181].

The high MSI in RVI often > 90%^[187,188] is thought to be due the relatively low RV nutrient needs, direct endocardial oxygen diffusion and good collateral blood supply^[188,189].

Prognostic importance of CMR-derived right ventricular infarction in AMI

RVI confers adverse short-term prognosis, with a large meta-analysis ($n = 7136$) demonstrating that RVI on ECG, echocardiography or radionuclide imaging predicted 30-d mortality and in-hospital MACE^[190]. Shah demonstrated the prognostic importance of right ventricular infarction on imaging, where RVEF < 38% on radionuclide ventriculography post STEMI was a strong independent predictor of 1-year mortality^[191]. Right ventricular infarction is a strong independent predictor of medium to long-term prognosis in a small number of CMR

Table 17 Key studies illustrating the independent predictive value of cardiovascular magnetic resonance markers for prognosis

CMR marker	Ref.	Year	<i>n</i>	CMR quantification	Main findings	Acute CMR time	Follow-up
IS	Husser <i>et al</i> ^[96]	2013	250	> 2SD	Extent of transmural infarction was only IP for MACE	7 d	163 wk
IS	Izquierdo <i>et al</i> ^[97]	2013	440	> 2SD	IS was IP for arrhythmic cardiac events in model including LVEF, hypertension	7 d	123 wk
IS	Eitel <i>et al</i> ^[34]	2011	208	> 5SD	IS was IP of MACE in model with MVO, LVEF, MSI, Killip, TIMI flow post-PPCI	3 d	18.5 mo
IS	Larose <i>et al</i> ^[67]	2010	103	FWHM	IS strongest IP for MACE in model with LVEF, CK. LGE > 23% for MACE	4.5 h	2 yr
IS	Bodi <i>et al</i> ^[38]	2009	214	> 2SD	Extent of transmural infarction (no. of segments > 50% transmural) IP for MACE	7 d	553 d
IS	Wu <i>et al</i> ^[99]	2008	122	Manual	IS only IP of 2 yr MACE in model containing LVEF, LVESVI (HR 1.06)	2 d	538 d
L-MVO	Regenfus <i>et al</i> ^[117]	2015	249	Manual	MVO extent strongest IP for MACE in model with IS, LVEF, TIMI and no. vessels	3.7 d	72 mo
L-MVO	Eitel <i>et al</i> ^[119]	2014	738	> 5SD	L-MVO > 1.4% LVM IP of MACE in model with LVEDVI, LVEF, clinical markers	7 d	6 mo
L-MVO	de Waha <i>et al</i> ^[120]	2012	438	Manual	L-MVO extent IP for MACE in model with IS, LV volumes. L-MVO/IS strongest IP	3 d	19 mo
L-MVO	de Waha <i>et al</i> ^[36]	2010	438	Manual	L-MVO strongest IP of MACE/mortality in model with IS, LVEF, STR, TIMI post	3 d	19 mo
L-MVO	Cochet <i>et al</i> ^[37]	2009	184	Manual	L-MVO strongest IP for MACE in model with GRACE, IS, LVEF. E-MVO weaker IP	"3-7 d"	12 mo
L-MVO	Bruder <i>et al</i> ^[116]	2008	143	Manual	L-MVO extent > 0.5% LV mass IP for MACE in model with IS, LVEF, age, DM, sex	4.5 d	12 mo
L-MVO	Hombach <i>et al</i> ^[6]	2005	110	Manual	L-MVO IP for MACE (<i>P</i> = 0.04) in model with LV end-diastolic volume and LVEF	6 d	268 d
IMH	Carrick <i>et al</i> ^[74]	2016	245	T2*	IMH strongest IP of CV death and HF. Multivariate model, L-MVO not predictor	3 d	830 d
IMH	Amabile <i>et al</i> ^[133]	2012	114	T2w-TSE	IMH presence was strongest predictor of MACE in model with MVO, LVEF, STR	4 d	12 mo
IMH	Husser <i>et al</i> ^[33]	2012	304	T2w-TSE	IMH IP for MACE in model with AAR, IS, L-MVO. T2w. No inc. value with LGE	6 d	140 wk
IMH	Eitel <i>et al</i> ^[125]	2011	346	T2w-TSE	IMH IP of MACE in model with L-MVO. T2w inc. value with LGE and cine	3 d	6 mo
MSI	Eitel <i>et al</i> ^[34]	2011	208	> 2SD/> 5SD	MSI only CMR IP of mortality in model with age, IS, MVO, LVEF, TIMI post, IS	3 d	19 mo
MSI	Eitel <i>et al</i> ^[161]	2010	208	> 2SD/> 5SD	MSI only IP for MACE/mortality in model with LVEF, MVO, IS, STR, TIMI post	3 d	6 mo
T1	Carrick <i>et al</i> ^[177]	2016	300	T1 map, > 2SD STIR, > 5SD	Infarct core T1 inverse association with risk of mortality and heart failure hospitalisation, in model with LVEF, infarct T2, IMH. Similar prognostic as L-MVO	2 d	2.5 yr

Criteria: Individual studies with *n* ≥ 100 and follow-up CMR ≥ 6 mo follow-up. IS: Infarct size; L-MVO: Late microvascular obstruction; IMH: Intramyocardial haemorrhage; MSI: Myocardial salvage index; SD: Standard deviations; STIR: T2-weighted short-tau inversion recovery; LGE: Late gadolinium enhancement; IP: Independent predictor; LV: Left ventricular; LVEF: Left ventricular ejection fraction; AAR: Area at risk; LVEDVI: Left ventricular end-diastolic volume; CK: Creatine kinase; T2w-TSE: T2-weighted turbo spin echo; MACE: Major adverse cardiovascular events; CV: Cardiovascular.

studies (Table 15).

WHEN IS THE OPTIMAL TIME TO PERFORM CMR ASSESSMENT IN MI?

In acute STEMI, IS, AAR and MSI are best imaged at 7 d post PPCI due to overestimation of necrosis on LGE, and IS at 7 d best predicts final IS, LV remodelling and function and prognosis^[5-7,9,18,20,21]. Human studies suggest that AAR is stable during the first week^[21,138]. Although Fernández-Jiménez *et al*^[165] demonstrated a bimodal AAR peak in pigs, their drop in AAR extent on T2w CMR at 2 d post-reperfusion may be due to a high incidence of IMH in pigs and peak IMH extent at 2 d^[74].

Indeed the drop in AAR extent on the gold standard of histological water analysis in their study at 2 d was much less pronounced, and at 7 d AAR extent had returned to stable peak levels. In addition, studies demonstrating close agreement between T2w-derived AAR and the reference non-invasive modality of SPECT^[138,139] were undertaken at 7 d post STEMI. MVO and IMH extent peak at 48 h then decrease^[18] but are present at 7 d^[9,18]. Although undertaking CMR at 7-d may potentially underestimate MVO and IMH extent^[9,18,74], this may be minimised by expressing MVO and IMH extent as a proportion of IS rather than LV mass, to correct for the corresponding reduction in IS. Thus, acutely post STEMI for the assessment of IS, MSI, MVO and IMH, imaging at 7 d may provide the best compromise in relation to their

temporal changes^[5-7,9,18,20,21] for accurate quantification and prediction of LV function, remodelling and prognosis. This needs to be balanced with contemporary clinical practice where patients are typically discharged at 3-4 d post-PPCI, and the risk of early attrition. Using final IS at follow-up as a primary outcome risks underestimating potential differences in treatment strategies due to greater infarct resorption with the larger infarcts.

Data on the chronology of IS suggests that infarct resorption is essentially complete by 3 mo post MI^[9,18,20,74]. However a key objective of follow-up CMR is to assess LV geometry and remodelling and hence must allow the relatively slower adaptations of ventricular volumes (approximately 12 mo), compared with changes in IS and LVEF to complete. LVEF shows no significant change after 1-mo post STEMI. Follow-up CMR at 3 and 6-mo may fail to provide an accurate assessment of LV volumes and remodelling. The evidence base suggests that in order to allow completion of the trio of IS, LVEF and LV volumetric changes, follow-up CMR should be performed at 12-mo post STEMI^[5,7,18,20,21]. When correlating CMR and clinical outcomes, the longer timepoint of 12-mo also permits more reliable clinical follow-up.

Standardisation of LGE, AAR and IMH sequences and quantification methods is equally important in light of newer T1, T2 and T2*-mapping sequences and inherent image quality issues associated with T2w-TSE.

CONCLUSION

Contrast-enhanced CMR offers robust, validated and reproducible surrogate markers, providing an accurate representation of pathophysiology, assessment of myocardial function and injury, and predictive value for medium to long-term LV function, remodelling and prognosis following PPCI for STEMI. Tables 16 and 17 summarise the key prospective studies illustrating the independent predictive value of CMR markers for LV remodelling (studies where $n > 100$, follow-up CMR ≥ 3 mo post PPCI) and prognosis (studies where $n > 100$, ≥ 6 mo follow-up) respectively.

In the acute phase, CMR can be performed accurately for up to 7 d post PPCI. CMR delivers no radiation to the patient and this makes it ideal for serial studies. The multimodal nature of CMR allows a multiparametric study of cardiac function, structure and volumes within a single study, which can be undertaken within approximately 45 min in the majority of patients. It is likely that CMR will become the mainstay of cardiac imaging, providing an important role in risk stratification and treatment post STEMI. Focus needs to be continued in translating findings on the prognostic importance of surrogate markers to development of therapeutic targets post STEMI.

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