

ACUTE CORONARY SYNDROMES

MRI in the assessment of ischaemic heart disease

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INTRODUCTION

Ischaemic heart disease (IHD) is the leading cause of death worldwide. Over the last two decades, cardiac MRI (CMR) has emerged as a promising non-invasive modality in the assessment of patients with suspected and established IHD due to its good spatial resolution, high reproducibility and myocardial tissue characterisation capabilities, thereby aiding in the diagnosis, guiding clinical decision-making and improving risk stratification.

This article provides an overview of why, when and where CMR may fit into the routine clinical practice.

A) CMR IMAGING TECHNIQUES

The cornerstone of CMR is its multiparametric nature, that is, its ability to assess multiple aspects of myocardial structure and function in a single examination with the aid of various imaging techniques. The combination of techniques used is tailored to the clinical question.

Cine imaging

CMR is the current non-invasive gold standard method to measure left and right ventricular (LV and RV) volumes and ejection fraction.^{1 2} For CMR volumetric assessment, the ventricles are sliced from base to apex and the endocardium and epicardium subsequently contoured (figure 1). Therefore, it is truly three-dimensional (3D) without relying on geometrical assumptions, unlike 2D echocardiography. However, the third axis information is limited compared with 3D echocardiography or 3D multislice CT. Both the CMR long-axis and short-axis views are similar to echocardiography, as well as the myocardial segmental nomenclature (except the 17th segment apical cap, usually omitted in echocardiography).³

Steady State Free Precession (SSFP) is the sequence of choice for cine imaging due to its clear definition of endocardial and epicardial borders. Regional LV/RV function can be analysed visually by documenting the presence and extent of segmental wall motion abnormality (hypokinesia/akinesia/dyskinesia) as in echocardiography,⁴ or quantitatively by measuring wall thickening and myocardial strain.⁵ Cine CMR can also be used during low-dose and high-dose dobutamine to assess myocardial viability and inducible ischaemia, respectively.^{6–8}

T2-weighted imaging

CMR can detect myocardial oedema, a hallmark of acute inflammation using T2-weighted imaging.

Learning objectives

- ▶ To understand the principles of using cardiac MRI (CMR) in patients with ischaemic heart disease (IHD).
- ▶ To understand the various CMR techniques used in the assessment of IHD.
- ▶ To review the current evidence and indications for the use of CMR in acute and chronic IHD.

Myocardial oedema is a reversible phenomenon, progressively resolving over time (<3 months).

In the setting of acute IHD, myocardial oedema by CMR has been validated as the myocardial area at risk (AAR) versus the gold-standard fluorescent microspheres.⁹

The presence and extent of myocardial salvage can be derived with CMR by subtracting the infarcted area measured by late gadolinium enhancement (LGE) from the AAR. Both AAR and myocardial salvage can be assessed retrospectively shortly after the acute event.¹⁰

T2-weighted short- τ inversion recovery (T2-STIR) is the most commonly used oedema imaging sequence. The different myocardial oedema sequences available are not interchangeable, and T2 mapping being most reproducible.¹¹

The assessment of the oedema images is usually done visually, but semi-automated image signal intensity methods are available (useful as surrogate end-points for clinical trials).¹²

First-pass myocardial perfusion imaging

This method tracks the perfusion of the myocardium in real-time.¹³ It is mainly used in conjunction with a stressor to evaluate the presence of myocardial inducible perfusion defects (a surrogate for inducible myocardial ischaemia), which appear in the images as hypointense areas (delayed contrast perfusion due to the upstream significant epicardial coronary stenosis). Images are acquired at peak stress and at rest for comparison.

The stressor agents commonly used are either vasodilators such as adenosine, dipyridamole (a pro-drug of adenosine) and, more recently, regadenoson or inotropic (dobutamine). Regadenoson is a more selective agent, with potential fewer side effects. Adenosine is the most commonly used, mainly due to patient comfort (secondary to its ultra short half-life). Vasodilator perfusion is preferable in the setting of resting regional wall motion abnormality (RWMA) and in LV hypertrophy,¹⁴



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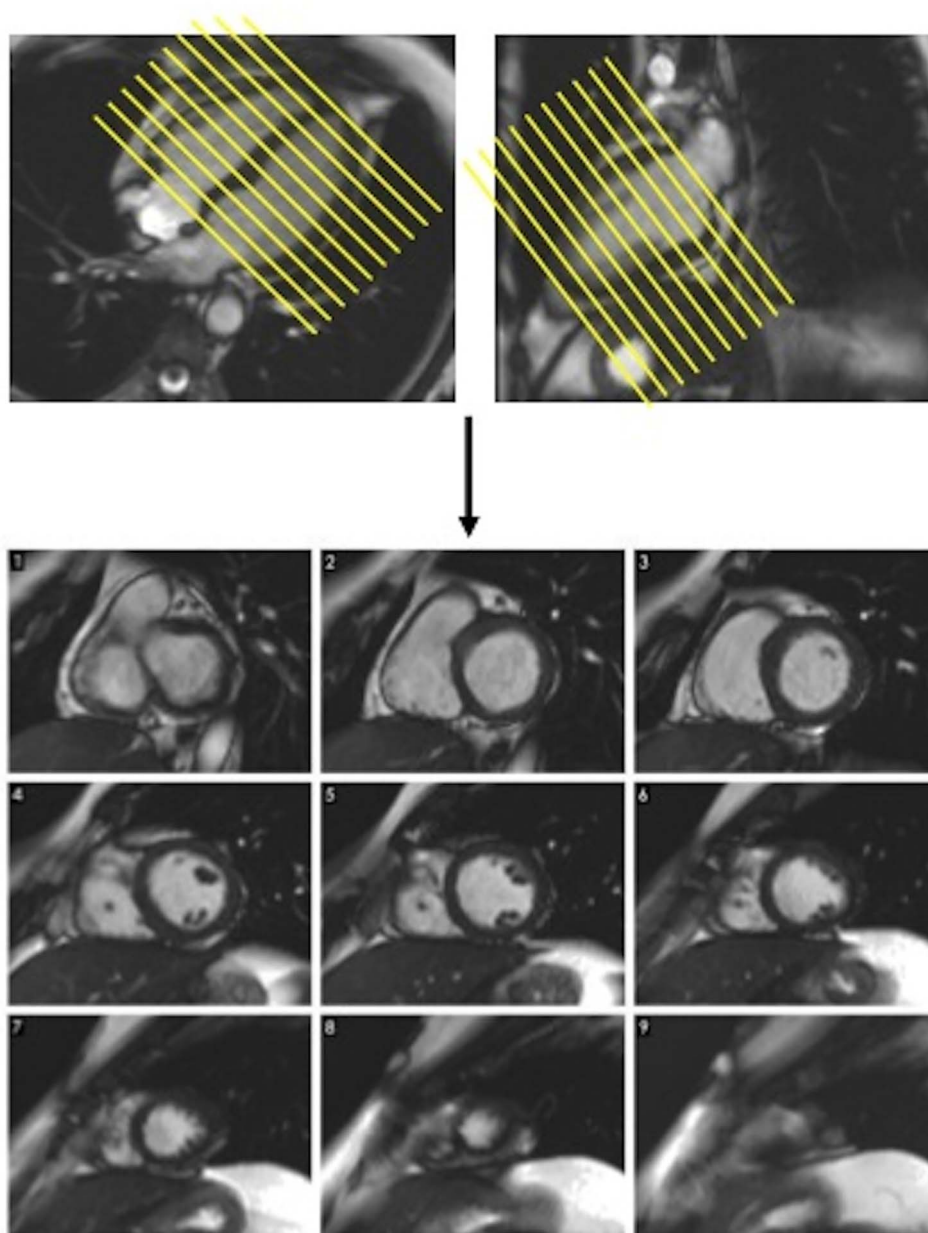


Figure 1 Top panel shows the four-chamber and two-chamber cines. Lower panel (1–9) short-axis cine dataset covering the heart, obtained by cutting the heart from base to apex.

while in suspected anomalous coronary arteries and myocardial bridging a chronotropic and inotropic agent (dobutamine) is more appropriate.

Recent technical developments in accelerating imaging acquisition¹⁵ and the introduction of 3 T (higher magnetic field strength) has improved the diagnostic accuracy of CMR stress imaging (better visualisation of small perfusion defects), capitalising on higher field homogeneity and higher performance gradients than at 1.5 T.¹⁶ However, the field homogeneity is more complex to achieve at 3 T vs 1.5 T and any remaining inhomogeneity does affect image quality.

Late gadolinium enhancement imaging

This T1-weighted technique is the cornerstone of myocardial tissue characterisation. It can

demonstrate the presence and extent of myocardial scarring.

In brief, the gadolinium-chelate contrast agent administered intravenously promptly diffuses into the extracellular myocardial compartment. In normal myocardium, the contrast quickly washes in and out of the myocardium. The presence of myocardial scarring results in increased extracellular space, where the contrast accumulates with longer washout time compared to normal myocardium (figure 2). The accumulation and distribution of the contrast agent in the diseased myocardium follows the pathophysiological process of the underlying condition. In IHD, the myocardial ischaemic-necrotic wave-front phenomenon starts in the subendocardium, becoming progressively more transmural with ischaemic time. Therefore, in

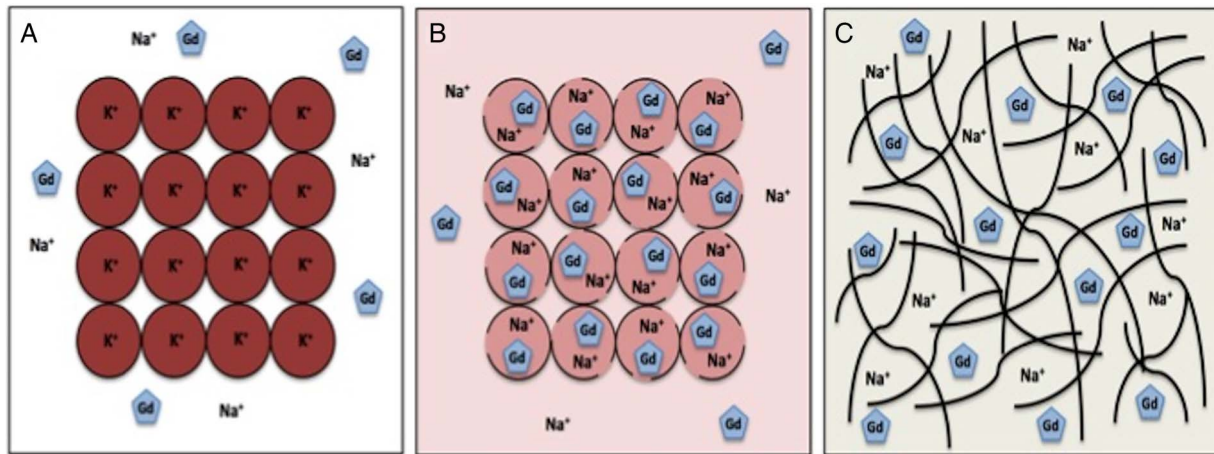


Figure 2 Gadolinium kinetics in normal myocardium and acute and chronic myocardial infarction. (A) Normal myocardium with an intact cell membrane. Gadolinium washes in and then promptly washes out of normal myocardium. (B) Acute myocardial infarction with ruptured cell membrane. Gadolinium washes out of acutely infarcted myocardium less quickly than normal due to an expanded volume of distribution as a result of disruption to the cell membrane. (C) Chronic myocardial infarction with collagen matrix scar formation. Gadolinium washes out of chronic myocardial infarction less quickly than normal due to increased interstitial space within the collagen matrix scar. K+=potassium ion; Na+=sodium ion; Gd, gadolinium. Adapted from Kim *et al.*¹¹⁰

IHD the LGE pattern can be either subendocardial or transmural, with the infarcted myocardium appearing as a hyperintense (bright) area (accumulation of contrast) and the normal myocardium as a hypointense area (no accumulation of contrast).

LGE volume changes, in the minutes following contrast administration in acute but not in chronic myocardial infarction (MI). In acute infarct transmural 25 min postcontrast injection better predicts infarct size and functional recovery.¹⁷

In the setting of acute MI, LGE imaging can also detect areas of hypointensity within the bright infarcted area, representing areas of microvascular

obstruction (MVO), precluding entry of gadolinium into these areas.

In patients with IHD, a standard CMR imaging protocol includes both cine and LGE imaging; myocardial oedema can be added in the acute setting to confirm the presence/absence of STIR. In addition, stress/rest first-pass perfusion can help to delineate the presence of inducible myocardial ischaemia (table 1). A typical CMR scan duration is ~45 min (figures 3 and 4).

Native T1 and extracellular volume fraction quantification (ECV)

Both native T1 and extracellular volume fraction quantification (ECV) are new technical developments offering an unprecedented opportunity to quantify changes in myocardial intracellular and extracellular compartments in a non-invasive manner, bringing a new dimension to myocardial tissue characterisation beyond conventional LGE imaging.

While alteration in native T1 may result from processes affecting the intracellular and/or extracellular compartments of the myocardium, ECV specifically quantifies expansion of the interstitial space. Currently, these sequences are mainly used for research, but their use for improved diagnosis and prognostication is promising.¹⁸

B) THE CMR CLINICAL REPORT

Images acquired by CMR need to be interpreted in the appropriate clinical context. Clinical information, including ECG, echocardiography, coronary angiography and cardiac biomarkers, is desirable to be able to tailor image acquisition based on the clinical question, and to allow a more accurate interpretation of the findings. A typical CMR report includes a description of the findings, followed by the clinical interpretation of the findings. Figure 5 illustrates a step-by-step approach on the

Table 1 CMR protocol for ischaemic heart disease¹⁰⁷

	Clinical application
(A) Acute MI or acute coronary syndromes	
1. Cines (short and long axis)	LV function Ischaemia/viability (during dobutamine)
2. Advanced tissue characterisation sequences (eg, T2 weighted imaging, T1 mapping)	Myocardial oedema
3. Optional—first pass perfusion at rest Consider stress in bystander disease assessment	MVO Ischaemia
4. Early gadolinium enhancement	MVO Thrombus
5. Late gadolinium enhancement	Viability Infarct size
(B) Chronic ischaemic heart disease	
1. Cines (short and long axis)	LV structure and function Ischaemia/viability (dobutamine)
2. Optional—low-dose dobutamine with 5–10 min infusion of 10 µg/kg/min of dobutamine	Assess contractile reserve (improvement in wall thickening)
3. Optional—adenosine stress–rest perfusion or high-dose dobutamine functional imaging	Inducible ischaemia
4. Early gadolinium enhancement	Thrombus
6. Late gadolinium enhancement	Viability Infarct size

CMR, cardiac MRI; LV, left ventricle; MI, myocardial infarction; MVO, microvascular obstruction.

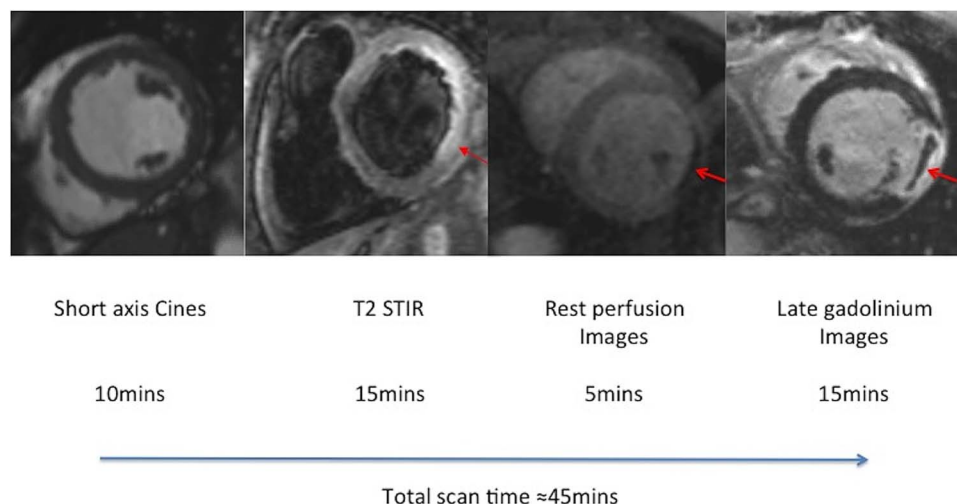


Figure 3 A case of acute ST elevation myocardial infarction with culprit diagonal coronary artery. The short axis cines showing the wall motion abnormality, T2 short- τ inversion recovery (STIR) imaging showing myocardial oedema or area at risk (arrow), rest perfusion showing early microvascular obstruction (arrow) and late gadolinium imaging showing lateral wall transmural enhancement with microvascular obstruction (arrow).

analysis and interpretation of a typical CMR report.

C) COMPARISON WITH OTHER IMAGING MODALITIES

In the assessment of IHD, clinicians are often posed with the dilemma of which non-invasive cardiovascular imaging modality to choose. CMR is

superior to several other functional imaging modalities, especially in certain circumstances, like in obese subjects, in females, in acute myocardial injury and in the assessment of myocardial viability. Table 2 summarises the relative merits and demerits of the different functional imaging modalities available, highlighting the superiority of CMR.^{19 20}

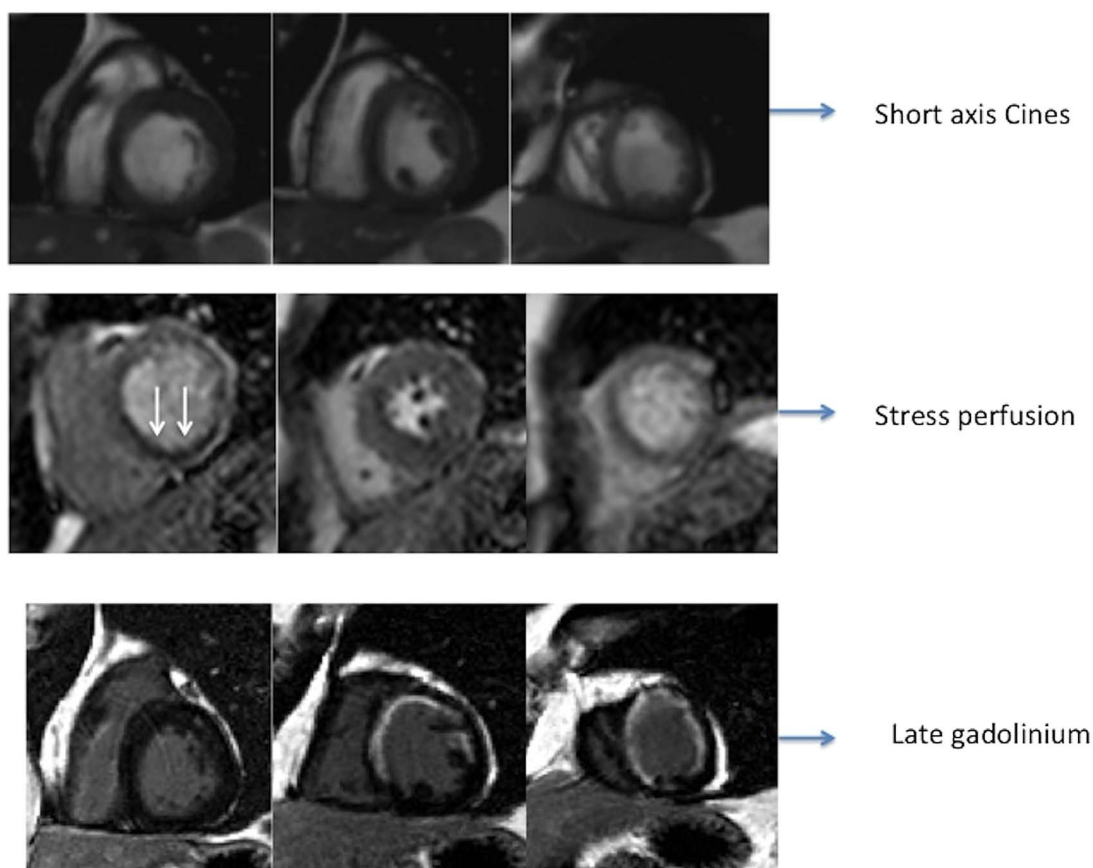


Figure 4 Assessment of chronic ischaemic heart disease: A patient with previous anterior myocardial infarction assessed for ischaemic heart disease. Stress perfusion imaging showed basal inferior perfusion defect (arrow), and late enhancement imaging showed transmural myocardial infarction in the mid-distal left anterior descending territory with viable inferior wall.

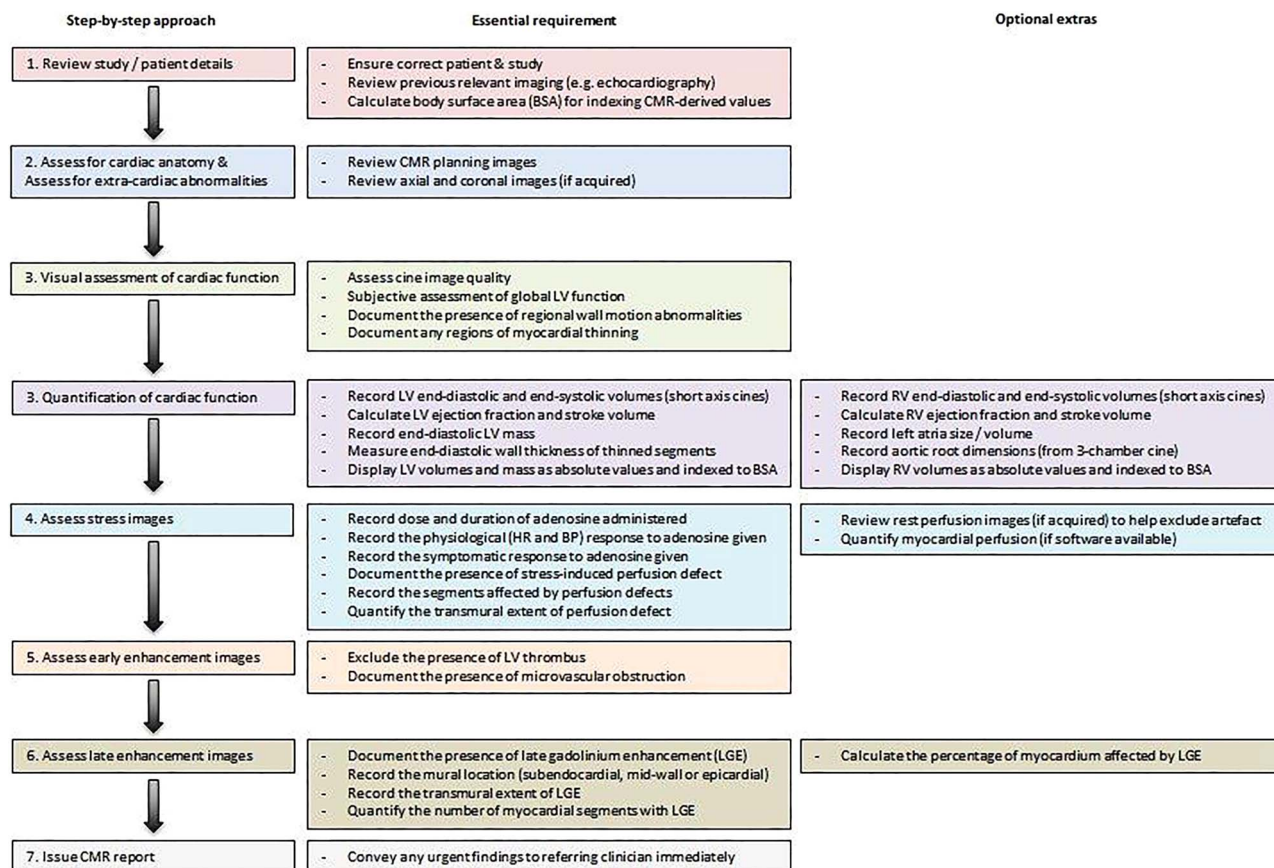


Figure 5 Step-by-step approach in reporting and interpretation of CMR. CMR, cardiac MRI; LV, left ventricle; RV, right ventricle.

D) CMR IN CLINICAL DECISION-MAKING—ACUTE CORONARY SYNDROMES

Non-ST elevation acute coronary syndrome

The evidence for using CMR in NSTEMI-acute coronary syndrome (ACS) is growing. The 2011 European Society of Cardiology (ESC) guidelines on the management of NSTEMI-ACS suggest that CMR should be considered in few specific settings²¹ (box 1).

CMR has a role in the detection of ACS in low-risk patients presenting with chest pain, normal ECG, normal cardiac biomarkers and prior to deciding on an invasive strategy. The sensitivity and specificity of CMR for detecting subsequent ACS in patients presenting with cardiac chest pain without MI is high (84% and 85%, respectively).²² By adding oedema imaging, the diagnostic accuracy increased further (up to 93%).²³ A normal stress CMR has a high negative predictive value in troponin-negative ACS.²⁴ In an NSTEMI study, stress CMR reliably predicted significant coronary stenosis (sensitivity, 96%; specificity, 83%).²⁵ Moreover, CMR assessment was superior to TIMI risk score.²⁵

In patients with multivessel disease (MVD), myocardial oedema imaging (T2-weighted sequence) can help in identifying the culprit vessel.²⁶

However, the CMR studies on NSTEMI-ACS are small and single centre. CMR within the emergency department is hardly used due to limited access, long scanning times and the cost. However, in our institution, it is sometimes offered prior to angiogram, to assess myocardial viability (LGE

imaging) and identify the culprit vessel (oedema imaging) to guide revascularisation.

ACS with unobstructed coronaries

In 7%–15% of patients presenting with ACS, no significant coronary obstruction on conventional coronary angiography is identified, representing a diagnostic and clinical dilemma.^{27–28}

These patients are thought to have a better prognosis, therefore they do not always receive secondary prevention medications.²⁹ However, a recent study suggests an overall all-cause mortality of 4.7% at 12 months.³⁰ CMR can play an important role in detecting the underlying diagnosis, such as acute myocarditis, acute MI with either spontaneous recanalisation or embolic, stress (Tako Tsubo) cardiomyopathy or other cardiomyopathies.^{31–33} However, in 1/3 of these patients no abnormalities are identified by CMR.³¹

ST-elevation MI

The morbidity and mortality from STEMI varies based on patient and treatment factors. Infarct size is directly associated with mortality. Patients with an infarct >12% of the LV have 7% mortality at 2 years compared to 0% with an infarct <12%.³⁴ The amount of transmural scar can predict adverse LV remodelling, with significant additional predictive value over troponin.³⁵ LGE-derived infarct size is a stronger predictor of all-cause mortality than LVEF and LV volumes in healed MI patients.³⁶

Table 2 Comparison of different functional imaging modalities in the assessment of IHD (CMR vs Echo vs SPECT)

	CMR	Echo	SPECT
Ejection fraction	+++	++	+
RWMA	+++	++	—
Wall thinning	++	+	—
Myocardial oedema	++	—	—
Viability	+++	++	+++
MVO	+++	—	—
Thrombus	++	+	—
Valve assessment	+	+++	—
RV	+++	++	+
Ischaemia assessment			
Increased BMI	++	+	++
Female	++	++	+
Adenosine	++	+	++
Dobutamine	++	++	++
Exercise	(+)	+++	++

BMI, body mass index; CMR, cardiac MRI; IHD, ischaemic heart disease; MVO, microvascular obstruction; RV, right ventricle; RWMA, regional wall motion abnormality; SPECT, single-photon emission computed tomography.

Transmurality is associated with worse outcome, and CMR-derived acute infarct size is a stronger predictor of future events.³⁷ Several methods exist to quantify transmurality, including semi-automated techniques (mainly used in research).³⁸ In

Box 1 Recent European Society of Cardiology (ESC) guidelines: Indications for use of cardiac MRI (CMR) in ischaemic heart disease (IHD)^{21 98 108}

In Non ST-Elevation—acute coronary syndrome (ESC Guideline 2011)²¹

In patients without recurrence of pain, normal echocardiography (ECG) findings, negative troponins tests, and a low-risk score, a non-invasive stress test for inducible ischaemia is recommended before deciding on an invasive strategy

CMR imaging is useful to assess myocardial viability and to detect myocarditis

In ST-Elevation Myocardial Infarction (STEMI) (ESC Guideline 2012)¹⁰⁸

CMR may be used as an alternative for assessment of infarct size and resting left ventricular (LV) function, if echocardiography is not feasible, after STEMI. For patients with multivessel disease, or in whom revascularisation of other vessels is considered, stress imaging to demonstrate ischaemia and viability is an option

In stable coronary artery disease (chronic ischaemic heart disease)⁹⁸

Basic testing: CMR is recommended to evaluate ventricular function and define structural cardiac abnormalities when transthoracic echocardiogram is suboptimal

Diagnosing ischaemia: Perfusion CMR or dobutamine stress CMR has good diagnostic accuracy

Risk stratification: LV function is the strongest predictor of long-term survival. In addition, new wall motion abnormalities (≥ 3 segments in the 17 segment model) induced by stress or stress-induced reversible perfusion deficits $>10\%$ (≥ 2 segments) of the LV myocardium regarded as high risk

Assess coronary anatomy: MR coronary angiography can delineate coronary anatomy. It is used for research purpose and is not recommended for diagnostic evaluation of stable CAD.

reperfused STEMI patients time to reperfusion determines the extent of reversible and irreversible myocardial injury as assessed by CMR. Particularly, salvaged myocardium is markedly reduced when reperfusion occurs >90 min of coronary occlusion.³⁹

Both CMR-derived infarct size and myocardial salvage have prognostic importance.^{40 41} Myocardial oedema is maximal and constant over the first week post MI, thereby providing a window for the retrospective evaluation of AAR whereas LGE recedes over time with corresponding recovery of function, indicating that acutely detected LGE does not necessarily equate with irreversible injury and may severely underestimate salvaged myocardium.⁴²

CMR is not routinely performed in STEMI patients. However, it provides an opportunity to further understand the degree of LV dysfunction and underlying myocardial damage.

Microvascular obstruction

This is a feature of acute myocardial damage, encountered in up to 30% of patients with STEMI.⁴³ The presence and extent of MVO after acute MI is associated with adverse LV remodelling and poor clinical outcome,^{44–46} and myocardial segments exhibiting MVO are more likely to develop wall thinning and not regain function.⁴⁷ MVO is a powerful predictor of global and regional functional recovery than the transmurality.⁴⁸ CMR-derived infarct size and MVO provide independent and incremental prognostic information in addition to clinical risk scores and LV ejection fraction.⁴⁹ In another study, MVO was an independent predictor of major adverse cardiac events (MACE) and cardiac death, whereas infarct size was not.⁵⁰ De Waha *et al*⁵¹ showed that the ratio of MVO/infarct size is a more powerful predictor for long-term outcome than either parameter alone.

Intramycardial haemorrhage

Intramycardial haemorrhage (IMH) is another sequelae of microvascular damage, appearing as hypointense areas in the rest first-pass perfusion and LGE images, similarly to MVO. The distinctive aspect of IMH compared with MVO is the hypointense signal also in the T2-weighted images caused by the haemoglobin breakdown products, not observed in MVO. Therefore, T2-weighted images are essential for distinguishing IMH from MVO.

Infarct size, myocardial salvage, MVO and IMH measured by CMR are increasingly used as surrogate endpoints in clinical trials of acute MI and reperfusion strategies.⁵² For example, the EXPIRA trial showed that in STEMI, thrombectomy prevents thrombus embolisation and improves microvascular integrity by reducing the presence and extent of MVO and infarct size measured by CMR.⁵³ Another study showed that IMH can predict MACE and adverse remodelling; however the study failed to show an improvement in the predictive value of LGE-CMR by adding T2 imaging.⁵⁴

Whether CMR is the best non-invasive imaging surrogate marker to assess improvement after treatment is debateable and further studies are needed to clarify this aspect.

STEMI with MVD

It is estimated that 40%–65% of the patients presenting with STEMI have MVD.^{55 56} The current ESC guidelines on STEMI advise a stress imaging guided approach (including CMR) to guide complete revascularisation in bystander disease detected at the time of primary percutaneous coronary intervention (PPCI). Adenosine stress CMR performed 1–5 days post-PPCI has been shown to be safe and effective for detection of ischaemia in non-culprit coronary stenosis with excellent overall diagnostic accuracy.⁵⁷ Dastidar *et al*⁵⁸ demonstrated that less than 40% patients undergoing PPCI with moderate to severe MVD needed further revascularisation when a stress CMR gatekeeper approach was used. The role of invasive and non-invasive imaging in this patient group is currently being investigated and debated.

Complications of ACS

CMR is superior to echocardiography for the identification of ventricular thrombi. They are easily identifiable early after contrast administration when both the cavity and the myocardium still appear bright, while the avascular thrombus does not take up contrast and appears hypointense.^{59 60} CMR is also able to detect other complications of MI including ventricular aneurysm, pseudoaneurysm, papillary muscle infarction with subsequent mitral regurgitation and ventricular septal defects that could lead to cardiac rupture⁶¹ (figure 6).

CMR can also detect RV involvement in acute MI.⁶² Early postinfarction RV ischaemic injury is common and is characterised by myocardial oedema, LGE and functional abnormalities. RV injury is not limited to inferior infarcts but also common in anterior infarcts.⁶³ RV infarction detected by CMR is a strong and independent predictor of clinical outcome after acute reperfused STEMI.^{64 65}

CMR IN CLINICAL PRACTICE—CHRONIC IHD Myocardial viability

Numerous studies have demonstrated that LV dysfunction in patients with coronary artery disease (CAD) may be reversible (myocardial stunning or hibernation).⁶⁶ Hibernating myocardium is in a downregulated functional state as a consequence of chronic ischaemia, but maintains the possibility to regain function if coronary blood flow is restored. Stunning is the dysfunction related to acute ischaemia.

LGE CMR allows the differentiation of viable from non-viable myocardium, predicting which dysfunctional myocardial segments could improve after successful revascularisation. However, extensive LV remodelling (LV end-systolic volume >141 mL) may limit functional improvement after

revascularisation, with negative long-term prognostic effects, despite the presence of viability.⁶⁷

A meta-analysis demonstrated significant survival benefit of revascularising patients with viable myocardium over medical management, with no significant difference between the treatments in non-viable myocardium,⁶⁸ thereby establishing the importance of viability. Assessing myocardial viability to guide management in chronic ischaemic systolic LV dysfunction is recognised in the 2014 ESC/EACTS guidelines on myocardial revascularisation.⁶⁹

The STICH trial recently questioned the role of viability, showing that in patients with CAD and LV dysfunction the assessment of myocardial viability did not identify patients with a different survival benefit from coronary artery bypass graft surgery, as compared with medical therapy alone.⁷⁰ However, viability imaging was at the physician's discretion, several modalities were used (but no CMR) and the definition of viability was questionable. Other studies are underway to clarify the role of viability in patients with LV dysfunction.

Multiple imaging modalities are available to assess viability, such as dobutamine stress echocardiography, single-photon emission computed tomography (SPECT) and positron emission tomography (PET).⁶⁸ The ESC/EACTS guidelines on myocardial revascularisation⁶⁹ recognise the high diagnostic accuracy of CMR for assessing the transmural extent of myocardial scar combined with its ability to assess contractile reserve. CMR has a high spatial resolution, enabling to detect up to 1 g of infarcted myocardium (up to 10 g with SPECT).⁷¹ In addition, the reproducibility of CMR assessment of chronic infarct is excellent.⁷² However, the guidelines also concede that the overall differences in performance between modalities are small and that local experience and availability are likely major determinants.⁶⁹

Two CMR parameters mainly used to assess myocardial viability are infarct transmural extent with LGE and contractile reserve with dobutamine CMR. In a study on patients with ischaemic LV dysfunction, the transmural extent of LGE predicted LV functional recovery after revascularisation.⁷³ In particular, the absence of LGE corresponded to a 78% chance of recovery at 3 months compared with 10% with 51%–75% transmural LGE. However, in 1%–50% transmural LGE, the chance of functional recovery was indeterminate and approximately 50%. Similar results were reproduced by other groups.⁷⁴

Low-dose dobutamine CMR is superior to LGE-CMR as a predictor of segmental recovery particularly in segments with 26%–75% LGE.⁷⁵

End-diastolic wall thickness

Myocardial thinning often represents myocardial scarring from previous infarction⁷⁶ with end-diastolic wall thickness (EDWT) <6 mm carrying a low probability of postrevascularisation functional recovery,⁷⁷ but thinned myocardium could also represent hibernating viable myocardium.⁷⁸ Therefore,

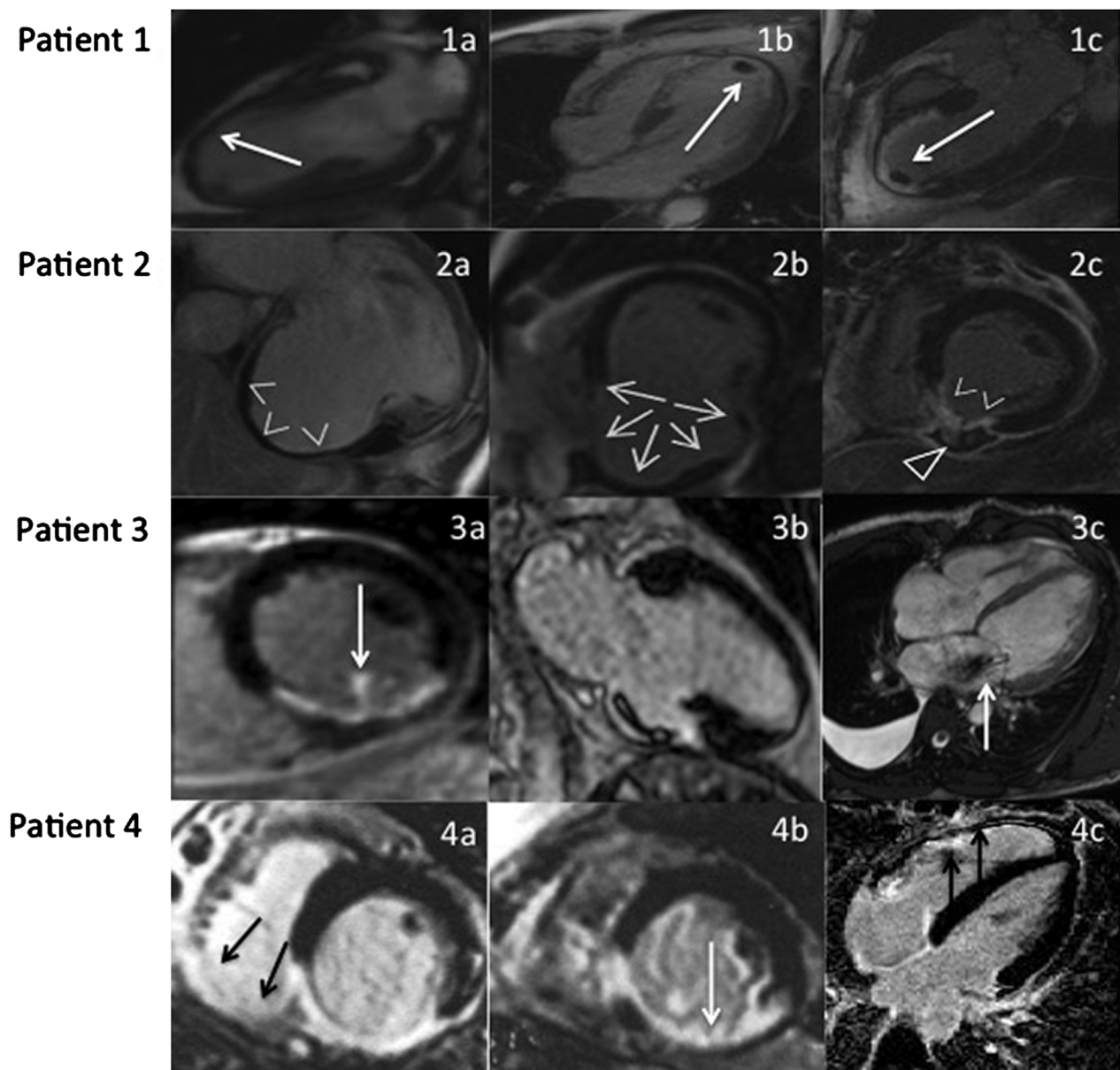


Figure 6 Complications in acute coronary syndrome (ACS). Cardiac MRI (CMR) of four ST elevation myocardial infarction; Pt 1 – 1a-c, pt 2 – 2a-c, pt 3 – 3a-c and pt 4 – 4a-c. 1a long axis cine showing thinned and akinetic anterior wall (arrow), 1b Early Gadolinium Enhancement (EGE) showing anterior infarction with apical LV thrombus (arrow), 1c LGE showing transmural infarct in LAD territory with thrombus (arrow), 2a EGE showing large laminar thrombus adherent to the aneurysmal cavity (arrowheads). 2b, The late image demonstrates a full-thickness myocardial infarction of the inferior wall (arrows), 2c post surgical repair CMR image showing inferior infarction but reduced in size (arrowheads), and the low signal structure observed represents surgical suture material (triangle). 3a & 3b - inferior infarction with complete papillary muscle infarction (arrow), 3c - 4chamber cine showing mitral regurgitation (arrow). 4a-c LGE showing inferior STEMI (white arrow) with RV infarction (black arrows).

absolute EDWT (thinning) per se should not represent a marker of viability. However, a study by Baer *et al*⁷⁹ demonstrated that the presence of significantly reduced LV end diastolic wall thickness reliably indicates irreversible myocardial damage.

Regional wall motion abnormality (RWMA/contractile reserve)

RWMA is only present when the transmural infarct extension is >50%^{80 81} and not in the presence of smaller infarctions. Thus, RWMA per se underestimates infarct size.

Stress CMR with dobutamine follows the same protocol used in echocardiography: the agent is

administered at increasing doses until target heart rate is achieved (might require the administration of atropine). In the presence of a flow-limiting coronary stenosis, the myocardium will display new RWMA on cine images as a surrogate for ischaemia. Conversely, improvement of RWMA during low-dose dobutamine represents a marker of myocardial viability.⁸² The sensitivity and specificity of dobutamine CMR is superior to dobutamine stress echocardiography.⁸³ Quantifying RWMA by myocardial tagging^{84 85} or postprocessing feature tracking software can increase diagnostic accuracy.⁸⁶

A meta-analysis highlighted the importance of integrating CMR parameters for viability

Table 3 CMR findings in assessment of IHD and its impact on clinical management

	Impact on clinical management*	Prognostic
LV EF	Guide heart failure therapy CRT/ICD implantation	Yes
RWMA	Assessment of hibernation	No
LGE	Distinguish ischaemic from non-IHD Assessment of viability (Guide revascularisation) Help in LV lead implantation (presence of transmural posterolateral LGE—poor responder)	Yes
Ischaemia	Diagnosis of IHD as the cause of chest pain Guide revascularisation	Yes
MVO	Predictive of poor clinical outcome (adverse remodelling) Extended hospital stay and close follow-up after discharge	Yes
Thrombus	Delineate the character and location Initiation of anticoagulation therapy	No
Myocardial oedema	Differentiate acute from chronic Assessment of area at risk and salvaged myocardium	Yes
RV function	Guide in appropriate right heart failure management	Yes

*Further details consult the European Society of Cardiology (ESC), AHA or National Institute for Health and Care Excellence (NICE) guidelines on ischaemic heart disease.
CMR; cardiac MRI; CRT, cardiac resynchronisation therapy; ICD, implantable cardioverter-defibrillator; IHD, ischaemic heart disease; LGE, late gadolinium enhancement; LV EF, left ventricular ejection fraction; MVO, microvascular obstruction; RV, right ventricle; RWMA, regional wall motion abnormality.

assessment,⁸⁷ demonstrating that LGE provided the highest sensitivity (95%) and negative predictive value (90%), whereas low-dose dobutamine offered the best specificity (91%) and positive predictive value (93%). A combination of viability parameters (transmurality, contractile reserve, EDWT, unenhanced rim thickness and segmental wall thickening of the unenhanced rim) may be a better predictor of recovery.⁸⁸

Inducible myocardial ischaemia

The FAME study⁸⁹ established that revascularisation of patients with symptomatic stable CAD guided by the presence of myocardial ischaemia measured invasively by fractional flow reserve (FFR) is prognostically important. Furthermore, a meta-analysis demonstrated that the absence of ischaemia confers prognostic benefit, with very low annualised event rates for cardiovascular death (0.3%) and MI (0.4%).⁹⁰ The ongoing MR-INFORM study is assessing whether a CMR stress perfusion strategy is non-inferior to FFR in stable CAD.⁹¹ The ongoing ISCHEMIA trial will demonstrate whether patients with moderate–

Table 4 Contraindications of CMR

	Absolute contraindication	Relative contraindication
MRI	▶ Intracranial aneurysm clips ▶ Automated implantable cardiac defibrillator (ICD) ▶ Non-MR conditional pacemaker ▶ Pacing dependent patient ▶ Cochlear, otological or ear implant ▶ Metallic intraocular foreign body ▶ Neurostimulator ▶ Non-MR conditional monitoring devices ▶ Non-MR conditional support devices	▶ Clinically unstable patients ▶ Long-stem joint prosthesis (at 3 T only) ▶ Severe claustrophobia
Gadolinium-chelate contrast agent	▶ Previous anaphylactic reaction to gadolinium contrast ▶ Known nephrogenic systemic fibrosis (NSF)	▶ Renal impairment (eGFR <30 mL/min/1.73 m²) ▶ Hepatorenal syndrome ▶ Pregnancy
Adenosine	▶ Previous anaphylactic reaction to adenosine ▶ Previous anaphylactic reaction to regadenoson ▶ Acute coronary or aortic syndrome ▶ High degree atrioventricular block ▶ Severe obstructive airways disease ▶ Symptomatic severe or critical aortic stenosis ▶ Systolic blood pressure < 90 mm Hg ▶ Bradycardia (heart rate <40 beats per minute)	▶ Mild-moderate obstructive airways disease ▶ Caffeine <6–12 h ▶ Concomitant theophylline ▶ Concomitant dipyridamole ▶ Aberrant conduction/pre-excitation
Dobutamine	▶ Previous anaphylactic reaction to dobutamine ▶ Acute coronary or aortic syndrome ▶ Symptomatic severe or critical aortic stenosis ▶ Obstructive hypertrophic cardiomyopathy ▶ High degree atrioventricular block	▶ Hypokalaemia ▶ Left ventricular thrombus ▶ Intracranial aneurysm ▶ Abdominal aortic aneurysm ▶ Severe ventricular arrhythmias ▶ Uncontrolled arterial hypertension ▶ Uncontrolled atrial fibrillation
Atropine	▶ Acute-angle glaucoma ▶ Myasthenia gravis ▶ Pyloric stenosis	▶ Obstructive uropathy

CMR, cardiac MRI; eGFR, estimated glomerular filtration rate; ICD, implantable cardiac defibrillator; NSF, nephrogenic systemic fibrosis.

Table 5 Challenges with CMR and practical tips^{84 109}

Challenge	Possible solution
Claustrophobia	▶ Use peri-CMR sedation (but this may interfere with patient compliance or scan instructions) ▶ Perform CMR scan with the patient lying prone ▶ Provide an angulated mirror in the CMR bore to enable the patient to look out of the scanner ▶ Invite a relative/friend to sit at the end of the scanner to talk to the patient during the scan
Shortness of breath/dyspnoea	▶ Perform inspiratory, rather than usual expiratory, breath-held cines ▶ perform free breathing sequences ▶ Perform real-time imaging for visual assessment of cardiac function ▶ Perform stress perfusion free-breathing, shallow breaths ▶ Use multislice free-breathing sequences for LGE
Arrhythmia	▶ Perform prospective, rather than usual retrospective, ECG gating ▶ Use arrhythmia detection and correction scanning protocols
Tachycardia	▶ Use faster, less motion sensitive sequences
Clinically unstable patient	▶ Defer CMR imaging until stable ▶ Use CMR compatible vital sign monitoring equipment ▶ Tailor CMR protocol to minimise patient time in the CMR scanner

2D, two dimensional; 3D, three dimensional; CMR, cardiac MRI; LGE, late gadolinium enhancement; SSFP, steady state free precession.

severe ischaemia on stress imaging (SPECT, CMR, stress echo) will benefit from coronary angiography and revascularisation.⁹² When compared with PET, CMR provides a similar diagnostic accuracy to PET but without exposure to ionising radiation.⁸²

MR-IMPACT, the first multicentre, multivendor, randomised trial suggested perfusion-CMR as a valuable alternative to SPECT.⁹³ The following MR-IMPACT II showed perfusion-CMR superior to SPECT, while its specificity was inferior to SPECT.⁹⁴ The large, prospective, CE-MARC study has recently established CMR's high diagnostic accuracy in IHD and its superiority over SPECT.⁹⁵ In both genders, CMR has greater sensitivity than SPECT with no intergender differences.⁹⁶ Stress CMR also performs favourably in cost-effective analyses assessing diagnostic pathways for the work-up of suspected CAD.⁹⁷

The role of stress CMR in ischaemia assessment has been recognised in international guidelines. Stress perfusion CMR is one of the class I recommended modalities as per the 2013 ESC guidelines

on stable CAD with a pre-test probability of CAD of 15%–85%.⁹⁸ The National Institute for Health and Care Excellence (NICE) recommends non-invasive ischaemia imaging (including stress CMR) in patients with intermediate CAD risk (30%–60%) and in high risk (61%–90%) after angiography to target revascularisation in MVD or if invasive angiography is not clinically appropriate.⁹⁹ Dastidar *et al*¹⁰⁰ showed that the prevalence of myocardial ischaemia is not different in the intermediate and high likelihood of CAD. Stress CMR receives class IIa recommendations in several clinical settings in the 2012 ACC/AHA chest pain guidelines.¹⁰¹ Quantitative myocardial perfusion reserve assessment by CMR is a promising new dimension.¹⁰² In an animal model study, CMR derived quantitative blood flow estimates have been correlated with true myocardial blood flow.¹⁰³ Perfusion CMR is in theory more related to coronary flow reserve (CFR) and not FFR; however, it has been validated against CFR and FFR.^{104 105}

Recently exercise stress cardiac MRI has been investigated in healthy volunteers showing that peak exercise wall motion assessed by cine CMR is feasible and can be performed at least as rapidly as stress echocardiography.¹⁰⁶

The different CMR findings and its prognostic and clinical impact, guiding the clinician in decision-making, are summarised in [table 3](#).

E) LIMITATIONS OF CMR

[Table 4](#) highlights the common contraindications of CMR. In addition, there are certain additional challenging clinical scenarios in scanning patients using CMR. [Table 5](#) provides few tips and tricks on how to overcome these challenges.

CONCLUSION

CMR is a well-established, comprehensive, increasingly used non-invasive imaging modality for the assessment of patients with IHD, both in the acute and chronic setting. CMR can assess cardiac anatomy, function, myocardial perfusion and tissue characterisation, without exposure to ionising radiation and in <1 h scan time. Its use in IHD is supported by robust and rapidly expanding evidence. The real challenge is to delineate how CMR can improve patient management and improve clinical outcomes in a cost-effective manner.

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Key messages

- ▶ Cardiac MRI (CMR) is a well-established comprehensive non-invasive imaging modality in the assessment of patients with CAD.
- ▶ It can assess cardiac anatomy, function, myocardial perfusion and tissue viability, without exposure to ionising radiation and in <1 h scan.
- ▶ Its use in ischaemic heart disease is supported by strong and rapidly expanding clinical evidence.
- ▶ The real challenge is to delineate how CMR can improve patient management and impact upon clinical outcomes, while proving to be cost-effective.

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