

Implementation of cardiac magnetic resonance imaging in everyday cardiology practice

A clinical consensus statement of the European Association of Cardiovascular Imaging of the ESC and ESC Council for Cardiology Practice

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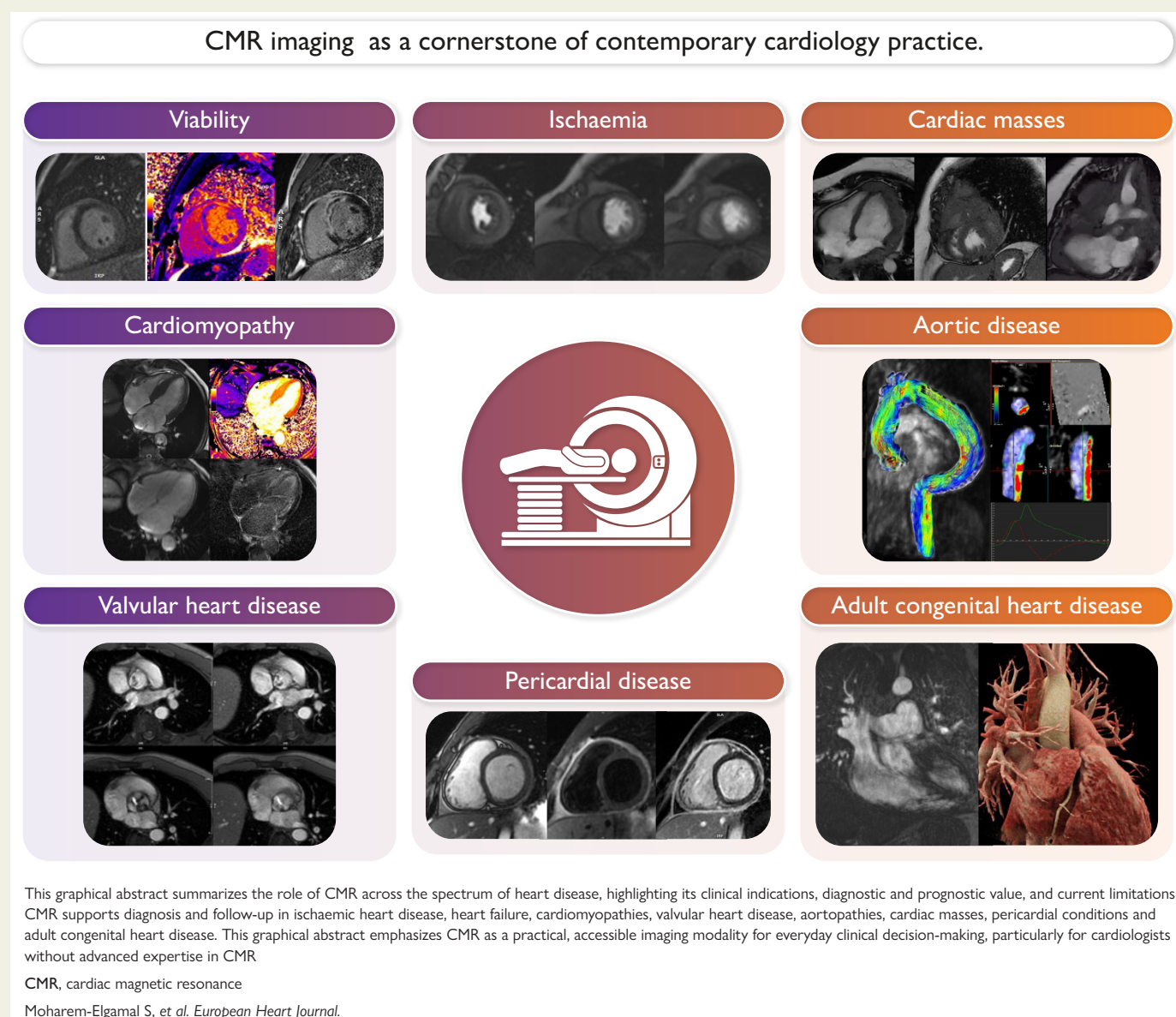
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Graphical Abstract



Abstract

Cardiac magnetic resonance (CMR) plays an established role in the assessment of cardiac patients. In this document, the aim is to demonstrate and clarify the role of CMR in the clinical management of heart disease for general cardiologists in everyday clinical practice. This document will cover the clinical indications, prognostic values, and limitations of CMR in the diagnosis and follow-up of heart disease, including ischaemic heart disease, heart failure, cardiomyopathies, valvular heart disease, aortopathies, cardiac masses, pericardial conditions, and adult congenital heart disease. This document also provides a basic understanding of the use of different CMR sequences and how cardiac function is assessed. With the increasing number of patients with cardiac implantable electronic devices, a brief understanding and advice on the use of CMR in this growing patient population who may need a scan during their lifetime is discussed. This document outlines the clinical applications of CMR and its use in everyday clinical practice for cardiologists who are not experts in CMR.

Keywords

Cardiac magnetic resonance (CMR) • Heart disease • Clinical practice

Introduction

Cardiac magnetic resonance (CMR) imaging has become a key modality in modern cardiac care, providing comprehensive non-invasive assessment of the heart's structure and function with high spatial resolution and advanced tissue characterization, which often proves pivotal in guiding clinical decision-making in a wide range of diseases. The EuroCMR Registry of 27 781 patients demonstrated that CMR led to a change in the management plan in 61.8% of patients and a new diagnosis in 8.7% of patients.¹ In this document, we will clarify the role of CMR in the clinical management of heart diseases. We aim to summarize the main clinical applications of CMR in everyday cardiology clinical practice for the general cardiologist.

In this clinical consensus statement, we will provide in-depth explanations on CMR's role across various cardiac conditions for the non-CMR expert, where we will discuss:

- How CMR can detect/assess?
- What role does CMR have in patient risk stratification?
- What clinical questions/information should be in the referral for CMR?
- What to look out for in the CMR report?

The learning objectives are (i) to provide a basic understanding of the clinical applications for the different CMR sequences; (ii) to describe what information can be obtained from a CMR study for a variety of cardiac conditions and how this can affect clinical management; (iii) to know the indications for CMR in the different cardiac conditions and how this is reflected in the current European Society of Cardiology (ESC) guidelines and recommendations; and (iv) to help manage the use of CMR in patients with cardiac implantable electronic devices (CIEDs) (*Graphical Abstract*).

Clinical applications of different CMR sequences

The CMR scan is performed using magnets of 1.5 or 3 Tesla field strength, multichannel phased-array coils, parallel imaging techniques and electrocardiogram gating (*Figure 1*). Open magnetic resonance imaging (MRI) with lower field strength can be used for functional imaging of the heart; however, higher field strength is needed for perfusion and viability imaging.² A CMR scan usually lasts from 45 to 60 min, but can be tailored to a shorter simplified scan³ or a longer scan when assessing complex congenital heart disease or cardiac masses. Abbreviated protocols, incorporating multiparametric mapping and gadolinium, can be used, allowing rapid scans (12–27 min) that provide core clinical information.³ Prior to making a referral, it is vital to know if a patient has an implant and whether it is hazardous or safe. This information can be found on online databases as www.mrisafety.com.⁴

CMR uses various pulse sequences like spin echo and gradient echo to generate images, enabling the comprehensive assessment of cardiac morphology, function, and tissue characterization (*Figure 2*). Some common sequences and their clinical applications include:

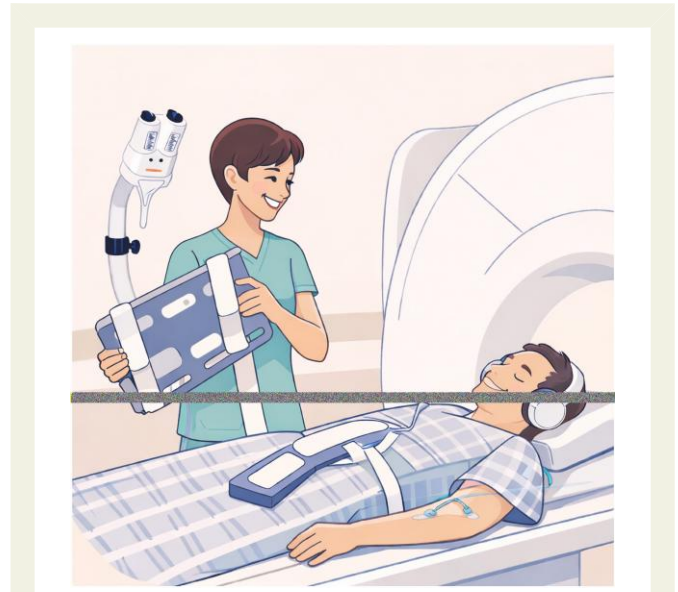


Figure 1 A patient being prepared for a CMR scan where multi-channel phased-array coils and electrocardiogram gating dots are put on his chest, Headphones are placed to protect the patient's hearing and receive instructions on when to hold his/her breath. A respiratory belt can be placed to monitor breath-holding quality. A buzzer will be given to the patient prior to going into the bore of the scanner. Here, the patient has a cannula inserted which will be connected to an IV line for the administration of gadolinium. If the patient were undergoing a stress CMR, an additional cannula, IV line, IV pump and haemodynamic monitoring equipment would be required. Animation generated by Chat GPT

- Black blood imaging with T1-weighted fast spin echo is used for the assessment of cardiac morphology, vascular structures, pericardium, and imaging of fat.
- Bright blood imaging with steady-state free precession for the assessment of cardiac structure and function, chamber size, global or regional wall motion abnormality, regional wall thinning, wall thickness, and ventricular mass by capturing cine images throughout the cardiac cycle. CMR feature tracking allows for the comprehensive assessment of myocardial deformation in the left ventricle, right ventricle, and atria without requiring additional specialized scan sequences.^{5–7}
- T2-weighted fast spin echo and T2 mapping sequences for the imaging of myocardial oedema.
- Perfusion sequence, fast gradient recalled echo, for the assessment of myocardial perfusion, and vascularity of a cardiac mass.
- T1 mapping is used in the assessment of extracellular volume expansion and diffuse fibrosis.
- 2D flow (2D phase-contrast) sequences are used to non-invasively measure and quantify blood flow, shunt volumes, and flow velocities in cardiovascular and cerebral vessels.
- 4D flow sequences also utilize phase-contrast imaging and are used to visualize and quantify blood flow in the heart and large vessels, offering a non-invasive haemodynamic assessment.
- Late gadolinium enhanced images (T1-weighted rapid GRE sequence combined with an inversion-recovery pre-pulse

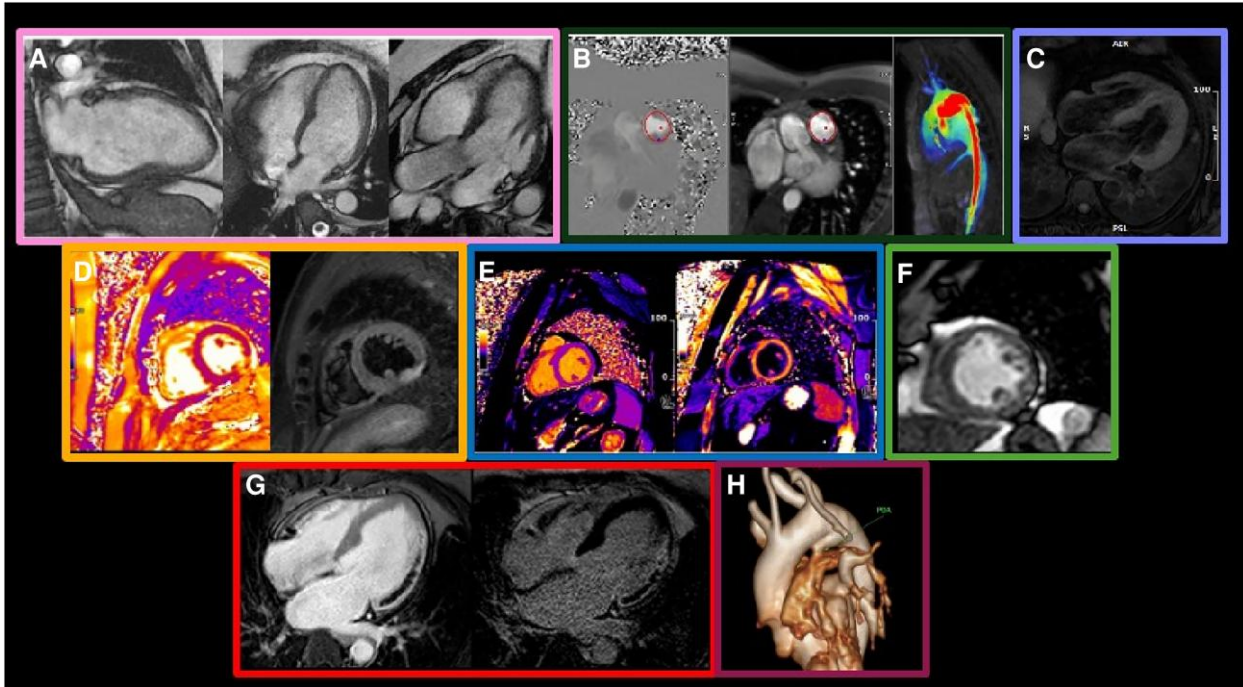


Figure 2 Common different CMR sequences. (A) CINE images allow the assessment of cardiac morphology, quantification of cardiac chambers, vascular ventricular function and regional wall motion. (B) 2D flow allows quantification of major vessel blood flow from phase-contrast images in one direction while 4D flow provides both qualitative and quantitative data on haemodynamic flow and properties within the heart and major blood vessels, allowing for simultaneous velocity encoding in all three directions. (C) T1-weighted images are used for the evaluation of cardiac morphology, vascular structures, pericardium, masses/tumours and imaging of fat. (D) STIR and T2 mapping images are used for myocardial oedema. (E) Perfusion imaging allows the assessment of rest and stress perfusion to identify myocardial ischaemia and infarct as well as if a structure/mass has a vascular supply. (F) Pre-contrast and post-contrast T1 mapping are useful for tissue characterization, particularly in the assessment of non-ischaemic cardiomyopathies. (G) Early and late gadolinium enhancement imaging are used to differentiate between scarred/fibrotic myocardium and normal myocardium, identify thrombi and areas of microvascular obstruction in patients with acute myocardial infarction. (H) MR angiograms can be performed with and without contrast to assess vascular structures

after the administration of gadolinium) are often used to detect scar and fibrosis by nulling the signal of normal myocardium, which appears dark (or no signal) and abnormal myocardium is bright (hyperenhancement).

Not all these sequences are used in every scan. Advanced sequences like 4D flow and wide-band pulse for cardiac implantable electronic device are usually limited to larger tertiary centres. The scanning protocol is tailored according to the clinical question. The referral needs to be clear, as once the patient has finished the scan, no additional images can be produced.

Only gadolinium-based contrast agents (GBCAs), which is different from the contrast used in computed tomography (CT) and invasive coronary angiography (ICA), are used in the clinical setting. When intravenous GBCAs are injected, they pass quickly, in seconds, to the cardiac chambers and coronary arteries before diffusing into the extracellular compartment of the myocardium. First-pass is used in the imaging of myocardial perfusion and some magnetic resonance angiography sequences. After the early stage (1–3 min), the contrast resides primarily in the normal myocardium and blood pool. Then, 10–15 min from injection, the contrast washes out of the normal myocardium, but

remains in the infarcted, infiltrated, or fibrosed myocardium. GBCA is safe with 0.4% of patients experiencing mild reactions (e.g. nausea and a mild skin rash), and extremely rare severe reactions.⁸ All GBCAs are chelated to render them non-toxic and to facilitate renal excretion. Previously reported nephrogenic systemic fibrosis has near-zero incidence with the switch from linear to cyclic GBCA, the use of weight-based dosing and avoidance of GBCA use in patients with estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m².⁹

Once all the images are acquired, it is assessed and analysed on a dedicated workstation. Left and right ventricular ejection fraction, end-diastolic and end-systolic volumes are calculated by drawing the endocardial and epicardial contours from short-axis cine images (Figure 3).

General limitations of CMR include claustrophobia, the need for breath holding (except if free breathing sequences are used), wide abdominal girth, haemodynamic instability, and the presence of ferromagnetic materials and non-MR-conditional devices.¹⁰ GBCAs are generally safe. Usual contraindications and precautions of GBCAs include hypersensitivity. Previous concerns of nephrogenic systemic fibrosis are now negligible with the use of cyclic GBCAs.¹¹ Severe chronic kidney disease

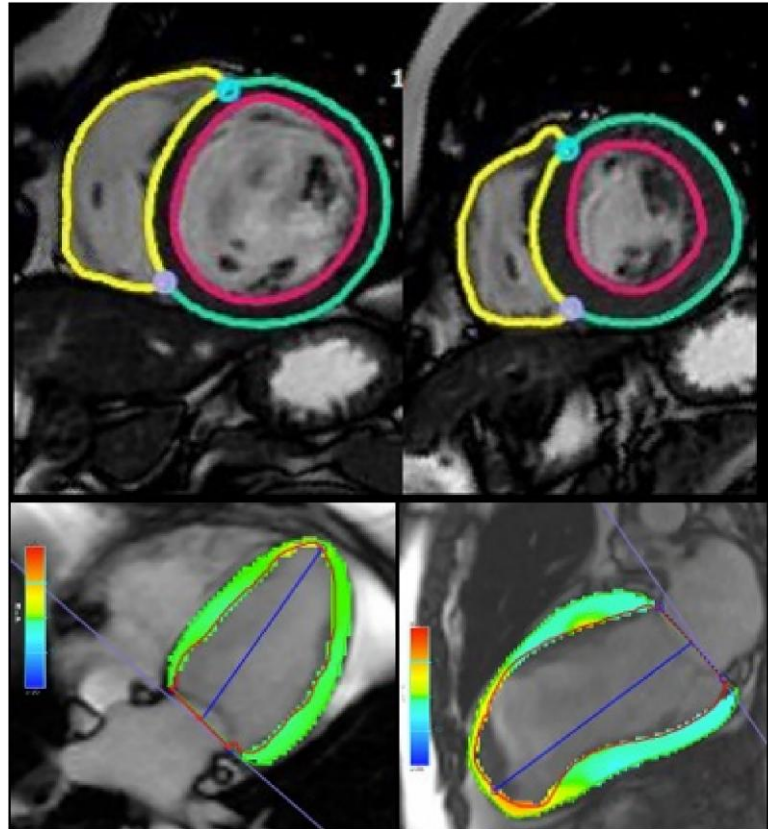


Figure 3 The top panel demonstrates the left and right ventricular ejection fraction, end diastolic and end systolic volumes being calculated by drawing the endocardial and epicardial contours from short-axis cine images. CMR Feature Tracking using apical 4- and 2-chamber views allows the non-invasive assessment of myocardial strain by tracking voxels on routine SSFP cine images

is not an absolute contraindication to CMR if it is deemed clinically necessary.¹²

Ischaemic heart disease

Ischaemic heart disease (IHD) comprises a large proportion of annual healthcare expenditure. Invasive coronary angiography is the gold standard for diagnosing coronary artery stenosis; however, management of IHD is now best guided by the physiologic significance of coronary artery stenosis. CMR provides not only useful information on systolic function and scarred myocardium but also the physiologic significance of coronary artery stenosis.¹³

Sequences used and scanning protocol:

- Cine images for the evaluation of wall motion abnormalities and biventricular function
- T2-weighted sequences (STIR and T2 mapping) to detect myocardial oedema for the differentiation of acute vs chronic myocardial injuries
- Early gadolinium enhancement (EGE) to detect microvascular obstruction (MVO) or thrombus
- Late gadolinium enhancement (LGE) sequences 15–20 min after contrast administration to assess infarct size, MVO, and thrombus

- $\pm$ stress imaging has been performed to detect inducible ischaemia

I. Acute myocardial infarction and complications

A CMR referral for post-acute myocardial infarction (AMI) is indicated when there is:

- an unclear clinical presentation to distinguish post-AMI from other aetiologies such as myocarditis/takotsubo;
 - conventional echocardiograms and/or angiograms are not diagnostic/conclusive;
 - differentiate acute from chronic myocardial infarction;
 - assess infarct size and viability;
 - diagnosis of mechanical complications
- How CMR can detect/assess?

The 2023 ESC Guidelines for the management of acute coronary syndromes recommend early risk stratification of AMI.¹⁴ Transthoracic echocardiography (TTE) constitutes the cornerstone for assessing the structural and functional consequences of myocardial infarction and for stratifying risk. CMR provides accurate measurement of left ventricular (LV) volumes and function and allows *in vivo* assessment of tissue characterization of

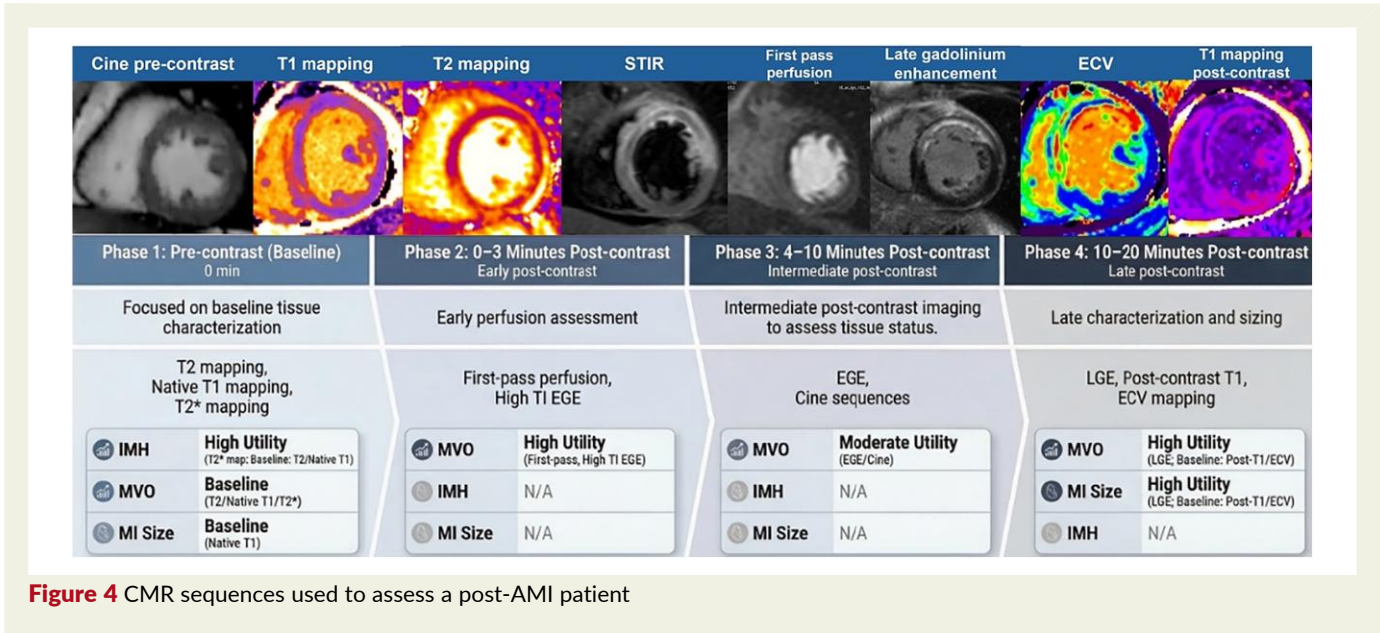


Figure 4 CMR sequences used to assess a post-AMI patient

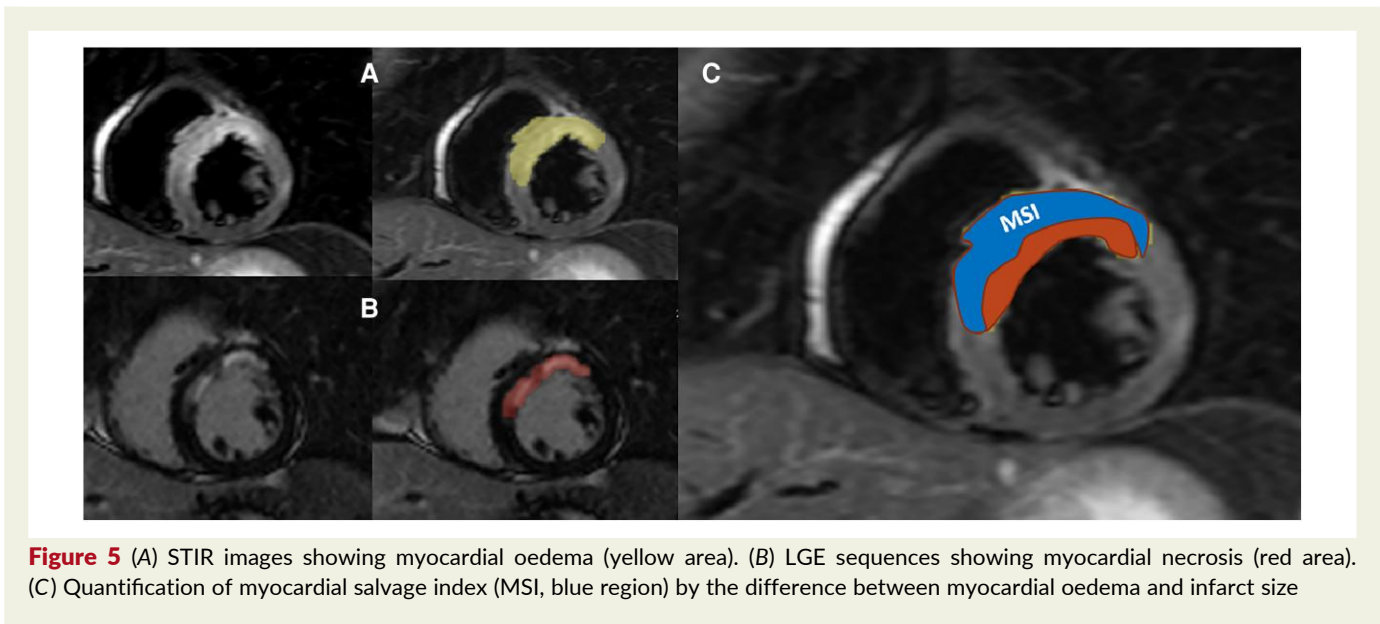


Figure 5 (A) STIR images showing myocardial oedema (yellow area). (B) LGE sequences showing myocardial necrosis (red area). (C) Quantification of myocardial salvage index (MSI, blue region) by the difference between myocardial oedema and infarct size

AMI as recently described by the Canadian Cardiovascular Society classification of AMI based on the severity of tissue damage into four sequential and progressive stages: stage 1: aborted myocardial infarction, stage 2: cardiomyocyte necrosis without microvascular injury in which LGE sequences can accurately quantify ischaemic myocardial necrosis and infarct size *in vivo* in patients, stage 3: cardiomyocyte necrosis combined with MVO, and stage 4: cardiomyocyte necrosis, MVO and reperfusion haemorrhage.¹⁵

In everyday clinical practice, CMR can provide additional diagnostic information to guide clinical management in specific scenarios—such as working diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA), delayed or incomplete revascularization, detection of LV thrombus, or suspected mechanical post-AMI complications (Figure 4).

- What role does CMR have in patient risk stratification?

CMR offers a variety of parameters which are useful for predicting outcomes post-AMI.

CMR evaluation of LV ejection fraction (LVEF)¹⁶ is used to predict outcomes and guide decisions regarding the need for implantable cardiac defibrillator (ICD) or cardiac resynchronization therapy implantation^{17–21}; however, international guideline recommendations are based on TTE measurements. T2-weighted images (STIR or T2 mapping sequences) can detect the presence of myocardial oedema, which is used to differentiate recent infarction from old scarring and estimate the myocardial area at risk²² (Figure 5) and to differentiate infarcted from injured myocardium, as in myocarditis and takotsubo cardiomyopathy.

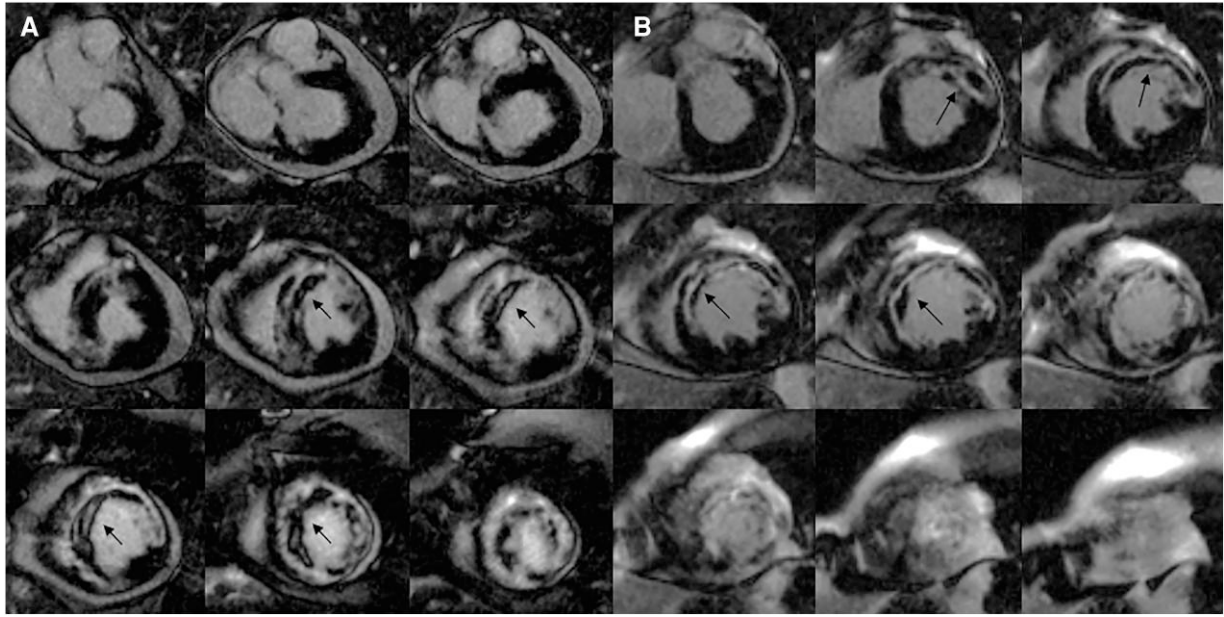


Figure 6 Short-axis LGE sequences of two different patients (A) and (B) with an anterior infarction with hypo-enhanced regions, areas of microvascular obstruction (arrow), surrounded by hyper-enhanced areas of extensive transmural infarction

The degree of infarct transmuralty is an excellent predictor of functional recovery (myocardial viability),²³ and LGE volume (a relative necrotic mass >20%) is a powerful predictor of ventricular remodelling and adverse prognosis at follow-up.^{17–21} Irreversible damage of microvascular circulation, MVO, can also be quantified on LGE sequences with complete resolution after 4–6 weeks (Figure 6).

TTE is the first-line imaging modality to evaluate mechanical complications post-AMI, particularly in haemodynamically unstable patients. However, CMR outperforms TTE in the detection of intracavitary thrombi,²⁴ right ventricular (RV) infarction,²⁵ free wall rupture²⁶ (Figure 7), and pseudoaneurysm²⁷ (Figure 8). A free wall rupture is characterized by a narrow neck connecting the LV cavity to the sac (neck to sac ratio <0.5), the 'myocardial cutoff sign' (a > 50% reduction in myocardial wall thickness 1 cm from the neck) as well as intense pericardial enhancement and thrombus on LGE images.²⁸

- What clinical questions/information should be in the referral?

Summary of the patient's clinical presentation, ICA findings and treatment performed, as well as the patient's haemodynamic status, presence of any device (e.g. Swan-Ganz), suspicion/confirmation of a mechanical complication and most recent eGFR.

- What to look out for in the CMR report?

In post-AMI, the report should include:

- Biventricular volumes and function: for risk stratification and measuring adverse remodelling over time.
- A description of the regional wall motion abnormalities that should conform to a specific coronary artery territory;

- Presence of myocardial oedema: It can differentiate acute from chronic myocardial infarction. Evaluating the area of myocardial oedema and comparing it with the area of LGE can identify the salvaged myocardium.
- Size, transmuralty and location of myocardial infarction scar by LGE: The degree of infarct transmuralty and myocardial viability can predict the functional recovery. Myocardial viability can guide revascularization decision-making in late AMI presentations. The larger infarcts indicate a higher risk of adverse remodelling, heart failure and mortality.
- MVO: The presence of MVO indicates severe myocardial injury
- Presence of LV thrombus
- Presence of mechanical post-AMI complications with a critical role in the differentiation of free wall rupture from pseudoaneurysm

II. MINOCA

The term MINOCA has undergone significant evolution since its introduction. It is useful to think of MINOCA in terms of both a working and final diagnosis. The working diagnosis of MINOCA is now defined as a clinical scenario when a patient presents with symptoms suggestive of acute coronary syndrome, with troponin elevation and non-obstructive (<50% stenosis) coronary arteries at the time of ICA.¹⁴ In reality, it comprises many conditions that are not myocardial infarcts, including myocarditis and takotsubo.^{29,30} It should therefore be distinguished from a final diagnosis of MINOCA, where a type 1 or type 2 myocardial infarction has been confirmed. CMR represents a key investigation to work through the diagnostic pathway towards a final diagnosis of MINOCA (Figure 9).^{31,32} Patients with suspected MINOCA should be scanned within

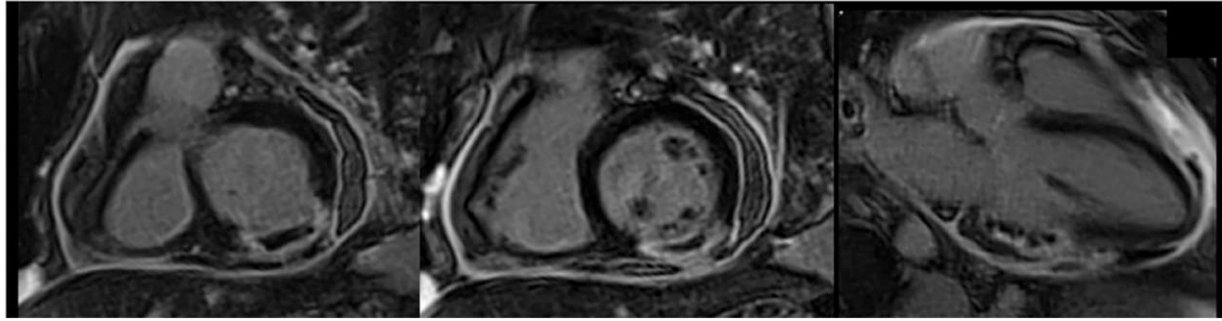


Figure 7 LGE images of a large transmural myocardial infarction which most likely represents free wall rupture and associated pericardial inflammation and effusion

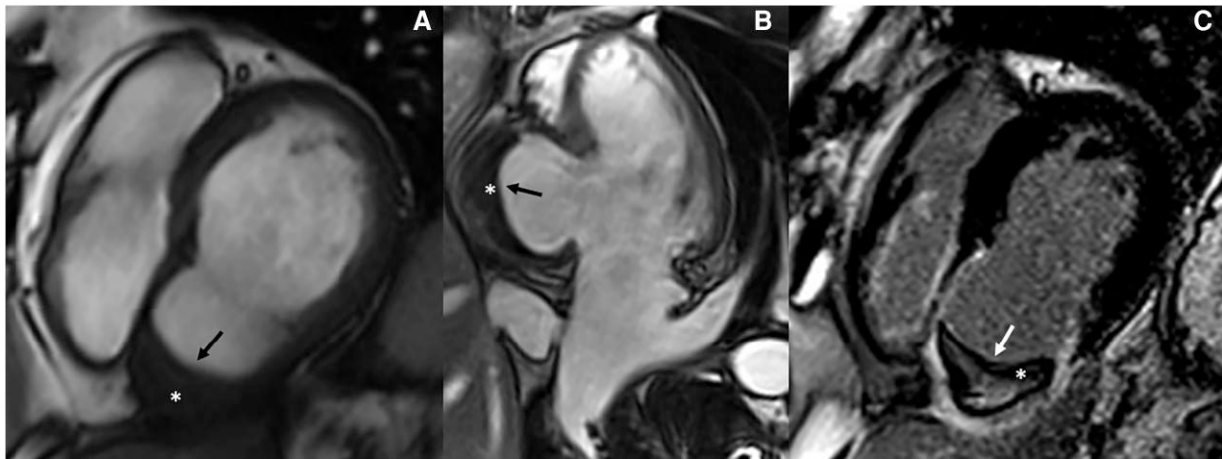


Figure 8 Inferior pseudoaneurysm (arrow) in a patient with a transmural inferior infarction. The aneurysm neck is smaller than the aneurysm sac. Thrombus (*) is seen inside the cavity: (A) SSFP cine, (B) post-contrast SSFP cine, and (C) LGE sequences

7–14 days of symptom onset.³³ Beyond 2 weeks, the ability to identify myocardial oedema decreases.

- How CMR can detect/assess?

The most useful CMR information in the evaluation of patients with a working diagnosis of MINOCA are the quantification of cardiac chamber size and systolic function, the identification of oedema through T2-weighted sequences, characterization of myocardial injury by LGE sequences, and the quantification of the extracellular volume (ECV) by the implementation of T1 and ECV mapping. The evidence of a normal CMR study or the identification of LGE with an infarct distribution (i.e. subendocardial or transmural) allows the final confirmation of the MINOCA diagnosis, whereas the detection of non-ischaemic LGE patterns (subepicardial or intramyocardial) together with the use of cine, T1, T2, and ECV sequences to identify a non-infarct aetiology such as myocarditis or a cardiomyopathy.

- What role does it have in prognosis/risk stratification?

Besides its important diagnostic role, CMR allows the risk stratification of patients with a suspected diagnosis of

MINOCA. The early performance of CMR after ICA allows a specific diagnosis in >70% of cases,^{34–38} thereby enabling specific disease and prognosis-modifying treatments. Moreover, evidence exists in the literature that patients with a confirmed MINOCA diagnosis and normal CMR show a worse prognosis compared with patients with evidence of an ischaemic scar pattern, as MINOCA patients were less likely to receive guideline-recommended myocardial infarction therapy compared with those with CMR-confirmed myocardial infarction.³⁹

- What clinical questions/information should be in the referral?

Clinical context, symptoms suggestive of acute coronary syndrome with troponin elevation, time of symptom onset, the ICA findings and no other overt cause for the clinical presentation.

- What to look out for in the CMR report?

In patients with suspected MINOCA, the CMR report should include a comprehensive assessment of ventricular volumes, stroke volume, ejection fraction and myocardial thickness. A description of any LV wall motion abnormality should be reviewed. Perhaps the most important information is a description of the

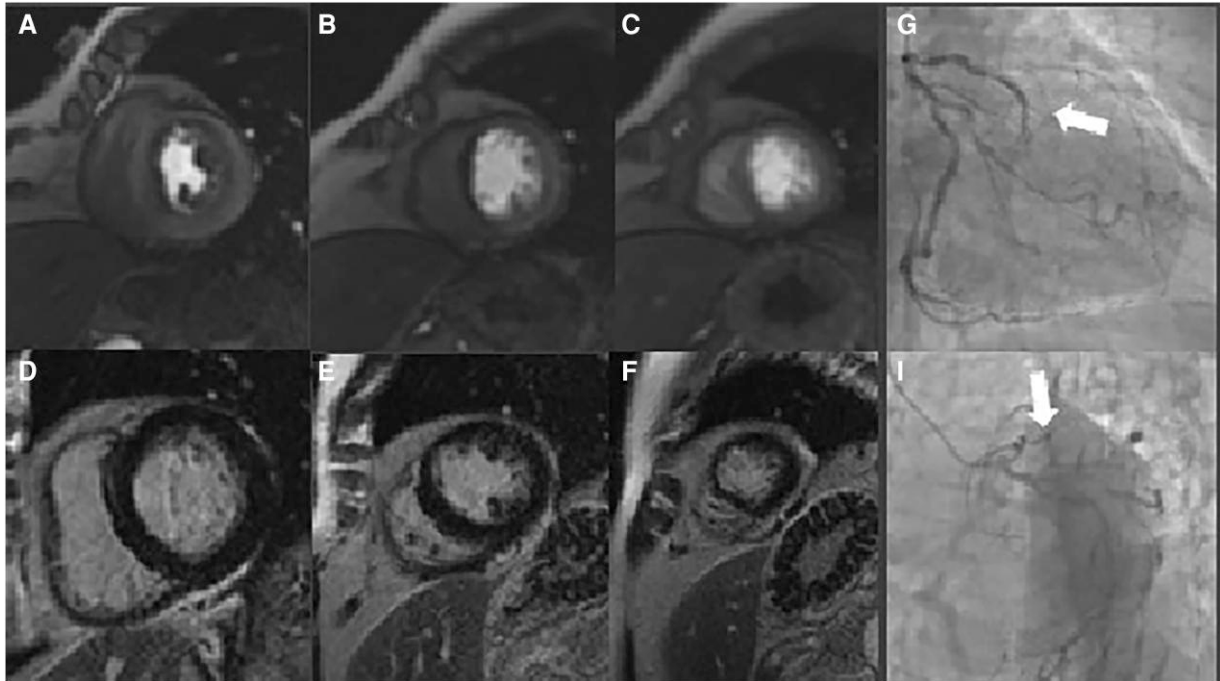


Figure 9 Stress and rest perfusion images of case demonstrating ischaemia. Panels A–C show an inducible perfusion defect of the mid anteroseptal and inferoseptal segments as well as a superimposed perfusion defect at the apical septal and anterior segments. D–F shows late subendocardial (26%–50%) enhancement of the part of apical septal segment that extends to part of the apical anterior segment. G and I show chronic total occlusion of LAD and RCA with retrograde filling from LCx on invasive coronary angiogram. The proximal OM1 shows severe stenosis.

presence and pattern of LGE. In patients with infarct pattern LGE, a final diagnosis of MINOCA can be made (assuming that this is not perceived to be chronic), whilst other patterns may suggest alternative diagnoses such as myocarditis. Myocarditis will be discussed further below in the non-ischaemic cardiomyopathy section. The pattern of myocardial oedema should also be noted, e.g. patchy/epicardial in myocarditis, regional with a specific coronary artery territory in AMI.

III. Chronic coronary syndrome

The 2024 ESC chronic coronary syndrome guidelines advise the use of CMR to evaluate global and regional function, LGE imaging for assessing myocardial scar, viability and the extent of transmural extent in post-infarction patients. The ESC guidelines also advocated the use of stress CMR to diagnose and quantify myocardial ischaemia and/or scar and to estimate the risk of major adverse cardiovascular events (MACE) in individuals with suspected chronic coronary syndrome and moderate or high (>15%–85%) pre-test likelihood of obstructive coronary artery disease (CAD).⁴⁰ The 2022 recommendations of the European Association of Cardiovascular Imaging and the American Society of Echocardiography for the use of non-invasive imaging in coronary syndromes endorsed the use of stress CMR to detect myocardial ischaemia and guide clinical decision-making in patients with high intermediate pre-test clinical likelihood of chronic coronary syndromes.⁴¹

- How can stress CMR detect myocardial ischaemia?

Stress CMR involves inducing coronary hyperaemia with the use of vasodilators and intravenously injecting a GBCA to detect any areas of reduced blood flow during T1-weighted first-pass perfusion imaging. An inducible perfusion defect appears as an unmatched hypointensity with a coronary distribution pattern that is most dense in the subendocardium and then gradually fades towards the epicardium.⁴² True perfusion defects are usually >2 pixels wide, identifiable at stress but not at rest and persist for several heartbeats beyond peak myocardial enhancement. For accurate post-processing and interpretation of stress CMR, perfusion, and corresponding LGE images must be displayed side-by-side to differentiate ischaemic, infarcted, or non-ischaemic regions.⁴² (Figure 10).

- What role does stress CMR have in prognosis and risk stratification?

Stress CMR can accurately identify the presence of functionally significant CAD, detect coronary microvascular dysfunction, evaluate for myocardial viability, provide risk stratification, and deliver robust cardiac prognostication to guide clinical decision-making.⁴³ Revascularization strategy guided by stress CMR demonstrate similar results to that guided by invasive measurements of fractional flow reserve and can be particularly helpful in clinically stable patients with a moderate-to-high pre-test probability of ischaemic heart disease. Use of stress CMR is indeed associated with fewer referrals for downstream ICA when compared with other modalities, and lower rates of unnecessary

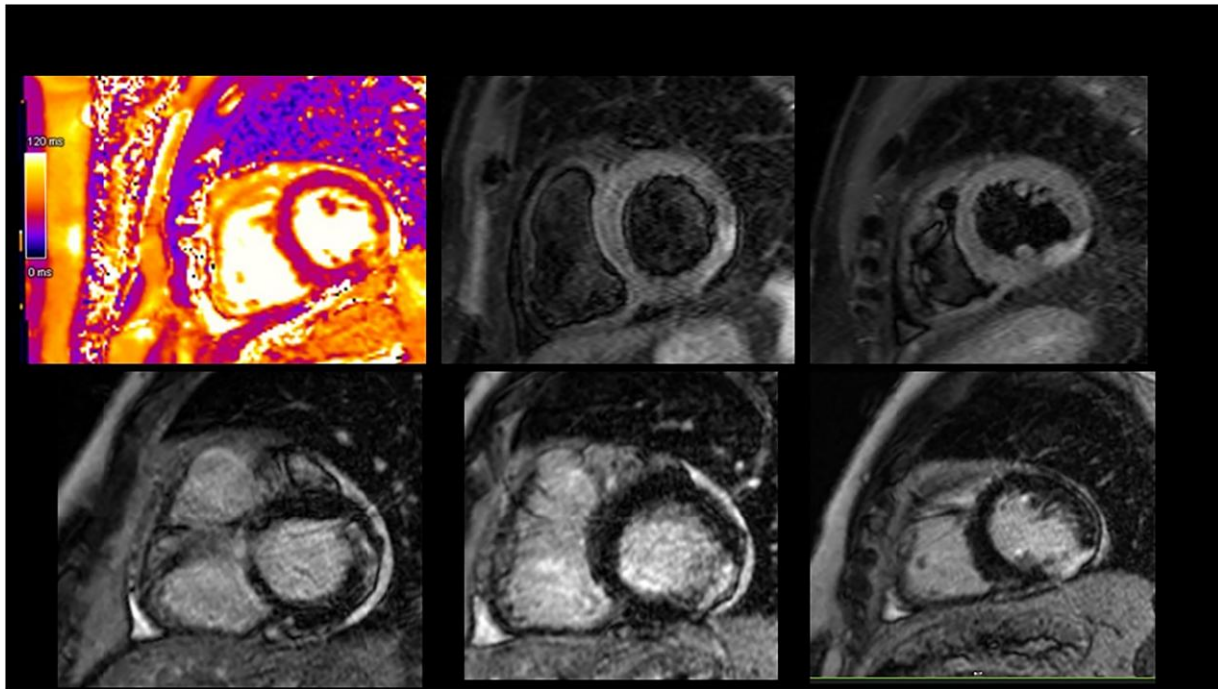


Figure 10 Cardiac MRI features of myocarditis. From left to right, the upper panel shows increased relaxation time of the mid inferior LV segment and T2-weighted STIR images reveal bright signals (hyperintensity) of the basal inferior and inferolateral as well as mid inferior segments. The lower panel shows epicardial late enhancement in the basal antero-lateral and inferolateral, mid inferolateral and inferior and apical inferior segments

stent implantation without apparent impact on subsequent risk of death and myocardial infarction.⁴⁴ Overall, a negative stress CMR indicates very low risk of future cardiovascular events with a warranty period exceeding 3 years.⁴⁵

- How are stress CMR and viability scans performed?

Stress CMR is more commonly performed using vasodilator agents such as adenosine or adenosine triphosphate (140 $\mu\text{g}/\text{kg}/\text{min}$ for 2–4 min, or up to 175–210 $\mu\text{g}/\text{kg}/\text{min}$), dipyridamole (0.14 mg/kg/min over 4–6 min; total dose 0.56–0.84 mg/kg) or regadenoson (400 μg single intravenous injection over 10 s) to induce coronary hyperaemia.⁴⁶ Automated techniques for absolute quantitative analysis of myocardial blood flow are becoming increasingly available in high-volume tertiary centres.⁴² This is increasingly important for patients with multivessel CAD for the assessment of regional ischaemia to guide revascularization strategies and for those patients with no obstructive CAD and risk factors for microvascular disease.^{47,48} Inotropic stress with dobutamine coupled with wall motion analysis can also be performed with good accuracy to assess ischaemia (typical maximum dose 40 $\mu\text{g}/\text{kg}/\text{min}$ $\pm$ atropine: 0.25 mg fractions up to 2 mg). The fundamental method for assessing myocardial viability is LGE. LGE detects non-viable myocardium and infers irreversible myocardial damage from the extent of transmural enhancement, which inversely relates to the likelihood of functional improvement after revascularization, both in acute and chronic settings. Notably, this also applies to markedly thinned (<5 mm) and akinetic

segments. Whilst LGE delivers high specificity for predicting absence of functional recovery, sensitivity may be limited in the presence of myocardial oedema and in LGE with an intermediate degree of transmural (25%–75%), where low-dose dobutamine (up to 10–20 $\mu\text{g}/\text{kg}/\text{min}$) exploring contractile reserve or metabolic imaging may be complementary.

- How to know from the report that the patient responded to the stressor agent?

Inadequate response to pharmacological stress may fail to unmask inducible perfusion defects and lead to a false-negative interpretation of the perfusion study. Heart rate increase >15 bpm and blood pressure drop between rest and stress conditions are poor surrogate markers of hyperaemic response. Splenic switch-off, defined as a reduction in splenic signal intensity during stress due to adenosine-induced splenic vasoconstriction, is a useful sign to assess hyperaemic response. This can be assessed either visually after contrast administration or quantitatively by splenic T1 mapping interrogation before gadolinium first-pass perfusion.⁴⁹ Peak stress myocardial blood flow by perfusion mapping can be alternatively used to confirm adequate stress, and when available, should be the preferred method.⁵⁰ However, in the absence of caffeine consumption, whether inadequate hyperaemic response to adenosine should be routinely interpreted as an indicator of an under-stressed and inconclusive study rather than a reflection of underlying coronary microvascular dysfunction remains to be elucidated.⁵¹

- What to look out for in the CMR report?

The report should determine the functional significance of CAD and provide insight as to whether the myocardium will recover after revascularization. When reviewing a report, the treating cardiologist should note:

- An increase in ventricular volumes and/or a reduced ejection fraction as this may indicate a long-standing and possibly less reversible recovery of function.
- Regional wall motion abnormalities should be mapped to the corresponding coronary artery territory and correlated with LGE images to determine viability- hibernating myocardium (no LGE) or scarred myocardium (LGE present).
- The presence of any inducible ischaemia, and the corresponding coronary artery territory.
- The presence and extent of myocardial infarction, the degree of transmural is an important indicator of viability. The CMR report should clearly state which segments are affected and whether transmural <50% (likely to be viable and recover after revascularization) or >50% (scarred myocardium that is unlikely to benefit from revascularization.^{23,52} The report should clearly state the coronary artery territory affected.
- Comparison of stress CMR with nuclear and stress echocardiogram

Single-photon emission computed tomography (SPECT) using vasodilator stress has a large amount of accumulated data supporting its diagnostic and prognostic value; it is widely available and covers the whole heart. However, CMR has a higher spatial resolution, no exposure to ionizing radiation and allows a more accurate multiparametric assessment. The CE-MARC study⁵³ showed higher sensitivity (86.3% vs 66.5%, $P < .001$) and similar specificity (83.2% vs 82.6%, $P = .916$) of CMR compared with SPECT, while in the MR-IMPACT II trial⁵⁴ CMR was non-inferior with a higher sensitivity (75% vs 59%, $P = .03$) but lower specificity (59% vs 72%, $P = .03$). The CE-MARC 2 study showed that a CMR- and a SPECT-oriented strategy similarly reduced the number of ICA in patients with suspected angina with no difference in MACE.⁵⁵ The PACIFIC 2 study demonstrated no significant difference in the accuracy for diagnosing fractional flow reserve-defined significant CAD in patients with prior percutaneous coronary intervention and/or MI, but the difference in sensitivity was 81% for positron emission tomography (PET), 67% for SPECT and 66% for CMR.⁵⁶ PET is the imaging modality most validated for the assessment of microvascular disease.⁵⁷

Dobutamine stress echocardiography has the advantage of continuous image acquisition, close communication with the patient and reliable ECG monitoring. Nevertheless, the patient's acoustic window and slice reproducibility can be a limitation. Nagel *et al.*⁵⁸ found a higher sensitivity and specificity for dobutamine CMR (86% vs 74% and 86% vs 70%, respectively, both $P < .05$) using invasive angiography as the reference standard.

IV. Limitations

For stress CMR, patient monitoring is limited by distance from the staff and magneto-electric effects that alter ECG tracings. Whilst quantitative analysis can help avoid the subjective interpretation

of perfusion images and facilitate the diagnosis of multivessel ischaemia, it is yet to be widely established. Likewise, CMR with physical exercise is only performed in a few specialized centres.

Myocardial LGE transmural is a strong predictor of functional recovery after revascularization; however, in the acute phase of an AMI, it is known to overestimate infarct size. Also, its predictive value is reduced in segments with an intermediate transmural is, and additional dobutamine testing may be useful.

Specific contraindications for stress CMR with a vasodilator agent include severe chronic obstructive pulmonary disease/bronchial asthma with significant wheezing, 2nd or 3rd-degree atrioventricular block or sinus node dysfunction in the absence of a permanent pacemaker, systolic blood pressure <90 mmHg or >200 mmHg at rest, recent acute coronary syndrome (<1 week), and haemodynamically significant LV outflow obstruction.⁵⁹ Patients must be able to lie flat and hold their breath for 10–15 s. Caffeine and methylxanthine intake must be avoided for 12–24 h before a vasodilator stress test, as it can cause false-negative results. Patients do need to fast. Dobutamine stress CMR should not be performed in patients with serious hypertension (>220/120 mmHg), in patients with acute coronary syndrome, haemodynamically significant LV outflow obstruction and narrow-angle glaucoma, and complex cardiac arrhythmias, including uncontrolled atrial fibrillation, and in patients with myocarditis, endocarditis, or pericarditis.⁵⁹

Key points

- CMR provides information on systolic function and the physiologic significance of CAD.
- CMR is an excellent technique for evaluating acute and chronic myocardial infarction.
- T2-weighted sequences assess the presence of myocardial oedema and are used to estimate the area-at-risk. T2* detect the presence of haemorrhagic infarctions.
- The degree and extent of infarct transmural is an excellent predictor of functional recovery, ventricular remodelling and adverse prognosis at follow-up.
- LGE sequences also allow the evaluation of MVO, which is linked to a higher rate of LV adverse remodelling and MACE.

Non-ischaemic cardiomyopathies

CMR has become an essential tool for the evaluation of known or suspected cardiomyopathies (CMPs).⁶⁰ CMR provides non-invasive characterization of myocardial tissue. The use of LGE and mapping sequences enables the early detection and precise phenotyping of different CMPs to guide appropriate management and aid in patient risk stratification.

Sequences used and scanning protocol:

- Cine sequence to assess cardiac function, volumes, wall thickness, mass, and morphology;
- T2 weighted sequences (STIR and T2 mapping) to detect myocardial oedema;
- Pre- and post-contrast T1 mapping to detect interstitial fibrosis and infiltrative processes;
- LGE to detect myocardial fibrosis;

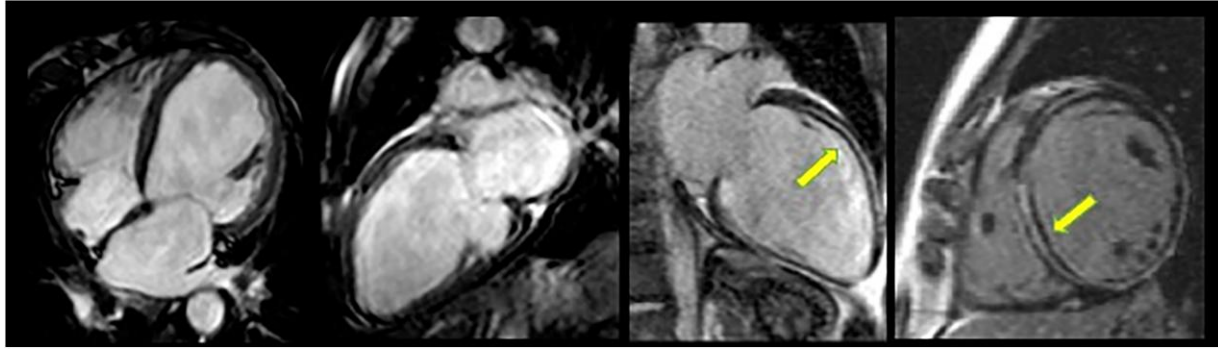


Figure 11 Cardiac MRI features of DCM. The upper panel shows CMR cine images in the 4 and 2 chamber orientation demonstrating biventricular dilatation and systolic dysfunction. The lower panel shows midwall late gadolinium enhancement of the anterior wall and septum

- $\pm T2^*$ sequence before giving GBCAs to detect iron overload.
- $\pm EGE$ for thrombi

In this section, we review the indications, prognosis, and risk stratification for the different CMPs.

I. Myocarditis

CMR is the first-line diagnostic tool for the evaluation of myocarditis in its diagnosis by offering 90% accuracy non-invasive through multiparametric tissue characterization.^{61,62} Follow-up CMR can assess whether the inflammation activity has resolved or has progressed to a dilated cardiomyopathy (DCM).^{63,64} It facilitates risk stratification and often replaces the need for biopsy.⁶⁵

- How CMR can detect/assess?

CMR is widely accepted as the preferred imaging tool for clinical suspicion of acute myocarditis.^{66,67} T2 STIR and T2 mapping sequences allow the non-invasive detection of myocardial oedema, a marker of inflammation in the acute/subacute phase (Figure 11).^{66,68,69} Native T1 mapping provides 89% diagnostic accuracy in patients hospitalized for acute myocarditis studied within 14 days of symptom onset,⁷⁰ although this drops to 45% if patients are imaged >14 days after clinical presentation.⁷¹ The detection of irreversible damage is made by LGE imaging, in which a high signal intensity area represents myocardial necrosis in the acute setting and fibrosis once it has become chronic. The LGE pattern is different from that due to ischaemic injury, which usually includes subendocardial layers, whereas myocarditis involves the subepicardium and/or the mid-wall.

- What role does CMR have in prognosis/risk stratification?

Myocardial oedema in the absence of a high signal intensity area on LGE imaging represents reversible myocardial injury and thus can predict functional recovery. LGE without oedema at 6-month follow-up is associated with worse prognosis, particularly with a midwall septal distribution.⁷² In a 10-year follow-up study of 183 patients with biopsy-proven myocarditis, the presence of midwall LGE in the antero-septal segments was

associated with a higher rate of mortality when compared with those with no LGE or other LGE patterns.⁷³

- What clinical questions/information should be in the referral?

The clinical presentation of myocarditis varies markedly, but a brief clinical history should be provided. The referral should specify a recent viral illness, fever or troponin rise and that they want to differentiate if this is due to inflammation or ischaemic injury.

- What to look out for in the CMR report?

The CMR report for the diagnosis of myocarditis should be based upon the Revised Lake Louise Criteria, where the scan requires the demonstration of both myocardial oedema (in the form of increased myocardial T2 relaxation time or increased T2 signal intensity) and myocardial injury (in the form of increased native T1, increased extracellular volume or presence of LGE).⁷⁴ If LGE is present, the pattern of distribution, midwall, and/or subepicardial, should be reported.

We advise a follow-up scan at 3 months to identify persistent high-risk LGE extent or patterns and residual inflammation.⁶⁴ Patients with persistently elevated T2 values and LV dysfunction at follow-up may still have ongoing myocardial inflammation. This plays a crucial role in the decision-making regarding the resumption of competitive sports.⁷⁵ Recently, a study showed that 8% of patients presenting with acute myocarditis had a 'concealed' inherited CMP prior to the myocarditis episode.⁷⁶

II. Dilated cardiomyopathy

Idiopathic DCM is the presence of left or biventricular dilatation with severely impaired systolic function in the absence of abnormal loading conditions (i.e. hypertension, valve disease, etc.) or IHD sufficient to cause global systolic impairment.

- How CMR can detect/assess?

CMR imaging is used to confirm the diagnosis of DCM and determine the aetiology through tissue characterization. DCM patients with advanced LV dysfunction may have diffuse

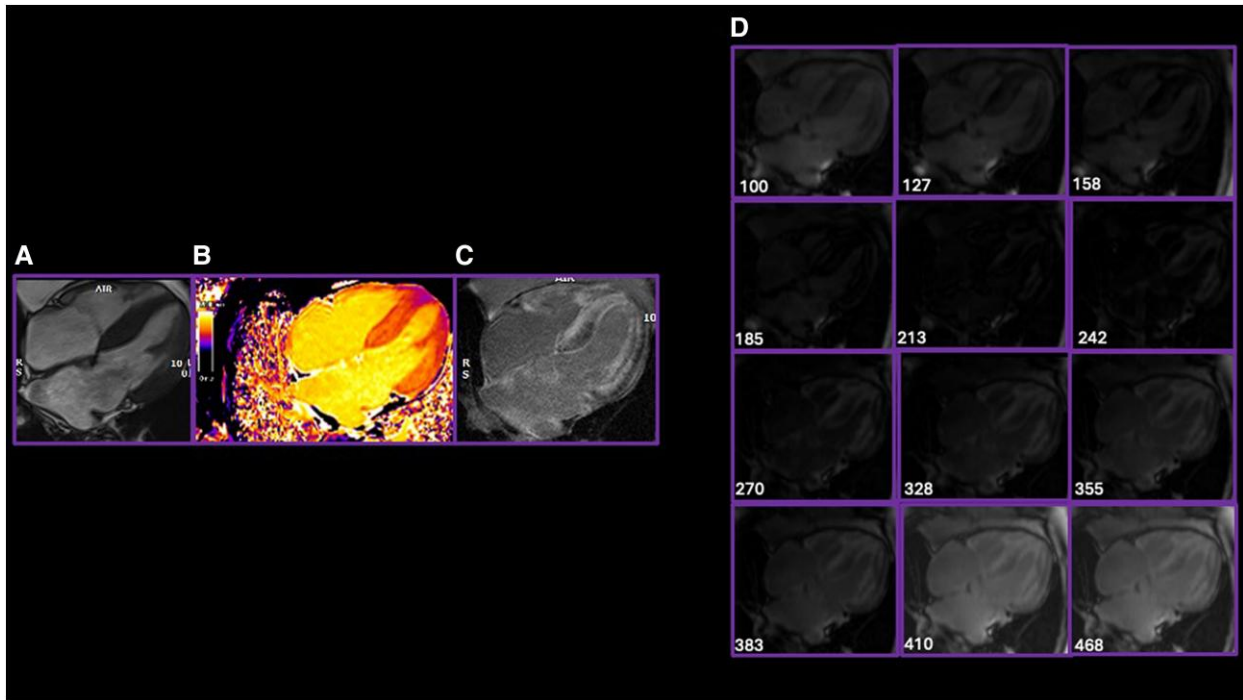


Figure 12 Cardiac MRI features of cardiac amyloidosis: (A) cine image in the 4-chamber orientation shows increased predominantly septal wall thickness, normal indexed LV volumes and severely dilated left atrium, (B) prolonged native T1 times for the field strength (native myocardial T1 1414 ± 72 ms) and (C) late gadolinium enhancement images show quick washout of gadolinium with diffuse subendocardial enhancement. There is enhancement of the subendocardium in the RV and of the left atrial walls. (D) The T1 scout images of the cardiac amyloidosis show the null point for myocardium is reached before the blood pool is nulled, indicating that the T1 (in ms) of myocardium is shorter than that of blood

myocardial wall thinning with a diastolic wall thickness of <5.5 mm. Previously, some cases were diagnosed as LV non-compaction cardiomyopathy, but the most recent ESC Cardiomyopathy Guidelines recommend the term 'hypertrabeculation' instead.⁶⁰ Hypertrabeculation is seen as a phenotype that can occur with other developmental abnormalities, ventricular hypertrophy, dilatation, and/or systolic dysfunction or as a normal variant. Excessive trabeculation may be a normal physiological response to an increased preload or afterload, such as pregnancy or athletic participation.^{77,78}

T2-weighted sequences are often used to exclude myocarditis. LGE aids in identifying the aetiology by its pattern of distribution: non-infarct (midwall, most common in DCM, or subepicardial) or infarct (subendocardial or transmural as observed in ischaemic cardiomyopathy) pattern (Figure 12). The use of gadolinium also helps to detect thrombus with EGE and LGE images.

- What role does CMR have in prognosis/risk stratification?

Midwall fibrosis on LGE imaging is present in 30%–40% of DCM patients and is associated with increased risk of all-cause mortality, hospital admission for cardiovascular causes, and sudden cardiac death (SCD) or ventricular tachycardia.⁷⁹ The presence of myocardial fibrosis was associated with a higher risk of all-cause mortality, cardiovascular mortality and cardiac transplantation, even after adjusting for LVEF

and other traditional risk factors.^{79–82} In patients with DCM⁸³ who survive longer than 1 year, demonstrate good functional status and have an LVEF $\leq 35\%$ after at least 3 months of optimal medical therapy, an ICD is advised to reduce the risk of sudden death and all-cause mortality.⁸⁴ However, the majority of these patients will never use their ICD, so there is considerable interest in whether myocardial fibrosis on CMR might improve patient selection for ICD. The presence of LGE predicted appropriate ICD shock in patients with primary prevention criteria,⁸³ with the BRITISH randomized controlled trial set to answer this question (ClinicalTrials.gov NCT05568069).

In neuromuscular dystrophies, such as Duchenne and Becker muscular dystrophies, the extent of LGE is strongly linked to predicting subsequent decline in LVEF and the development of heart failure.⁸⁵

- What clinical questions/information should be in the referral?

The most common question is: what is the aetiology of the heart failure/CMP? Any information about the clinical history and presentation (including family history, list of medications, substance abuse history) and previous investigations should be provided as additional sequences may be required (e.g. STIR and T2 mapping in suspected myocarditis, T2* in suspected iron overload).

- What to look out for in the CMR report?

When reviewing a CMR report for suspected DCM, the primary aim is to confirm LV dilatation and impaired function while ruling out other aetiologies and looking for prognostic indicators. The presence of a reduced LVEF, increased LV volumes, and global hypokinesia are common findings. There is often bi-ventricular involvement with RV dilatation and impaired contractility. As previously mentioned, the presence of myocardial oedema suggests myocarditis which can evolve to DCM. If LGE is present, the pattern of LGE should be described as this aids in identifying the aetiology. Midwall fibrosis is found in 30%–40% of idiopathic DCM. A subepicardial ring-like pattern is often associated with genetic conditions that are associated with greater arrhythmic risks like Lamin A/C, phospholamban and filamin C.⁶⁰ The presence of intracardiac thrombi indicates sluggish blood flow, while pleural effusion indicates congestion and should prompt management accordingly.

III. Hypertrophic cardiomyopathy

A CMR referral is warranted in assessing patients with known/suspected hypertrophic cardiomyopathy (HCM) when:

- Echocardiogram is inconclusive
- To differentiate HCM from other causes of hypertrophy and phenocopies
- Risk stratification for SCD
- Planning for myectomy
- Serial assessment to monitor disease progression
- Screening of first-degree relatives to detect an early phenotype.^{86,87}
- How CMR can detect/assess?

CMR's sharp contrast and high spatial resolution are superior to TTE in detecting focal increased LV wall thickness, especially of the anterolateral, inferoseptal and apical segments, and to display abnormal mitral valve apparatus (i.e. elongated mitral valve leaflets, apically displaced papillary muscles, accessory muscle band, etc.) (see [Movies S1](#) and [S2](#)).^{42,46,84,86–89} The use of parametric mapping and gadolinium enhancement sequences also provides clues for alternate diagnoses/phenocopies (e.g. amyloidosis, Fabry disease, or hypertensive heart disease).^{42,46,84,86,87}

- What role does CMR have in prognosis/risk stratification?

Risk stratification of HCM patients is an integral part of clinical management. For risk stratification, CMR accurately assesses LV function and mass, and tracks disease progression. Although the higher blood pool to myocardium contrast favours CMR in comparison to TTE, intermodal, as well as inter-reader discrepancies in assessing maximal LV wall thickness, suggest a cautionary use of this parameter for risk stratification.⁹⁰ CMR detects and quantifies LGE, which correlates with overall outcome, and refines SCD risk stratification to guide ICD implantation when risk stratification is otherwise uncertain.^{87,88} Extensive LGE 15% indicates an increased risk for SCD.^{91,92} CMR detects LV apical aneurysms more effectively than TTE⁹³ with important prognostic implications.⁹⁴ Finally, CMR provides more precise

delineation of LV wall thickness and hypertrophy, and is being used increasingly in screening evaluations.

- What clinical questions/information should be in the referral?

A summary of the clinical history, examination and echocardiographic findings should be available to the CMR specialist. A history of hypertension, any findings suggestive of non-sarcomeric HCM or a positive family history should also be mentioned.

- What to look out for in the CMR report?

CMR allows accurate assessment of LV wall thickness and the location of the hypertrophied segment(s). The clinical diagnosis of HCM is highly dependent on the accurate measurement of an LV wall thickness ≥ 15 mm in any myocardial segment that is not explained solely by loading conditions.⁶⁰ Lesser degrees of wall thickening (13–14 mm) require additional clinical findings (e.g. family history, genetic findings, and ECG abnormalities). High-risk features as an increased wall thickness ≥ 30 mm and apical aneurysms, should be highlighted in the report, as some patients may only be identified by CMR. In hypertrophic obstructive cardiomyopathy, the report should also describe the systolic anterior motion of the mitral valve and any papillary muscle abnormalities. If LGE is present, the treating cardiologist should also note the pattern and extent of LGE to differentiate HCM from other aetiologies and for risk stratification.

In the screening of family members, the measurement of LV maximal wall thickness as well as elongated mitral valve leaflets, myocardial crypts, expanded extracellular space (with ECV mapping), and extent of LGE should be reported. Morphological features that differentiate HCM from other aetiologies of LV hypertrophy (LVH) are described in the following sections.

IV. Cardiac amyloidosis

CMR is an ancillary imaging modality that can help in the diagnosis of cardiac amyloidosis (CA), especially when echocardiographic findings are equivocal and clinical suspicion for cardiac/extracardiac involvement remains high.⁹⁵ CMR cannot distinguish between AL and ATTR amyloid, which is important given the different treatment strategies for these two conditions.

- How CMR can detect/assess?

Characteristic CMR findings of CA include ([Figure 12](#)):

- native T1 values of the myocardium are usually significantly elevated in CA, often 100–150 ms higher, than the mild elevation of HCM)^{96,97};
- TI scouts for the typical gadolinium kinetics featuring an abnormal myocardial signal suppression pattern;
- diffuse subendocardial or transmural LGE, or bilateral septal subendocardial LGE with dark mid-wall (zebra pattern) with relative sparing of apical segments;
- significant expansion of the extracellular matrix—ECV $\geq 40\%$ ^{98,99};
- atrial myopathy by LGE and strain analysis;
- intracardiac thrombosis;
- visceral amyloid burden by ECV mapping.¹⁰⁰

Many patients with early ATTR or patients with AL do not have the characteristic LGE for CA.¹⁰¹ Therefore, obtaining ECV is of importance when there is a clinical high suspicion for CA but no typical LGE. In these cases, we advise having a low threshold to proceed with nuclear imaging or biopsy.

- What role does it have in prognosis/risk stratification?

Native T1 mapping and LGE demonstrated comparable diagnostic performance, but only LGE is an independent predictor of mortality in CA.^{96,102} ECV mapping may have better diagnostic and prognostic performance than that of LGE,¹⁰² and also provides a possible biomarker to track treatment response.¹⁰³

Prolonged myocardial T2 time is also a marker of treatment response and an independent predictor of mortality in AL amyloidosis, regardless of ECV and serum biomarkers, but no correlation has been observed in ATTR.^{104,105}

- What clinical questions/information should be in the referral?

Major reasons for CMR referral from the 2023 ESC Guidelines for the management of CMPs include⁶⁰:

- **Identification of cardiac involvement in asymptomatic TTR gene carriers**, patients with biopsy-proven systemic AL amyloidosis and abnormal levels of cardiac biomarkers, or monoclonal gammopathy of undetermined significance with abnormal free light chain (FLC) levels and abnormal levels of cardiac biomarkers.
- **Screening for cardiac amyloidosis in patients with new symptomatic heart failure** AND elevated FLC levels, unexplained increased LV wall thickness, low-flow low-gradient aortic stenosis, unexplained peripheral sensorimotor neuropathy, known or suspected familial amyloidosis, or monoclonal gammopathy including multiple myeloma.
- **Evaluation of biopsy-proven AL or ATTR cardiac amyloidosis** aimed at quantification of amyloid burden and assessing response to therapy/disease progression.

- What to look out for in the CMR report?

The hallmark features of CA include high native T1 values and ECV, a diffuse LGE in the form of subendocardial, transmural, or bilateral septal subendocardial with dark mid-wall (zebra pattern) and atrial enhancement as well as abnormal gadolinium kinetics commonly described as a 'failure to null'. Structural and functional changes in CA include concentric LVH, biatrial dilatation, atrial septal thickening, and a reduced stroke volume.^{95,106}

V. Fabry cardiomyopathy

CMR enables accurate differential diagnosis of LVH and provides a precise assessment of morphological, functional, and tissue characterization of cardiac involvement in Fabry disease. CMR also has a key role in the serial monitoring after enzyme replacement therapy and risk stratification.

- How CMR can detect/assess?

In Fabry cardiomyopathy, T1 mapping is useful to detect early cardiac involvement in the accumulation phase, yet progressive

pseudonormalization of T1 relaxation times occurs in subsequent stages, which are dominated by inflammation, hypertrophic response, and fibrotic changes, confounding its prognostic utility.¹⁰⁷ LGE is usually identified at a later stage of the disease, usually located in the basal inferolateral segment of the LV and with mid-wall distribution, yet different patterns and earlier occurrences can occur.

- What role does it have in prognosis/risk stratification?

Whilst detection of increased myocardial T2 values reveals an inflammatory component accompanying myocardial damage, T2 normalization was found to correlate with LV mass reduction after treatment and therefore measurement of T2 relaxation time has been proposed for monitoring therapy response.¹⁰⁷⁻¹⁰⁹ Native T1 mapping dispersion, defined as the standard deviation of per voxel T1 times, has been recently described as a parameter able to reflect sphingolipid accumulation, fibrosis and inflammation, and included into a validated prognostic index of 5-year risk estimation of adverse cardiac outcome for individual patients with Fabry disease.¹¹⁰

- What clinical questions/information should be in the referral?

Fabry disease is a systemic disease associated with important diagnostic delays. If there is a clinical suspicion of Fabry disease in a patient with LVH, a brief history of the clinical findings suggestive of the systemic disease, family history, ECG, echocardiogram, and any other investigation should be provided by the referrer. If the referral is a follow-up scan after enzyme replacement therapy, this should also be mentioned. If the initial CMR scan was done elsewhere, this should be communicated to facilitate the transfer of images for comparison. It is also important to mention whether the patient has renal impairment and provide a recent eGFR.

- What to look out for in the CMR report?

For the initial diagnosis, the report should include:

- The presence and pattern of wall thickening and LVH
- Quantification of the LV mass
- T1 mapping with low T1 values is often the most critical finding to differentiate it from other causes of LVH
- Identification of the classical LGE pattern: midwall enhancement (fibrosis) at the basal inferolateral segment of the left ventricle

In serial monitoring after enzyme replacement therapy, it is important to look out for:

- Documentation of changes in LV thickness and mass. To avoid variability, the CMR specialist should remeasure wall thickness and mass on the serial scans to assess treatment response.
- Presence of inflammation
- Identification of LGE even in the presence of normal wall thickness.

VI. Cardiac sarcoidosis

The new clinical consensus statement for the management of cardiac sarcoidosis (CS): A clinical consensus statement of the

Heart Failure Association, the European Association of Cardiovascular Imaging, the ESC Working Group on Myocardial & Pericardial Diseases, and the European Heart Rhythm Association of the ESC summarizes the use of cardiovascular imaging, including CMR.¹¹¹ Patients with a histological diagnosis of extra-cardiac sarcoidosis may be referred to CMR to confirm a CS diagnosis.⁸⁵⁻⁸⁹

- How CMR can detect/assess?

The RV involvement in CS is frequent and can sometimes be mistaken for an arrhythmogenic cardiomyopathy (AC).¹¹² Typically, an LGE pattern is seen involving the basal LV segments and the RV side of the septum, but any segment of the myocardium can show a patchy, non-ischaemic distribution, sparing the endocardium.¹¹³ Associated wall thinning and aneurysms also contribute to diagnosis, especially in the late disease stages.^{42,46,111,114-117} Being CS a focal disease, CMR may also serve as a guide to endomyocardial biopsy, increasing its diagnostic yield.¹¹⁸

- What role does it have in prognosis/risk stratification?

An impaired LVEF has been described as an independent factor for poor prognosis.¹¹⁹⁻¹²¹ The extent of LGE is related to increased risk of ventricular arrhythmias, SCD and reduced steroid responsiveness.^{88,111,118} CMR may indeed be used to monitor response to treatment (i.e.: reduction of myocardial oedema on T2-weighted sequences, mirroring reduced myocardial inflammation),^{88,116-118} In this regard, combining metabolic activity (as an expression of inflammation) by PET to tissue characterization by CMR, has been shown not only to increase diagnostic accuracy but also to strengthen the prognostic value, as compared to either imaging test alone.¹²²

- What clinical questions/information should be in the referral?

Pertinent clinical information in the referral includes:

- confirmed histological diagnosis of extra-cardiac sarcoidosis
- unexplained cardiac presentation of advanced atrioventricular block, sustained ventricular tachycardia, and/or heart failure
- established CS and follow-up CMR to assess treatment response.
- presence of an implantable device (e.g. ICD, LINQ) should be mentioned in the referral.

- What to look out for in the CMR report?

When receiving a CMR report for diagnosis or serial assessment, it is important to review findings of tissue characterization as well as functional assessment.

- Evaluation of ventricular function and volumes;
- Assessment for regional wall motion abnormalities (RWMA), wall thinning and aneurysms;
- Active myocardial inflammation as evidenced by increased signal intensity of T2-weighted images and increased values on T2 mapping;

- A non-ischaemic LGE pattern is typically midwall or subepicardial, but the characteristic distribution of involving the RV side of the basal anteroseptum is highly specific for CS.¹¹³ (Figure 13).

It is critical to look out for assessment of significant prognostic factors as LV systolic dysfunction and basal septal thinning (late stage).

Serial CMR can be used to monitor CS patients' response to treatments. In patients who already have a CIED, the image quality and the amount of information that can be provided may be affected.

VII. Arrhythmogenic Cardiomyopathy

Diagnosis of AC is multi-parametric, with CMR holding a pivotal role for volumetric/functional assessment and tissue characterization, both keys to reaching a final diagnosis when clinical, ECG and echocardiographic findings suggest the disease.¹²³⁻¹²⁵

- How CMR can detect/assess?

CMR should provide the referring physician with disease confirmation and phenotyping (distinguishing right-dominant, biventricular and left-dominant AC,^{42,46,124,125} exclusion of phenocopies (i.e. sarcoidosis), accurate assessment of biventricular function, risk stratification and monitoring of disease progression.^{42,46,124-126}

The first Task Force criteria for ACM in 1994 and the revised criteria in 2010 both demonstrated good accuracy in diagnosing the RV phenotype, but lacked sensitivity for assessing LV involvement and did not include tissue characterization by CMR.^{123,127} The new European Task Force consensus in 2024 provides the diagnostic criteria for the LV phenotypic features and the use of CMR tissue characterization (Table 1).^{125,126} Fibrous or fibrofatty myocardial replacement is often challenging to interpret in the right ventricle, but represents key features of the disease. Where these are identified in the left ventricle on CMR sequences, they should prompt genetic testing to confirm a left-dominant AC phenotype (see Movie S3).^{124,125}

- What role does it have in prognosis/risk stratification?

In AC patients, CMR findings can aid in estimating a patient's risk for arrhythmic events and in identifying patients most likely to benefit from an ICD. An abnormal CMR has a worse outcome in right-dominant AC, irrespective of conventional prognostic markers, and the greater the number of abnormalities on CMR, the higher the SCD risk.^{88,128} Indeed, CMR features such as severe RV dilatation and dysfunction, RV and/or LV fat infiltration and presence of LGE are strong predictors of increased arrhythmic events and worse outcome.^{129,130}

- What clinical questions/information should be in the referral?

A summary of the clinical assessment, ECG and echocardiographic findings should be available to the CMR specialist. The referrer should also mention if the scan is requested for: clinical suspicion, disease progression or a positive family history of AC.

- What to look out for in the CMR report?

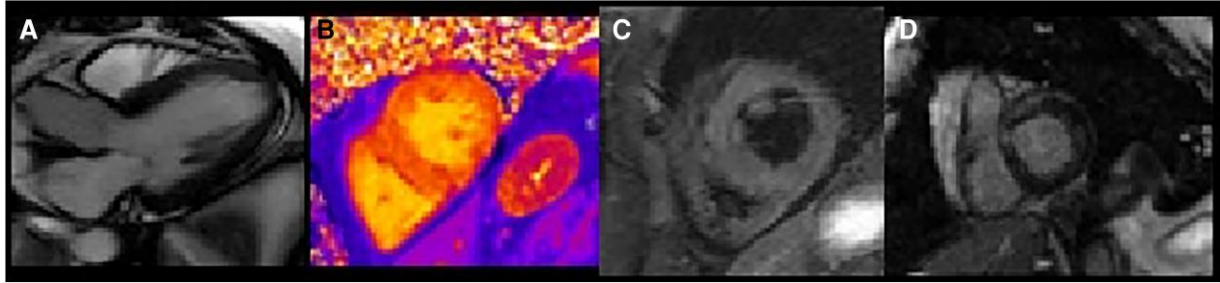


Figure 13 Cardiac MRI of a patient with inactive cardiac sarcoidosis shows (A) 3-chamber cine image with normal wall thickness a SAX, (B) T2 mapping and (C) STIR images show no evidence of myocardial oedema while (D) LGE images with fibrosis on right ventricular side of the basal anteroseptal segment extending into the subepicardium of the LV anterior walls from a previous acute inflammatory phase

When reviewing a CMR report for suspected AC, the report should include the major and minor criteria for the international criteria ('Padua criteria') as outlined in [Table 1](#) with particular attention to the presence of:

- RV and RV outflow tract dilatation and dysfunction, as well as LV involvement¹²⁵
- presence of any RWMA or RV aneurysm
- presence of fat or fibrosis and its site.

VIII. Iron overload cardiomyopathy

In the modern era, the management of iron overload with iron chelation therapy guided by CMR T2* relaxometry is key for the prevention and treatment of iron overload cardiomyopathy.¹³¹

- How CMR can detect/assess?

CMR T2* relaxometry is the method of choice for reliable quantification and monitoring of myocardial and liver iron overload. T2* values correlate with hepatic and myocardial iron content better than serum ferritin or liver iron concentration and closely predict indices of LV function. In patients with only mild increases of cardiac iron, native T1 mapping demonstrates better reproducibility, but this appears to be of less clinical relevance in advanced disease stages. For the measurement of myocardial T2* *in vivo*, a mid-ventricular short-axis slice is acquired, and a homogeneous region of interest restricted to the septum is defined, encompassing both subepicardial and subendocardial regions.^{46,132}

- What role does CMR have in patient risk stratification?

Monitoring LVEF is an important part of cardiac surveillance in patients with iron overload but is less than ideal for adequate prediction of heart failure events. In the clinical scenario of patients with or at high risk of iron overload cardiomyopathy, T2* mapping allows detection of pre-clinical disease and enables individually tailored chelation regimens to reduce the incident risk of heart failure.^{133,134} A T2* value of >20 ms at 1.5 T is normal, while a value of 10–20 ms and <10 ms indicates moderate and severe myocardial iron overload—those with the lowest T2*

values have the highest risk of developing arrhythmia and heart failure^{135–137} ([Figure 14](#)).

- What clinical questions/information should be in the referral?

Major reasons for CMR referral include:

- cardiac involvement in asymptomatic individuals with transfusion-dependent anaemias and primary haemochromatosis at risk of myocardial iron overload
- Monitoring of cardiac and liver response to chelation regimens by T2* relaxometry
- Differentiate iron overload cardiomyopathy from other potential causes of heart failure (CMPs, myocarditis from transfusion-transmitted hepatitis)

- What to look out for in the CMR report?

The CMR report for iron overload cardiomyopathy should be mainly focused on quantifying the myocardial iron overload to risk-stratify and guide chelation therapy. The report should include:

- T2* values of the myocardium and liver to assess iron accumulation. At 1.5 T, a myocardial T2* value measurement indicate: >20 ms normal, 10–20 ms moderate myocardial iron overload and <10 ms severe myocardial iron overload.^{135–137} Those with severe myocardial iron overload have the highest risk of developing arrhythmia and heart failure.
- Non-ischaemic pattern of LGE due to damage (scarring) from iron overload.¹³⁸
- Shorter values of native T1 mapping have been reported to be more sensitive indicator of iron accumulation.¹³⁹
- LV dilatation and impairment as well as RV dilatation and enlargement in later stages.¹⁴⁰

Indication of CMR in recent ESC guidelines

In the first ESC Cardiomyopathy Guidelines published in 2023, the authors highlighted the role of CMR findings to identify

Table 1 The morpho-functional ventricular and structural myocardial abnormalities ‘Padua criteria’ for diagnosis of arrhythmogenic cardiomyopathy¹²⁵

Category	Right ventricle (upgraded 2010 ITF diagnostic criteria)	left ventricle (new diagnostic criteria)
I. Morpho-functional ventricular abnormalities	By echocardiography, CMR or angiography: Major - Regional RV akinesia, dyskinesia, or bulging plus one of the following: - global RV dilatation (increase of RV EDV according to the imaging test specific nomograms) - global RV systolic dysfunction (reduction of RV EF according to the imaging test specific nomograms) Minor - Regional RV akinesia, dyskinesia or aneurysm of RV free wall	By echocardiography, CMR or angiography: Minor - Global LV systolic dysfunction (depression of LV EF or reduction of echocardiographic global longitudinal strain), with or without LV dilatation (increase of LV EDV according to the imaging test specific nomograms for age, sex, and BSA) Minor - Regional LV hypokinesia or akinesia of LV free wall, septum, or both
II. Structural myocardial abnormalities	By CE-CMR: Major - Transmural LGE (stria pattern) of ≥ 1 RV region(s) (inlet, outlet, and apex in 2 orthogonal views) By EMB (limited indications): Major - Fibrous replacement of the myocardium in ≥ 1 sample, with or without fatty tissue	By CE-CMR: Major LV LGE (stria pattern) of ≥ 1 Bull’s Eye segment(s) (in 2 orthogonal views) of the free wall (subepicardial or midmyocardial), septum, or both (excluding septal junctional LGE)

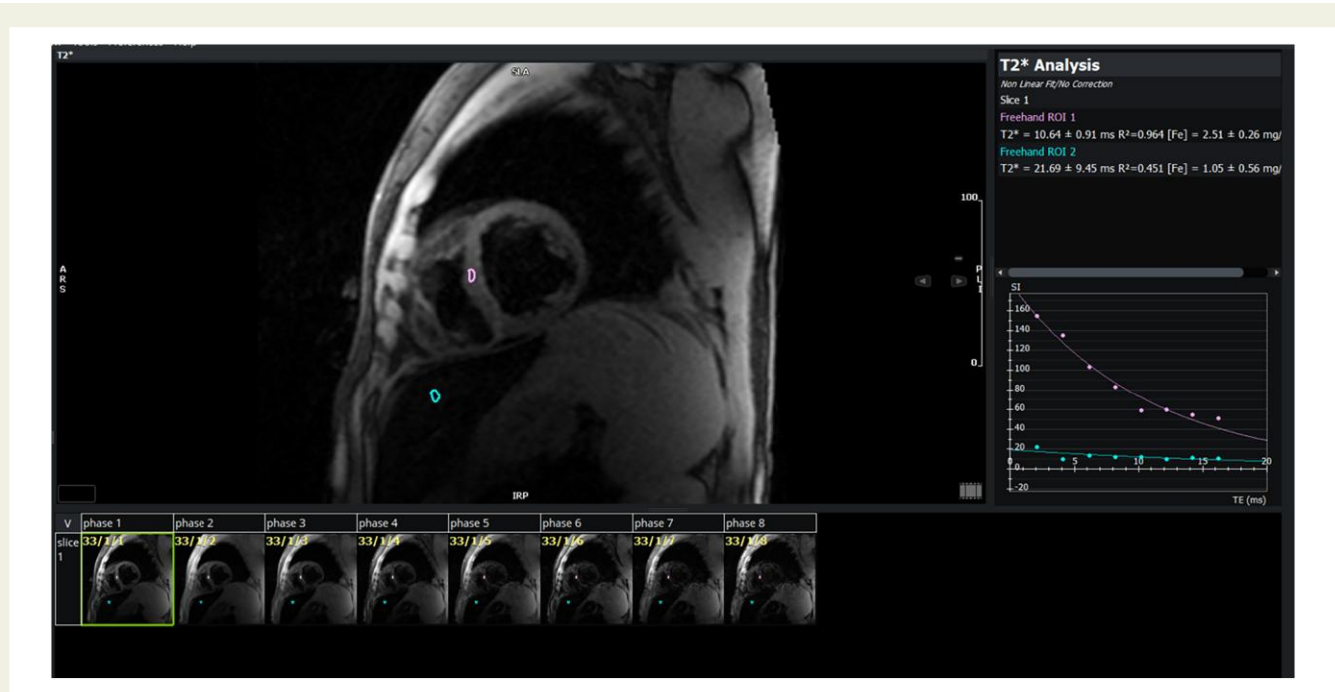


Figure 14 Cardiac MRI features of iron overload in the form of deviated T2* time with early decay of signals of liver and heart

the aetiology of known or suspected.⁶⁰ Serial assessment by CMR was recommended every 2–5 years, depending on initial severity and clinical course, disease progression and monitoring of therapy response.

Limitations

Limitations are similar to those previously described in the IHD section. As described later in this document, imaging artefacts can be caused by CIEDs and have been used to limit CMR imaging in the past. Currently, a number of solutions are available to mitigate these artefacts,

Key points

- CMR can aid in identifying the aetiology as this may have important therapeutic implications, particularly in infiltrative CMPs;
- CMR can identify important prognostic markers as replacement and interstitial fibrosis;
- Mapping sequences and LGE are important imaging biomarkers to monitor therapy and disease progression in conditions like amyloidosis and iron overload CMP.

Valvular heart disease

TTE is the first-line modality for the assessment of valvular heart disease (VHD) and often provides sufficient data for a complete evaluation. CMR is increasingly used to complement VHD assessment in cases of limited echocardiographic examination, or discordant clinical and echocardiographic findings¹⁴¹ as well as a secondary test to confirm the severity, particularly for regurgitant lesions. In addition, the 2025 ESC/EACTS Guidelines on the diagnosis and treatment of VHD recommend the use of CMR in order to precisely quantify ventricular and atrial size, assess systolic function, quantify regurgitant volume and fraction and tissue characterization abnormalities.¹⁴²

CMR enables comprehensive evaluation of valve morphology, ventricular and atrial size, ventricular function and hypertrophy, as well as ventricular inflow/outflow by the cine images bSSFP or fast spoiled gradient echo (FSPGR) sequences. Stenotic or regurgitant flow jets can be visualized with cine images in which the high velocity jet appears bright (high signal) and turbulent flow appears dark (low signal), creating a signal void due to dephasing of the moving protons in the blood. However, visual assessment alone is not sufficient to assess VHD severity. Phase-contrast velocity mapping is used to quantify regurgitant volume and fraction and to measure velocity along the course of a jet. Tissue characterization sequences are also used in the assessment of VHD. In this section, the focus will be on the aortic and mitral valves.

Aortic valve

- How CMR can detect/assess?

In aortic regurgitation (AR), CMR can provide the clinician with comprehensive information to evaluate the valve disease, including:

- aortic valve morphology

- mechanisms underlying the AR through the study of the aortic valve and ascending aorta anatomy by bSSFP sequences
- quantification of the regurgitant volume and fraction through phase contrast sequences
- the haemodynamic consequences of AR on cardiac volumes, systolic function and myocardial mass (Figure 15).

Formal cut-offs to define the severity of severe AR are yet to be defined. The CMR values of 26%–48% have been associated with severe AR defined by echocardiography.^{143–146} A commonly used threshold suggested to assess the severity of AR can be found in Table 2.^{141,147}

For the evaluation of aortic stenosis (AS), CMR allows the measurement of the valvular planimetric opening area by cine images and assessment of the LV hypertrophic remodelling response and its ultimate decompensation towards heart failure (Figure 16). However, CMR is not the method of choice to grade the severity of valve defects since planimetric aortic valve area assessment is not equivalent to those derived from the continuity equation and it does not allow a precise measurement of forward flows and gradients due to the low spatial and temporal resolution of phase contrast sequences compared with echocardiography.

Novel techniques such as 4D-flow-CMR are emerging, which may ultimately allow assessment of transvalvular flows of all cardiac valves with only one retrospective analysis, potentially enabling the measurement of blood flow at any level in a previously acquired 3D volume of the heart.¹⁴⁸

- What role does it have in prognosis/risk stratification?

Besides the ability of diagnostic assessment of AR, CMR allows a precise quantification of ventricular volumes, regurgitant volume and fraction, which have been directly correlated to the probability of symptoms worsening and future surgical indication in asymptomatic patients.¹⁴⁷ Indeed, some specific CMR parameters have also been associated with patients' outcome (i.e. combination of heart failure, hospitalization and cardiovascular death) as in the case of the holodiastolic retrograde flow in the descending aorta.¹⁴³

CMR tissue characterization sequences (i.e. T1 and ECV mapping and LGE) allow the detection of non-infarct myocardial fibrosis in patients with AS providing an objective marker of left ventricular decompensation that provides powerful patient risk stratification.¹⁴⁹ In the EVOLVED trial, asymptomatic patients with severe AS randomized to early intervention did not live longer than patients managed conservatively but did experience less AS-related hospital admissions and less symptoms at 1 year.¹⁵⁰ CMR can therefore be used to help identify patients who might benefit from earlier aortic valve intervention and can reveal patients who have concomitant CA, thus modifying patients' prognosis.

Mitral valve

- How CMR can detect/assess?

CMR is a 'second tool' to assess the severity of mitral regurgitation (MR) particularly when echocardiogram is inconclusive. The

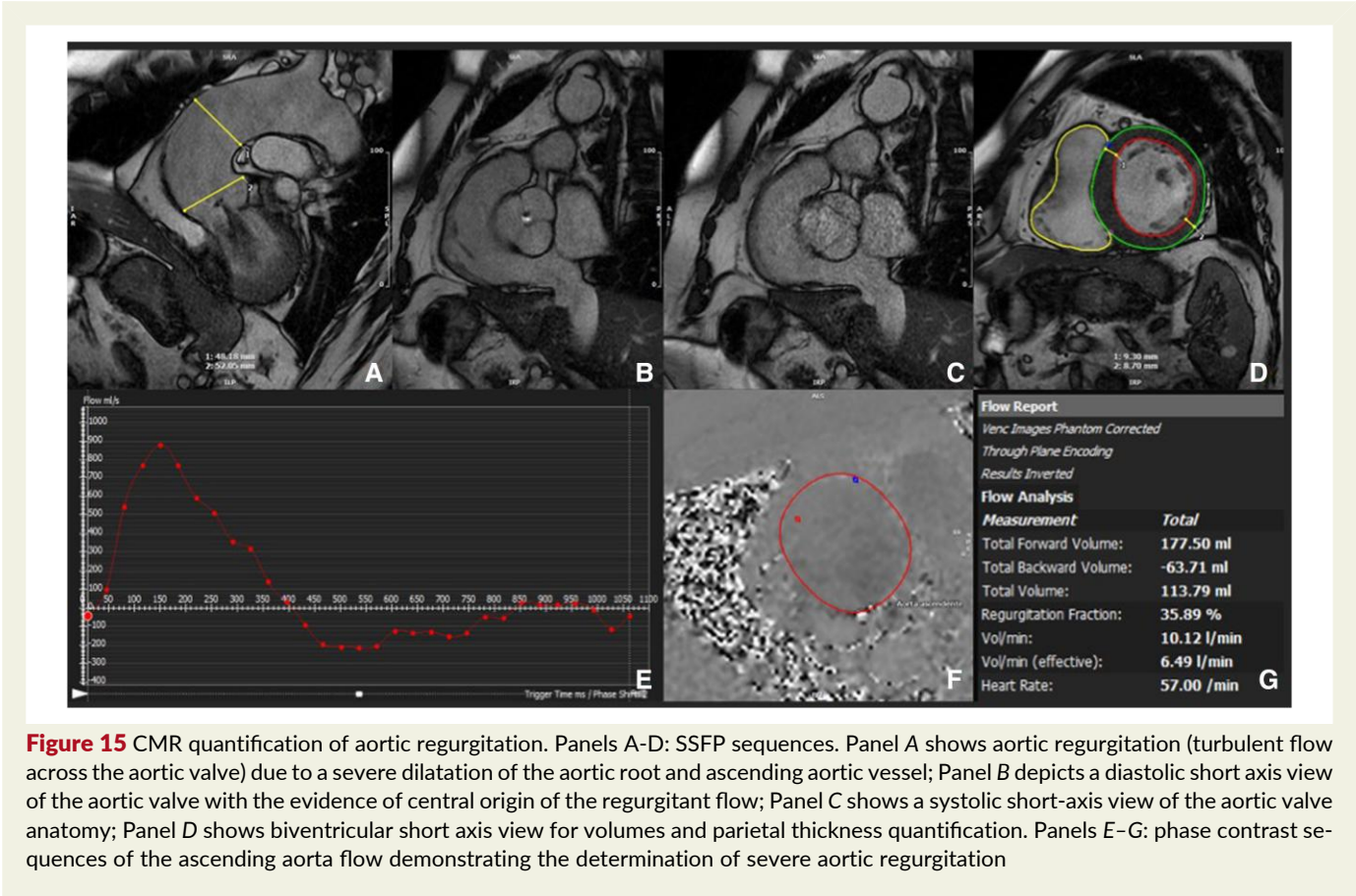


Figure 15 CMR quantification of aortic regurgitation. Panels A-D: SSFP sequences. Panel A shows aortic regurgitation (turbulent flow across the aortic valve) due to a severe dilatation of the aortic root and ascending aortic vessel; Panel B depicts a diastolic short axis view of the aortic valve with the evidence of central origin of the regurgitant flow; Panel C shows a systolic short-axis view of the aortic valve anatomy; Panel D shows biventricular short axis view for volumes and parietal thickness quantification. Panels E-G: phase contrast sequences of the ascending aorta flow demonstrating the determination of severe aortic regurgitation

Table 2 Suggested CMR-specific AR severity thresholds (141,147)

CMR grading of regurgitation severity			
	Mild	Moderate	Severe
AR	AR regurgitant fraction <30%	AR regurgitant fraction = 30%–35%	AR regurgitant fraction >35%

degree of regurgitation can be assessed with quantitative methods, which include direct and indirect methods to calculate the regurgitant volume. The direct methodology for MR is challenging to perform because the direct assessment of regurgitation across the valve is more difficult due to significant motion of the atrio-ventricular plane during systole in comparison to the semilunar valve. An indirect approach to calculate the atrio-ventricular valve regurgitant volume is calculated as the ventricular stroke volume (derived by planimetry of short-axis cine sequences and volume quantification) minus the forward flow across the semilunar valve (derived by phase-contrast CMR).¹⁵¹ The indirect method is highly reproducible as it is not affected by the direction or eccentricity of the regurgitant jet, the presence of AR, nor does it rely on LV geometrical assumptions.¹⁵² However, like AR, there is no consensus on which

cut-off values should be used to define the severity of MR by CMR. Table 3^{127,144,153} shows the suggested cut-off values to define MR severity.^{127,144,153} Visual assessment of the extent of signal loss due to spin dephasing on cine CMR acquisitions and planimetry of the regurgitant orifice can also be used to describe the regurgitation.

LGE technique allows the detection of fibrosis in primary MR [particularly in mitral valve prolapse (MVP)—mitral annular disjunction (MAD)], and in functional MR. The prevalence of LGE in MVP varies across different cohort studies, ranging between 28% and 93%, and is commonly located in the inferolateral basal wall, papillary muscles and less commonly in the myocardium adjacent to these portions.¹⁵⁴

CMR has an emerging role in MAD identification and characterization.¹⁵⁵ MAD is defined by a separation, visible in both diastole and systole, between the ventricular myocardium and the mitral annulus supporting the posterior mitral leaflet. While true MAD is a structural detachment, pseudo-MAD is a separation between the myocardium and annulus only during systole due to the leaflet curling up against the left atrial wall (Figure 17). Recent data suggests inferolateral MAD could be seen as a cue for the clinical to carefully investigate the arrhythmic profile in patients that report arrhythmic symptoms¹⁵⁶ as demonstrated in a retrospective multicentre cohort, where a history of idiopathic ventricular fibrillation was more frequently associated with inferolateral MAD and MVP than healthy controls (11% vs 1%, $P = .02$, 7% vs 0%, $P = .016$ for MAD and MVP, respectively).¹⁵⁷

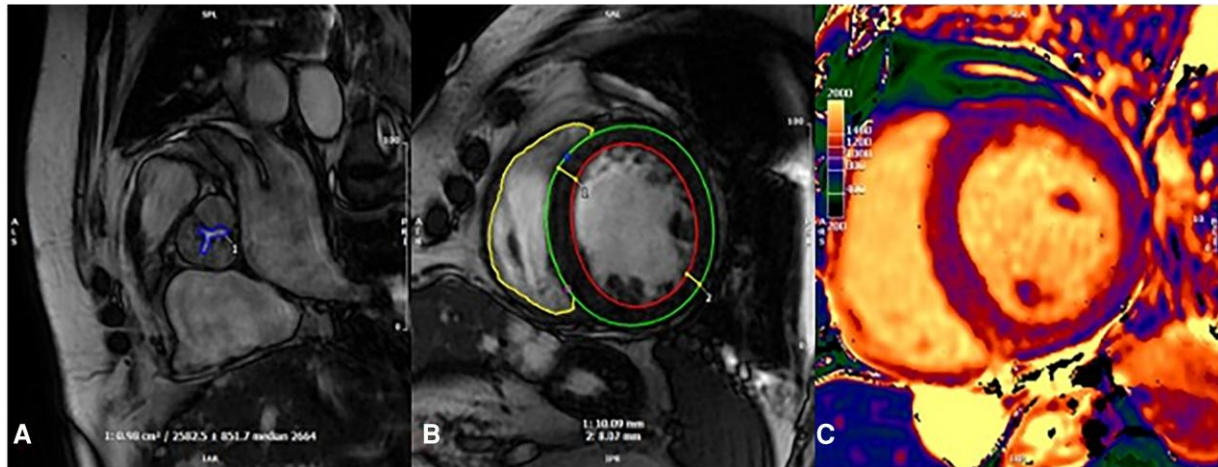


Figure 16 CMR characterization of aortic valve stenosis. Panel A depicts the quantification of aortic valve planimetric area through short-axis cine images. Panel B shows biventricular short-axis view for volumes and parietal thickness quantification. Panel C shows elevated native myocardial T1 mapping demonstrating diffuse interstitial fibrosis

- What role does it have in prognosis/risk stratification?

In the context of MVP, CMR should be performed:

- in patients who survived a cardiac arrest or experienced sustained ventricular arrhythmias;
- before implanting an ICD for secondary prevention;
- when echocardiography does not provide an accurate evaluation of LV and RV function and structural characterization;
- with unexplained syncope and/or non-sustained ventricular tachycardia.¹⁵⁵

Myocardial fibrosis, detected through LGE in MVP is associated with an increased risk of MACE, particularly arrhythmias, independent of the degree of MR.¹⁵⁴ In patients with functional MR and extensive myocardial fibrosis (i.e. >30% of left ventricular mass), the prognosis is usually poor, with mitral valve intervention to be undertaken only in selected cases.¹⁵⁸ However, validation of a CMR-based myocardial fibrosis cut-offs is needed before implementing this in decision-making for routine clinical practice.

Recent studies demonstrate that longer native T1 values in the context of MVP are associated with reduced global and circumferential strain¹⁵⁹ and that an elevated extracellular volume in MVP and MAD is associated with an increase in cardiac arrest, even in the absence of LGE.¹⁶⁰

VHD evaluation

- What clinical questions/information should be in the referral?

The referral should mention the clinical context (e.g. assess AS, MVP with potential symptoms of arrhythmia). Some TTE information may be relevant as the morphology of the aortic valve and its velocities.

- What to look out for in the CMR report?

CMR evaluation for VHD provides a comprehensive of valve morphology, haemodynamics and its effect on the cardiac chambers. The report should include:

General

- Left and right ventricular function and size.
- Presence of LV hypertrophy/distribution, maximum wall thickness, mass
- Left and right atrium size (left atrial volume and diameter)
- Scarring or infarction on LGE
- Description of myocardial viability
- Any other pathology identified (infiltrative/storage disease)

It is important to review the structural and functional assessment of the cardiac chambers, as indices of remodelling, like LV dilatation, LVH and fibrosis suggest VHD progression and the need of intervention.

Aortic valve qualitative assessment

- Leaflets (morphology, thickened/motion/prolapse/masses)
- Presence of sub or supra valvular stenosis

Aortic valve quantitative assessment

- Maximum velocity and severity of AS
- Regurgitant volume
- Regurgitant fraction: mild <30%, moderate 30%–35%, severe >35%^{141,147}

Mitral valve qualitative assessment

- Leaflets (thickened/motion/prolapse/flail/masses)
- Annulus (mitral annular disjunction/thickened/fibrosis)
- Chordae (thickened/shortened/rupture or tear)

Mitral valve quantitative assessment

Table 3 Suggested CMR-Specific MR severity thresholds (^{127,144,153})

CMR grading of regurgitation severity				
Type of MR	Mild	Mild to Moderate	Moderate to Severe	Severe
Primary	MR regurgitant fraction <30%	MR regurgitant fraction = 30%–39%	MR regurgitant fraction 40–49% MR volume >55–60 mL	MR regurgitant fraction >50%
	Mild	Moderate	Severe	
Secondary	MR volume <30 mL	MR volume = 30–60 mL	MR volume ≥60 mL	

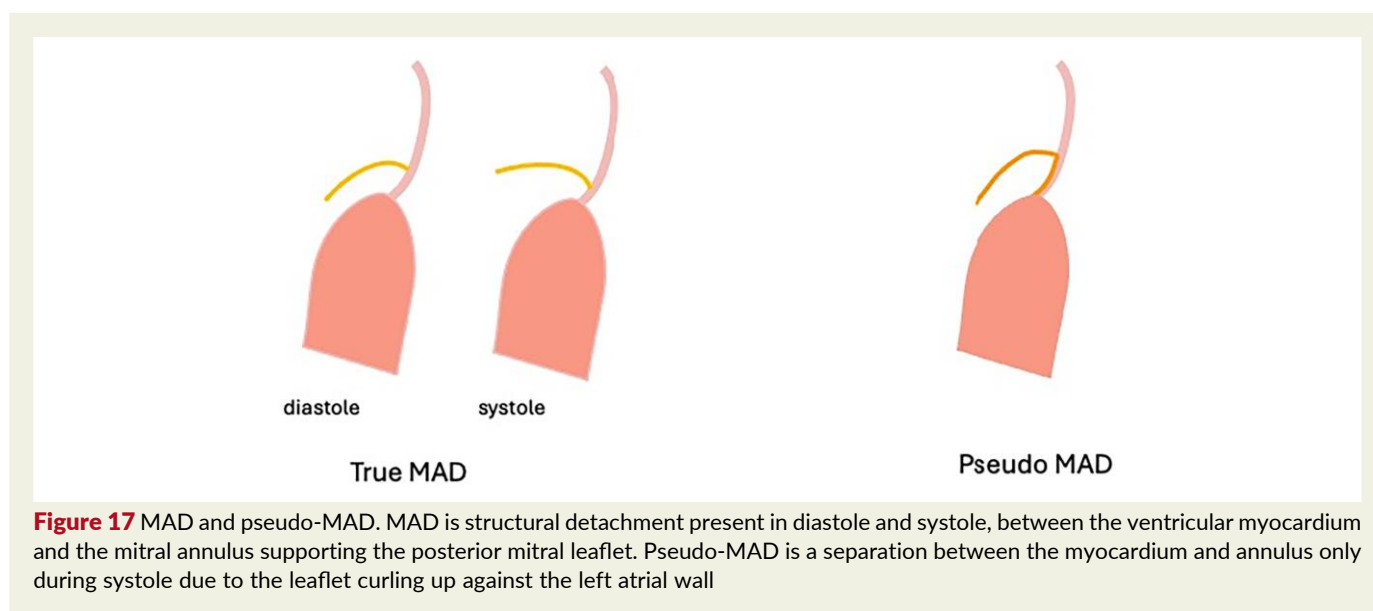


Figure 17 MAD and pseudo-MAD. MAD is structural detachment present in diastole and systole, between the ventricular myocardium and the mitral annulus supporting the posterior mitral leaflet. Pseudo-MAD is a separation between the myocardium and annulus only during systole due to the leaflet curling up against the left atrial wall

- Regurgitant volume
- Regurgitant fraction: mild <30%, mild to moderate 30%–39%, moderate to severe 40%–49%, severe 50%^{127,141,144,153}

Key points

- CMR has a complementary role in VHD assessment
- CMR can be used to quantify the severity of regurgitant lesions when echocardiographic measurements are equivocal or discordant with clinical findings. TTE/transoesophageal echocardiography are superior to assess leaflets/chordal morphology.
- CMR tissue characterization sequences are useful for risk stratification, in identifying patients who would benefit from prompt valve intervention and in patients presenting with potential arrhythmia symptoms.

Aortic diseases

CMR assessment enables the diagnosis, serial monitoring and facilitates surgical planning of aortic diseases. CMR should be considered for:

- Baseline/serial monitoring of thoracic aortic dilatation and aneurysms
- Baseline/serial monitoring of bicuspid aortopathy
- Baseline/serial monitoring of genetic/hereditary aortopathies
- Serial monitoring of stable and uncomplicated type B aortic dissections

CMR offers radiation-free imaging of the entire aorta which is an important consideration, particularly in the follow-up of young patients.

- How CMR can detect/assess?

A complete evaluation of aortic diseases can be made with multiple CMR sequences, including morphology (cine SSFP), diameters (3D angiography), wall tissue characterization (T1 or T2-weighted images), along with aortic valve morphology (cine SSFP) and function (phase contrast).^{147,161} CMR is useful for diagnosing and following aortic dilatation/aneurysm, inflammation/infection,^{162,163} congenital or genetic diseases and follow-up after acute aortic syndromes.¹⁶⁴ Gadolinium administration is not mandatory,^{165,166} but allows rapid angiogram acquisition and the assessment of aortic wall inflammation. In recent years,

4D flow sequences have allowed the evaluation of complex flows in the aorta and haemodynamic parameters with diagnostic and prognostic implications.¹⁶⁷⁻¹⁷⁰

- What role does it have in prognosis/risk stratification?

In patients with aortic dilatation, the maximal diameter is the main predictor of dissection/rupture. Surgery is indicated in dilatation of the aortic root or ascending aorta with a tricuspid aortic valve and a maximum diameter of ≥ 55 mm, maximal aortic sinus diameter ≥ 50 mm in Marfan syndrome, or ≥ 45 mm in Marfan syndrome with risk factors or when aortic valve replacement is also indicated.¹⁷¹ Surgery for bicuspid aortopathy of the root phenotype is advised when the maximum aortic diameter is ≥ 50 mm. An annual growth rate > 3 mm/year is considered a risk factor for aortic events.¹⁷² In the case of extreme body surface area (i.e. Turner's), the use of indexed diameters should be reported; therefore, a diameter > 25 mm/m² has been associated with adverse aortic events.¹⁷³ Finally, the Z-score is advised in paediatric populations or Marfan patients.⁹⁵⁻⁹⁷

- What clinical questions/information should be in the referral?

To optimize the protocol, the referral should mention the clinical context (acute aortic syndrome, aneurysm, genetic, or inflammatory diseases). Although TTE information may be relevant (e.g. morphology of aortic valve and its velocities), it is not strictly necessary. In follow-up studies, the time elapsed since the previous study, or the acute event is important for the correct interpretation of new findings.

- What to look out for in the CMR report?

The report should include anatomical variants/congenital abnormalities, stenosis or dilatation with minimum/maximal diameter and location. The suggested method for measurements of aortic diameters is inner-to-inner edge in diastole except for the aortic annulus measurements before transcatheter aortic valve replacement which are measured in systole.^{161,174} Cusp-to-cusp measurement is the advised method for the assessment of the aortic root.^{174,175} Any asymmetry (difference between diameters ≥ 5 mm) should be reported as those cases show greater discrepancy with other imaging techniques such as TTE.¹⁷⁶ The rest of the aorta should be measured in the anteroposterior diameter (or the largest diameter if the aorta is noncircular) and the diameter perpendicular to it. The aorta must be measured at different anatomical landmarks (Figure 18).¹⁶⁴ The aortic growth rate must be reported with side-by-side comparison from the previous study using an identical measuring technique.

The aortic valve morphology and function should also be reported. False lumen diameters, characteristics, entry tears, and flow patterns should be described in the case of aortic dissection (Figure 19).^{168,177}

- Comparison with echocardiogram and CT

TTE is limited by the acoustic window and evaluation of the complete thoracic aorta. CT is widely available and allows fast acquisition of the thoracic and abdominal aorta (mostly in acute aortic syndromes) with superior spatial resolution;

nevertheless, it requires ionizing radiation, a limitation mostly for the long-term follow-up of young patients with familial diseases.

CMR also presents limitations in monitoring unstable patients, very long scanning times (especially compared with CT) if multiple sequences are needed and the inability to evaluate aortic wall calcifications.^{172,178}

Adult congenital heart disease

There are increasing numbers of adults with congenital heart disease (ACHD) who are mostly followed up at specialist units. However, some conditions may present or be picked up during imaging in adulthood and include conditions like: atrial septal defects, anomalously connected pulmonary veins, partial atrioventricular septal defects, congenitally corrected transposition of the great arteries, Ebstein anomaly, aortic coarctation, and patent ductus arteriosus. Complex ACHD conditions are typically followed within specialized ACHD services at tertiary centres. As such, their ongoing management, including advanced imaging surveillance, would not usually fall within the scope of routine care provided by general cardiologists; therefore, it would be beyond the scope of this manuscript.

CMR has taken an increasingly important role in the assessment and follow-up of ACHD by comprehensive assessment of:

- CMR provides an unlimited field of view to access structures throughout the thorax.
- Morphology and function of the left and right ventricle: The RV geometry is complex, forming a crescent shape that wraps around the left ventricle. Depending on the underlying ACHD, the RV geometry can change when it is exposed to volume and/or pressure overloads.¹⁷⁹ CMR is the gold standard for accurate and reproducible measurements of the right ventricle regardless of its morphology or orientation within the thorax, and without geometrical assumptions.¹⁸⁰
- Great vessels and their relationship to cardiac structures: This is assessed on cine images, black-blood still-images and angio/3D whole heart sequence $\pm$ GBICA.
- Morphological assessment of valves and their haemodynamic assessment by phase-contrast velocity mapping and, more recently 4D flow sequences.
- Shunt evaluation: CMR is a well-established non-invasive method to accurately quantify intracardiac shunts and will be discussed in further detail below.
- The assessment of how the myocardium is affected by T1 mapping and LGE sequences.
- How CMR can detect/assess -some specific indications
 - i. Shunts: atrial septal defect, ventricular septal defect, or patent ductus arteriosus (see Movie S4)

CMR can address some remaining questions after echocardiography:

- Non-invasive quantification of the shunt
- Accurate biventricular size and function
- any associated anomalies as anomalous pulmonary venous drainage (Figure 20).

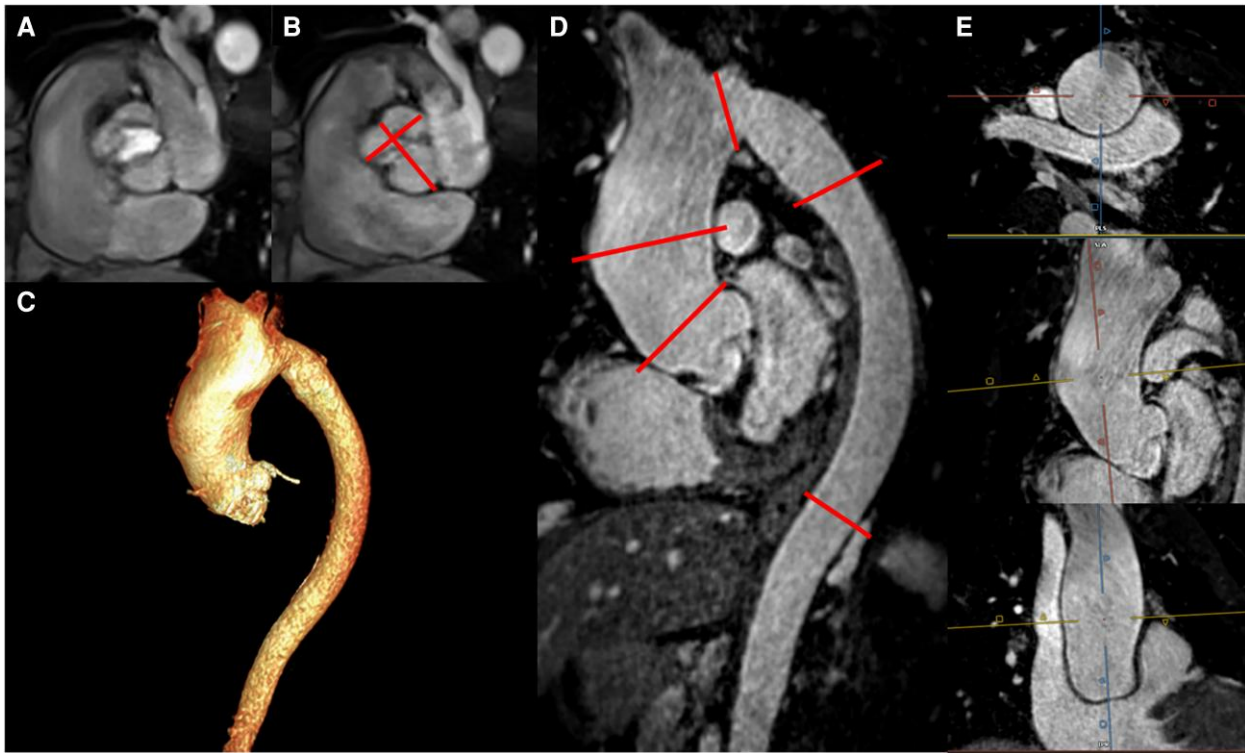


Figure 18 A patient with a bicuspid aortic valve and ascending aorta dilatation. A and B, cine b-SSFP of the aortic valve showing fusion of the right and left coronary cusps and an asymmetric aortic root. C, 3D angiography volume rendering and D, MPR with the recommended anatomical landmarks for measurement, using the double-oblique technique as shown in E

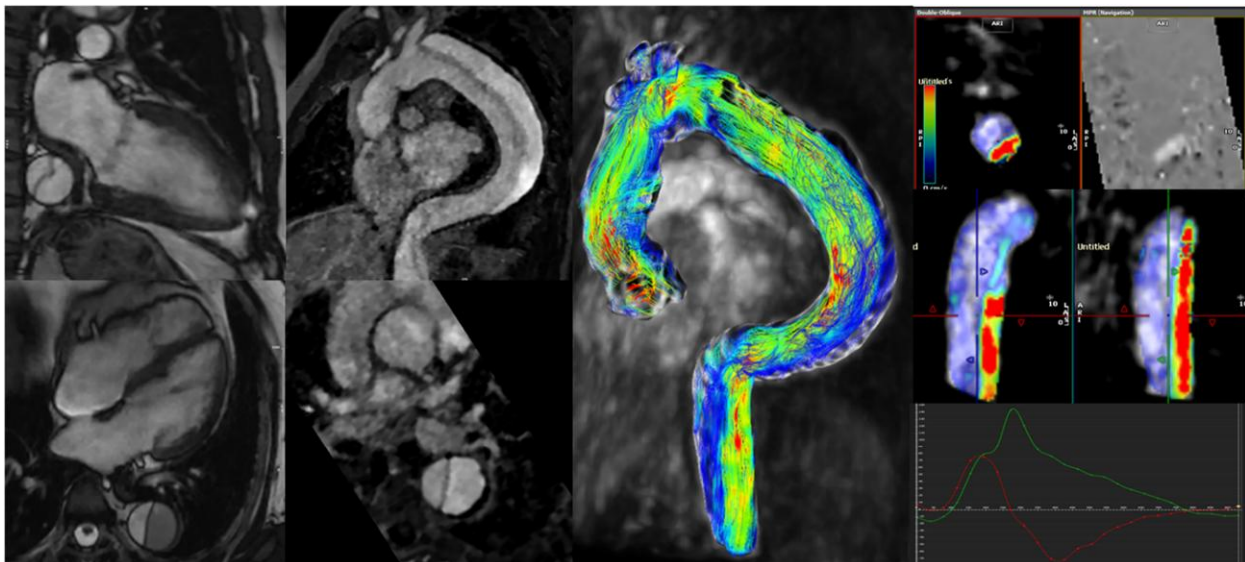


Figure 19 Chronic aortic dissection. A and B, cine b-SSFP 2-ch and 4-ch view showing a cross-section of the descending aorta with an evident intimal flap. C and D, 3D angiography sagittal plane and double-oblique cross-section for diameter measurement and evaluation of the true lumen vs the false lumen with partial thrombosis (arrow). E-G, 4D-flow sequence allowing analysis of flow direction and quantification of both the true (green line) and false lumen (red line) throughout the aorta

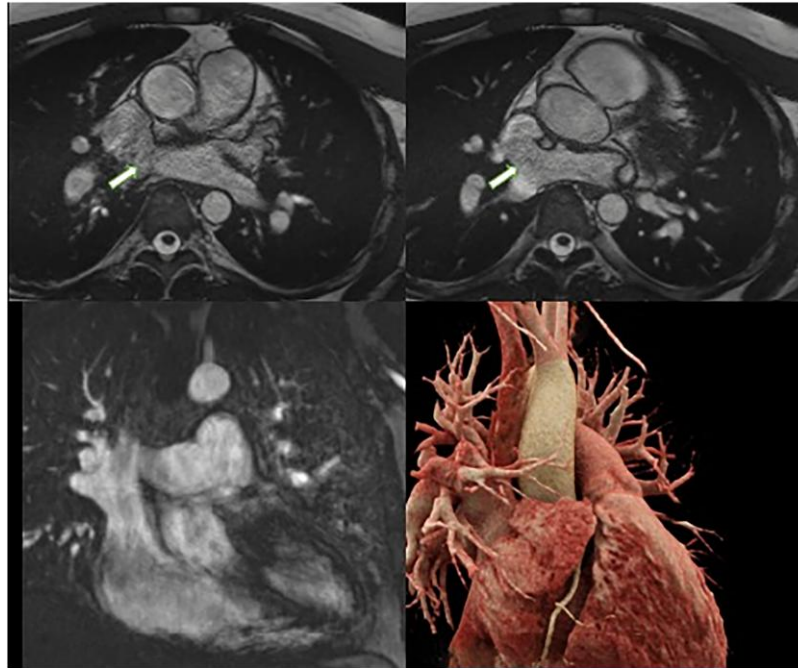


Figure 20 Sinus venosus ASD and partial anomalous pulmonary venous drainage. The top panel shows axial cine images in which the display the communicating defect high in the interatrial septum between the left atrium and the SVC. The lower panel shows an MRA and a volume rendered image of the drainage of the blood flow from the right upper lung pulmonary veins into the right SVC

A Qp:Qs of $>1.5:1$ indicates a haemodynamically significant shunt.¹⁸¹ CMR Qp:Qs shunt quantification can be performed either indirectly or directly. Indirect quantification can be performed using phase-contrast velocity mapping perpendicular to the aorta and main pulmonary artery to measure blood flow or volumetric analysis of short-axis images to compare RV stroke volume to LV stroke volume.^{177,182} However, these techniques are inaccurate in the presence of significant valvular regurgitation.

Shunt quantification for a haemodynamically significant PDA is unique as indirect flow calculation will give a Qp:Qs <1 . If the QP is measured at the MPA, it will fail to capture the extra blood flow the pulmonary arteries receive just distally via the duct. Here, Qp is the LV stroke volume as it receives all the pulmonary venous blood flow and Qs is total systemic flow (superior vena cava + descending aorta).¹⁸³ 4D flow enables the direct non-invasive measurement of shunt volume, mean jet diameter, and area.¹⁶⁹ However, this technique is not available in all CMR centres.

ii. Aortic coarctation (see [Movie S5](#)):

- severity and site of stenosis and its relation to the left subclavian artery.
- thoracic aorta morphology and presence of collateral vessels
- in previous coarctation repair, restenosis or aneurysm formation
- associated pathology as bicuspid aortic valve and aortopathy
- LVH

- quantification of collateral flow by comparing through-plane flow measurements immediately proximal to the coarctation stenosis and at the level of the diaphragm
- presence of a diastolic tail is a sign of significant (re-)coarctation

iii. Ebstein anomaly and tricuspid regurgitation (see [Movie S6](#)):

- Evaluation of the RA–RV anatomy and tricuspid valve morphology and extent of apical displacement by stack of transaxial, four-chamber, RV 3, and 2-chamber cine images. Transaxial cine images allow more consistent inter- and intraobserver measurements as compared with short-axis cine images as the axial plane affords a better depiction of tricuspid valve displacement and point of leaflet coaptation. The RV volume values may be higher than normal in the presence of severe tricuspid regurgitation.
- Associated anomalies and/or secundum atrial septal defect should be reported
- Quantification of an intracardiac shunt
- Severity of tricuspid regurgitation is derived by an indirect approach from ventricular stroke volume (derived by planimetry of short-axis cine sequences and volume quantification) to forward flow across the semilunar valve (derived by phase-contrast CMR).¹⁸⁴
- Congenitally corrected transposition of the great arteries (see [Movie S7](#))
- It is a malformation that consists of discordant atrio-ventricular and discordant ventriculo-arterial connections. The right ventricle is subaortic and the systemic ventricle.

Cine images show these abnormal connections, multiple coarse trabeculations, the moderator band of the RV and allow functional and volumetric evaluation

- Associated lesions include dextrocardia, a ventricular septal defect (VSD), (sub-)pulmonary stenosis and Ebstein-like malformation of the left-sided tricuspid valve.
- Quantification of shunting through a VSD and the severity of any (sub-)pulmonary stenosis or tricuspid regurgitation should be assessed by flow and cine sequences.

- What clinical questions/information should be in the referral?

The referrer should clearly state the clinical suspicion and be encouraged to speak to the cardiac imager to discuss why the scan has been requested if it is thought the anatomy is complex. It is important that the clinical question is clear as once the images are acquired and the scan is finished, no additional images can be acquired.

- What to look out for in the CMR report for the ACHD evaluation depending on the reason for referral?
 - Segmental, sequential, and analytic description of the anatomy
 - Left and right ventricular regional and global function
 - Presence of LV and/or RV hypertrophy, distribution, maximum wall thickness, and mass
 - Left and right atrial size (left atrial volume and diameter)
 - Scarring or fibrosis on late gadolinium enhancement—enhancement of the RV–LV insertion regions is a common, non-specific finding where one or both ventricles are hypertrophied or dilated and of doubtful clinical significance.
 - Description of the valve leaflets and orifice opening, any abnormalities of the subvalvular apparatus
 - Valve quantitative assessment: Maximum velocity and severity of stenosis, regurgitant volume and regurgitant fraction (%)
 - Presence of sub- or supraaortic and or pulmonary stenosis
 - Shunt quantification
 - Aortic measurements
 - Examples of findings of some specific ACHD conditions are as described above.
- Key points
 - CMR provides an unlimited field of view to access to structures throughout the thorax
 - CMR can answer functional as well as anatomic queries made by the referrer, including the location and severity of stenosis and/or regurgitation, the size and function of the cardiac chambers, and measurement of shunt flow
 - CMR is the gold standard for the assessment of the RV size and function

Pericardial disease

CMR may serve as a beneficial second-line imaging technique, especially in patients with suboptimal acoustic windows, localized pathology, or with an unfavourable disease progression. The scanning protocol includes spin-echo T1-weighted sequences to assess pericardial anatomy and thickening, cine-SSFP for function and haemodynamic compromise, and

STIR and LGE imaging for pericardial inflammation. Normal pericardial thickness is typically 2 mm.¹⁸⁵

- Pericardial effusion
- How CMR can detect/assess?

CMR can detect pericardial fluid without the use of GBCA, may differentiate a transudate from exudate and identify loculations/septations. Haemodynamic compromise is suggested by the presence of heart chamber collapse and signs of ventricular interdependence. Detection of other heart diseases (e.g. myocarditis or myocardial infarction with rupture), or in adjacent organs (e.g. pulmonary nodules) may clarify the aetiology of the effusion.

- What clinical questions should be asked in the referral?

The clinical suspicion of pericardial effusion should be stated and the clinical context (infectious/rheumatic diseases, after infarction or cardiac surgery, chest pain, etc.).

- What to look out for in the CMR report

The report should specify the location and magnitude of the effusion, whether it is suggestive of a transudate or exudate, and the haemodynamic repercussion. Associated cardiac and extra-cardiac findings should be noted.

Pericardial constriction

- How CMR can detect/assess?

Pericardial constriction occurs as a complication of acute or recurrent pericarditis. In acute pericarditis, the pericardium is thickened and hyperintense on STIR and LGE images, while the presence of ventricular interdependence suggests constriction and morphological changes of the ventricles (*Figure 21*). It provides a complete anatomical depiction of the affected pericardium, which may be useful for surgical planning.

- What role does it have in prognosis/risk stratification?

CMR can differentiate an acute/subacute pericarditis, which may respond to initiation or up-titration of anti-inflammatory agents,¹⁸⁶ from a chronic phase that may require pericardiectomy. In recurrent pericarditis, a smaller extent of LGE was associated with clinical recovery and a longer time to recurrence,¹⁸⁷ and a CMR-guided treatment was associated with a lower dose of steroids and the need for pericardiocentesis.¹⁸⁸

- What clinical questions should be asked in the referral?

The clinical context should be stated, including ongoing drug treatment and previous CMR findings. The presence of pericardial inflammation and signs of constriction should be inquired.

- What to look out for in the CMR report

The report should include the presence of effusion and/or constriction, location, thickness, presence of active inflammation—acute/subacute or chronic stage and presence of myocarditis.

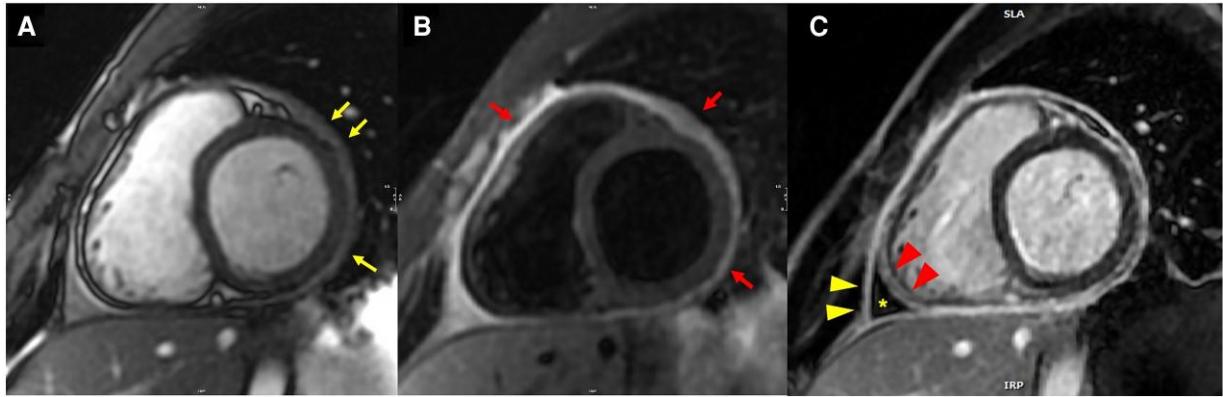


Figure 21 Basal short-axis of a patient with acute pericarditis. A, cine b-SSFP showing thickened pericardium most evident over the left ventricular lateral wall (arrows). B, STIR showing circumferential pericardial hyperintensity suggestive of inflammation (arrows). C, in the LGE image hyperenhancement of both pericardial layers (arrowheads) can be differentiated due to the presence of a small pericardial effusion (asterisk)

• Comparison with Echocardiogram and CT

TTE is widely available, can assess effusion and haemodynamic consequences of constriction in more detail and guide pericardiocentesis. However, it is limited in posterior effusions and cannot accurately assess pericardial thickening or inflammation.

CT has a wide field-of-view, excellent anatomic definition and can detect calcification. However, it may be difficult to differentiate pericardial thickening from fluid and depict pericardial enhancement suggestive of inflammation.

- Key points
- CMR is a second-line imaging modality for pericardial diseases.
- CMR can distinguish a transudate from an exudate effusion.
- CMR can guide the therapeutic management of pericardial constriction by providing the location/extent of pericardial thickening and whether this is acute/chronic stage of inflammation.

Cardiac masses

CMR has emerged as a powerful diagnostic tool for the identification and evaluation of cardiac masses, due to its unique combination of excellent soft tissue contrast and high spatial resolution, allowing for accurate detection and characterization of cardiac tumours and pseudomasses, including thrombi, vegetations, and other benign non-neoplastic lesions.¹⁸⁹ It can help distinguish between benign and malignant lesions, characterize the extent of infiltration, and provide critical information for treatment planning and monitoring. Expert interpretation of CMR findings may obviate the need for invasive biopsy in the context of benign tumours, but in challenging scenarios, histopathological characterization by tissue biopsy is required for definite diagnosis and treatment guidance.

• How CMR can detect disease?

Standardized and tailored CMR protocols should be employed for optimal assessment and accurate diagnosis of cardiac masses.

A CMR protocol¹⁹⁰ for cardiac mass evaluation should include the following sequences in imaging planes that optimally visualize the mass:

- localizer and cine bSSFP sequences in standard and at least two orthogonal customized imaging planes for optimal mass localization and morphological assessment;
- T1-weighted and T2-weighted sequences to evaluate signal intensity characteristics;
- T1 and T2 mapping for quantitative assessment of tissue properties, offering additional information for accurate characterization of tissue composition and pleuro-pericardial effusion;
- first-pass perfusion imaging and EGE to assess for vascularity;
- LGE imaging to identify areas of fibrosis and necrosis;
- $\pm$ real-time imaging or motion-correction techniques to minimize motion artefacts;
- $\pm$ dark-blood techniques can be beneficial for the evaluation of intracavitary thrombi or small masses adjacent to the blood pool;
- $\pm$ optional 3D sequences can be useful for complex masses or when surgical planning is required;
- $\pm$ the addition of myocardial tagging sequences can be useful in the evaluation of malignant cardiac masses that appear to infiltrate the myocardium or pericardium to evaluate adherence and immobility of the pericardial-myocardial interface.¹⁹¹

• What role does CMR have in prognosis and risk stratification?

In suspected cardiac tumour, CMR yields high diagnostic accuracy and is a robust and independent predictor of long-term mortality. Tumour size, location, invasiveness, and tissue characteristics can help predict the risk of embolization, arrhythmias,

and other complications, as well as the likelihood of malignant vs benign lesions.

Differentiation between benign and malignant lesions is key to stratify prognosis, to guide downstream management and repeat imaging, and to avert the need for invasive biopsy in

Table 4 What to check before referring a patient with CIED to CMR

Make sure that there is no alternative imaging test to answer the clinical question (especially for non-MR conditional devices)

The clinician should provide the clinical necessity of CMR for which the possibility of complication is weighted.

Refer the patient to a centre experienced in scanning patients wearing CIEDs

Wait 6 weeks after CIED implantation before referring the patient to CMR. In some centres, it is done before this on a case-by-case basis

Check MR-conditional (MR-safe, MR-conditional, non-MR conditional) of all CIED's components; if in doubt ask/refer the patient to the Pacing/Electrophysiology Clinic and/or check on dedicated MR safety website

Check whether the patient is MR-dependent or not! Provide the CMR unit with this information

Make sure the patient will receive Pacing/Electrophysiology Clinic check-up before and after CMR scan for safe CIED programming

Make sure that the medical device ID card is available to the CMR unit when planning the scan and on the day of scanning

situations where masses are benign and do not warrant surgery. CMR features that aid in differentiating benign from malignant masses include location, size, morphology, mobility, signal intensity, and early and late enhancement patterns. Malignant tumours tend to exhibit irregular borders, heterogeneous signal intensity, rapid and heterogeneous contrast uptake on first-pass perfusion, and areas of necrosis on LGE imaging. First-pass perfusion imaging is essential for evaluating the vascular supply of a cardiac mass and is particularly valuable in differentiating between malignant and benign tumours and identifying the presence of intratumoral necrosis. During the first-pass perfusion phase, malignant tumours typically demonstrate rapid contrast enhancement due to their hypervascularization, while benign tumours and non-neoplastic masses show delayed or minimal enhancement. Areas of intratumoral necrosis or cystic degeneration may exhibit hypoperfusion or no enhancement, aiding in further characterization of the mass. In contrast, benign tumours often have well-defined borders, homogeneous signal intensity, and a delayed or minimal enhancement pattern.

- What clinical questions should be asked in the referral?

Major reasons for the CMR referral include the clinical need to (i) distinguish between tumoral vs non-tumoral (including imaging pitfalls, anatomic variants), and/or benign vs malignant lesions; (ii) characterize the extent of infiltration; (iii) detect tumour-associated complications (e.g. pleuro-pericardial effusion, superimposed thrombus); (iv) provide critical information for treatment planning and monitoring effectiveness or early signs of recurrence (size, growth rate, degree of invasiveness, propensity to embolization, changes in signal intensity, perfusion, and enhancement pattern, or infiltration of surrounding tissues).

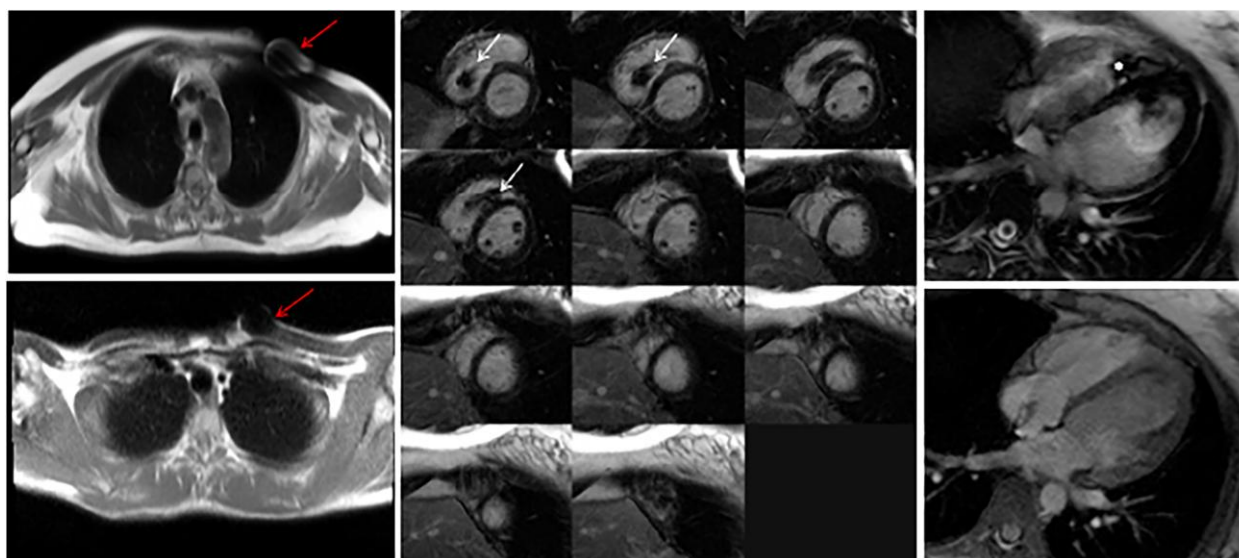


Figure 22 Left, axial localizers showing the typical local artefact of a pacemaker (top, red arrow) and loop recorder (bottom, red arrow) generator. Middle, short-axis post-contrast sequences showing no evidence of late gadolinium enhancement (LGE); a pacemaker lead (white arrow) is noted within the right ventricle. Right, steady state free precession 4-chamber cine sequence showing artefact from the pace-maker lead (top, asterisk); the artefact is resolved by using gradient echo cine sequences (bottom)

Table 5 Indications of CMR

Indications of CMR	
Ischaemic heart disease	
Acute myocardial infarction and complications	• an unclear clinical presentation to distinguish post-AMI from other aetiologies as myocarditis/Takotsubo; • conventional echocardiograms and/or angiograms are not diagnostic/inconclusive; • differentiate acute from chronic myocardial infarction; • assess infarct size and viability; • diagnosis of mechanical complications
MINOCA	Presentation of symptoms suggestive of acute coronary syndrome, with troponin elevation and non-obstructive (<50% stenosis) coronary arteries at the time of ICA
Chronic coronary syndrome	• to evaluate global and regional function • use of stress CMR to diagnose and quantify myocardial ischaemia and/or scar in suspected CCS with moderate or high (>15%–85%) pre-test likelihood of obstructive CAD • to assess myocardial scar, viability and the extent of transmural in post-MI patients
Non-ischaemic cardiomyopathies	
Myocarditis	• first-line diagnostic tool for the evaluation of myocarditis • follow-up CMR to assess whether inflammation activity has resolved or has progressed to a DCM • facilitates risk stratification, often replacing the need for biopsy
DCM	• confirm the diagnosis of DCM and determine the aetiology • prognosis/risk stratification
HCM	• when echocardiogram is inconclusive • to differentiate HCM from other causes of hypertrophy and phenocopies • risk stratification for sudden cardiac death • planning for myectomy • serial assessment to monitor disease progression • screening of 1st degree relatives to detect an early phenotype
Cardiac amyloidosis	• ancillary imaging modality in the diagnosis of cardiac amyloidosis • prognosis/risk stratification
Fabry disease	• in the differential diagnosis of left ventricular hypertrophy • precise assessment of morphological, functional, and tissue characterization • serial monitoring after enzyme replacement therapy • risk stratification
CARDIAC SARCOIDOSIS	• confirm cardiac involvement in histological diagnosis of extra-cardiac sarcoidosis • guide to endomyocardial biopsy, increasing its diagnostic yield • prognosis/risk stratification
Iron overload cardiomyopathy	• diagnosis and monitoring of myocardial and liver iron overload • risk stratification- lowest T2* values have the highest risk of developing arrhythmia and heart failure
Valvular Heart Disease	• used to complement VHD assessment in cases of limited or discordant clinical and echocardiographic findings • secondary test to confirm the severity of VHD, particularly in regurgitant lesions. • prognosis/risk stratification by quantification of ventricular volumes, assessment of valve severity and detection of fibrosis
Aortic diseases	• Baseline/serial monitoring of thoracic aortic dilatation and aneurysms • Baseline/serial monitoring of bicuspid aortopathy • Baseline/serial monitoring of genetic/hereditary aortopathies • Serial monitoring of stable and uncomplicated type B aortic dissections
Adult congenital heart disease	• Morphology and function of LV and RV, particularly the RV • Great vessels and their relationship to cardiac structures • Morphological assessment of valves and their haemodynamic assessment • Shunt evaluation • ±Myocardial Fibrosis

Continued

Table 5 Continued

Indications of CMR	
Pericardial disease	• Evaluate pericarditis or pericardial constriction
Cardiac tumours	• Confirm presence/absence of suspected mass with description of location, extent, anatomic relationships, features benign vs malignant lesions, extent of infiltration if malignant • provide critical information for treatment planning and monitoring effectiveness or early signs of recurrence

- What to look out for in the CMR report?

The report should include (i) confirmation of the presence/absence of suspected mass; (ii) detailed description of location, extent, anatomic relationships, complications and associated features of the mass; (iii) mass size in three cross-sectional dimensions; (iv) evaluation of margins as smooth, irregular or infiltrating; (v) description of T1-weighted, T1-weighted PSIR, T2-weighted, parametric mapping, and contrast; (vi) assessment of red-flag features that may suggest malignancy; (vii) a differential diagnosis should be offered, while it should be made clear that CMR, like any imaging technique, cannot be used to offer a tissue diagnosis.¹⁹⁰

- Key points
 - It is important to note the different benign and possibly malignant features when receiving a report;
 - CMR, like any imaging technique, does not replace a histopathological diagnosis, particularly when malignancy is suspected.

Use of CMR in patients with implantable devices

CIEDs, such as pacemakers and ICDs, have long been considered a contraindication to CMR, with patients wearing devices being fifty times less likely to undergo CMR than the general population.^{192,193} The main concerns of scanning patients wearing CIEDs derive from the potential interactions and adverse effects of the CMR system on electronic devices: (i) although rare, the static magnetic field may cause device or lead displacement, (ii) CIED function may be impaired by gradient-induced electrical currents (i.e. oversensing, disabled tachycardia detection), (iii) radiofrequency energy may induce heating effect, and (iv) battery life may be altered.^{192,193}

Considering the increasing prevalence and likelihood of a life-time indication to CMR of patients wearing CIEDs, and CMR's wider clinical applications, there has been a need to develop MR-conditional devices to increase safe accessibility to CMR.¹⁹²⁻¹⁹⁶ MR-conditional devices are considered safe within defined scanning conditions, regarding static magnetic field, time-varying gradient magnetic fields and radiofrequency fields. MR-conditional is determined by the entire CIED system, not the single components; therefore, it has to be remembered that generators and leads from different manufacturers, although both MR-conditional, render the system non-MR-conditional, as they may not have been tested combined.^{195,197}

Non-MR-conditional should, however, not be considered a permanent categorization as some previously non-MR conditional leads and generators have been retrospectively tested and defined as MR-conditional.^{195,197} To date, there have been no deaths attributable to CMR in patients with CIEDs, when performed following the correct protocols, and no major events were reported in more than 2000 thoracic and non-thoracic scans of non-MR-conditional devices that were properly programmed and correctly scanned.¹⁹²⁻¹⁹⁶ It is therefore important that clinicians know that it is nowadays safe to scan patients with almost all devices, provided that the scanning protocol is strictly followed, under close medical supervision in experienced centres, and following a careful risk-benefit ratio assessment establishing that there is no alternative imaging test^{195,197} (Table 4; Figure 22). Abandoned, fractured, or epicardial leads still render the entire CIED system MR-unsafe, although no complications have been reported to date.¹⁹⁷

Access to CMR for patients wearing CIEDs is also limited by the reported lower image quality. The metallic components of CIEDs induce a local gradient field around the device, which is responsible for metallic susceptibility artefacts, which are larger for larger generators (i.e. ICDs), and can affect image quality and interpretability. However, using spoiled gradient echo sequences for cine imaging, as opposed to the mostly used balanced steady state free precession sequences, and wideband late gadolinium enhancement for post-contrast assessments usually helps reduce the extent of artefacts.^{192,193,195} (Figure 19, right panel).

In summary, if CMR is clinically indicated and no alternative imaging test is available, CMR can be safely performed in patients wearing CIEDs, provided that scanning protocols are strictly followed under experienced medical supervision. Before referring a patient to CMR, the referring physician should check the MR safety of all CIED's components and make sure that the medical device ID card is available to the MRI unit staff to set the most appropriate device programming and scanning protocol.

Conclusion

The advances in CMR over the past decades have led to an increase in its clinical use in everyday practice for its capability to aid in the diagnosis, risk stratification, prognosis, and follow-up of a wide array of cardiac conditions (Table 5).

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Supplementary data

Supplementary data are available at [European Heart Journal](#) online.

Declarations

Disclosure of Interest

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Data Availability

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