International Journal of Research in Medical Sciences
Khair LA. Int J Res Med Sci. 2026 May;14(5):2162-2167
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20261369

Review Article

Cardiac magnetic resonance in ischemic cardiomyopathy: from tissue characterization to prognostic stratification

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Received: 15 March 2026
Accepted: 20 April 2026

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ABSTRACT

Ischemic cardiomyopathy (ICM), the most prevalent cause of heart failure globally, remains a significant contributor to cardiovascular morbidity and mortality. Despite advances in medical and interventional therapies, outcomes for patients with ICM remain suboptimal, underscoring the need for accurate diagnostic tools and effective risk stratification. Cardiac magnetic resonance imaging (CMR) has emerged as a powerful, non-invasive modality that provides detailed anatomical, functional, and tissue-level insights essential for managing this complex condition. A literature search was performed using PubMed, Embase, and the Cochrane Library to identify relevant studies published between January 2005 and January 2026. Search terms included combinations of "cardiac magnetic resonance," "ischemic cardiomyopathy," "late gadolinium enhancement," "stress perfusion CMR," "myocardial viability," "prognosis," and "revascularization." Randomized controlled trials, cohort studies, systematic reviews, meta-analyses, and major society guidelines evaluating CMR in adult patients with suspected or established ischemic cardiomyopathy were included. This review explores the comprehensive role of CMR in the diagnosis, differentiation, and prognostication of ICM. It highlights CMR's superiority in quantifying ventricular function and detecting myocardial ischemia using routine cine and perfusion sequences, while also emphasizing the emerging role of CMR speckle tracking—referred to as feature tracking (CMR-FT)—in the detection of ischemia and the assessment of myocardial viability. The review further evaluates the role of CMR in distinguishing ischemic from non-ischemic cardiomyopathies through advanced tissue characterization techniques, including parametric mapping and late gadolinium enhancement (LGE). Additionally, it discusses CMR's utility in guiding revascularization decisions and predicting clinical outcomes based on scar burden and myocardial viability. Although certain limitations remain, such as accessibility and patient compatibility, CMR continues to represent the standard for non-invasive cardiac imaging in ICM, providing clinicians with critical information across all stages of patient care, however, the expanding applications of CMR must be interpreted within evolving evidence and guideline recommendations rather than generalized superiority claims.

Keywords: Ischemic cardiomyopathy, Cardiac magnetic resonance, Late gadolinium enhancement, Balanced steady-state free precession, Feature tracking

INTRODUCTION

Ischemic heart disease continues to rank as the leading cause of death and disability worldwide and it remains the most prevalent underlying cause of cardiomyopathy and heart failure.[1] In a pooled analysis of 13 major multicenter heart failure trials, ischemic cardiomyopathy (ICM) was responsible for nearly 70% of all heart failure cases.[2] Clinically, ICM is defined by impaired left ventricular (LV) systolic function in the setting of significant coronary artery disease.[2]

Despite significant progress in pharmacotherapy, device-based treatments, and revascularization techniques, ICM still imposes a heavy global health burden. Mortality rates hover around 16% at one year and can climb to nearly 40% by five years in both the U.S. and Europe.[3] These figures underscore the urgent need for early, accurate diagnosis,

refined risk stratification, and personalized therapeutic approaches.

A key clinical hurdle remains the differentiation of ischemic from non-ischemic cardiomyopathy—an essential step that directly impacts management decisions, including candidacy for revascularization, device therapy, and prognosis. In this context, cardiac magnetic resonance imaging (CMR) has emerged as an indispensable tool.

CMR offers a comprehensive, radiation-free assessment of cardiac structure and function. It accurately measures ventricular volumes and systolic performance, detects myocardial ischemia through stress perfusion imaging, and distinguishes between ischemic and non-ischemic tissue using advanced tissue characterization techniques such as T1, T2, T2*, and late gadolinium enhancement (LGE). Beyond diagnostic precision, CMR provides vital prognostic information and guides treatment planning—without the limitations of acoustic windows that affect other imaging modalities.[4]

However, CMR is not without limitations. Barriers include limited scanner availability, higher operational costs, and longer acquisition times compared with other imaging modalities. In addition, assessment and quantification of LGE and myocardial perfusion—although clinically valuable—may be subject to interobserver variability and remain less standardized across centers compared with established nuclear imaging techniques. Patient-related factors such as claustrophobia and contraindicated metallic implants (e.g., certain cerebral aneurysm clips or intraocular metallic fragments) may further restrict its applicability in selected populations.[4-6]

Review objectives

This review aims to explore the multifaceted role of cardiac MRI in: diagnosing ischemic cardiomyopathy, differentiating ischemic from non-ischemic forms, detecting myocardial ischemia in cardiomyopathy patients, informing revascularization strategies, and assessing prognosis in individuals with ICM.

METHODS

A structured literature search was conducted using PubMed, Embase, and the Cochrane Library to identify relevant studies published between January 2005 and January 2026 in the English language. The search strategy incorporated combinations of the following keywords: "cardiac magnetic resonance," "ischemic cardiomyopathy," "late gadolinium enhancement," "stress perfusion CMR," "myocardial viability," "prognosis," and "revascularization." Eligible studies included randomized controlled trials, prospective or retrospective cohort studies, meta-analyses, systematic reviews, and major international society guidelines evaluating the role of CMR in adult patients (≥18 years) with established or suspected ischemic cardiomyopathy. Exclusion criteria comprised studies focusing exclusively on non-ischemic cardiomyopathies, congenital heart disease, pediatric populations, purely technical MRI physics without clinical correlation, case reports or small case series (<20 patients).

DISCUSSION

Standard CMR protocol in the evaluation of ischemic cardiomyopathy

Per guidelines from the Society for Cardiovascular Magnetic Resonance (SCMR) and the European Association of Cardiovascular Imaging (EACVI), the standard CMR protocol for ICM includes the following components: cine bSSFP imaging for ventricular volumes, global and regional systolic function, T2-weighted or T2 mapping for identifying myocardial edema, native and post-contrast T1 mapping, including extracellular volume (ECV) quantification, for evaluating diffuse fibrosis, and LGE imaging for detecting and quantifying focal myocardial fibrosis and scar tissue.[7]

Together, these sequences allow for a holistic evaluation of the ischemic myocardium. Based on the current evidence, CMR plays four critical roles in the diagnosis, management, and prognostic evaluation of ICM (Figure 1).

Ventricular volume and function assessment

CMR is considered the gold standard for quantifying left ventricular volumes, mass, and ejection fraction.[4] Cine imaging with balanced steady-state free precession (bSSFP) sequences provides excellent blood-myocardium contrast, enabling accurate tracing of endocardial and epicardial borders.

Typically, cine sequences include contiguous short-axis slices spanning from the base to the apex of the heart, supplemented by long-axis views (two-, three-, and four-chamber).

Standard slice thickness is around 8 mm with minimal spacing, and temporal resolution is ≤40 ms. Manual or semi-automated contouring at end-diastole and end-systole enables calculation of stroke volume, ejection fraction, and myocardial mass.

Notably, papillary muscles are generally considered part of myocardial mass, not cavity volume.[8]

Myocardial tissue characterization: differentiating ICM from NICM

A major strength of CMR is its ability to non-invasively characterize myocardial tissue. Through a combination of parametric mapping (T1, T2, T2*) and contrast-enhanced imaging, CMR can detect edema, inflammation, fibrosis, infiltration, and iron deposition.

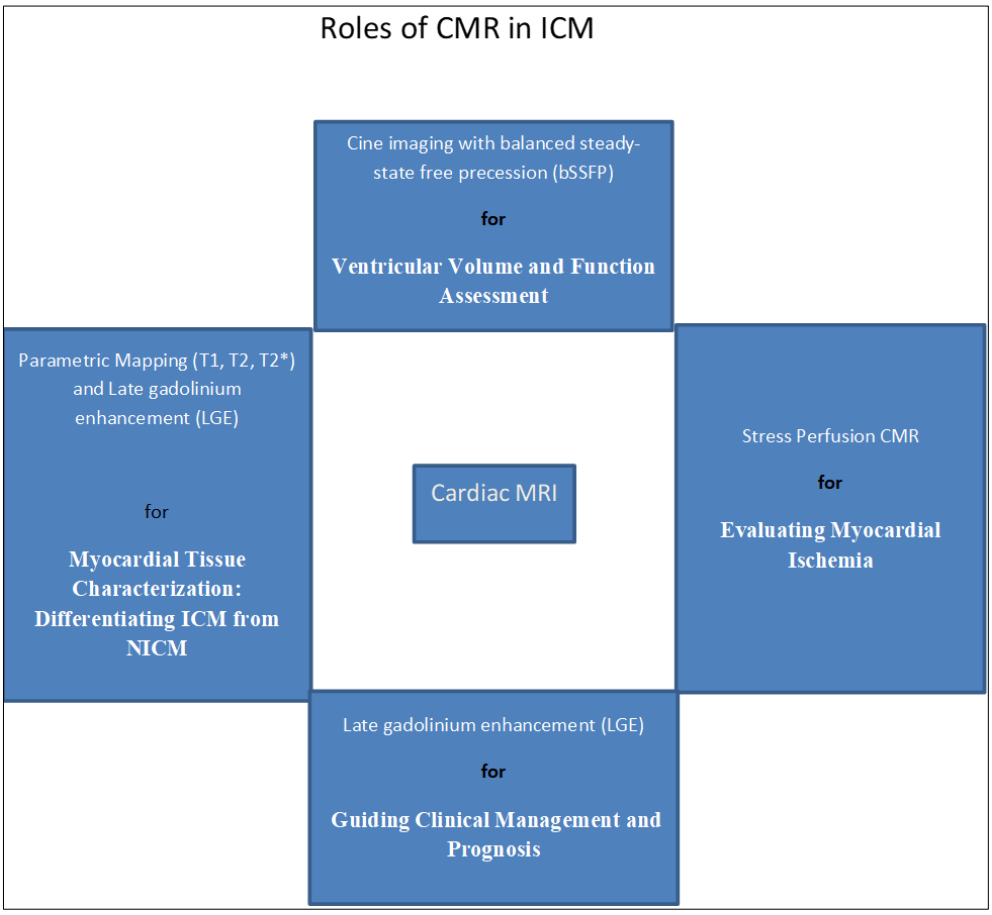

Figure 1: Summary of the 4 roles of CMR in ICM.

Quantitative mapping techniques provide voxel-wise numeric data, significantly enhancing tissue characterization—especially valuable in patients with arrhythmias or diffuse myocardial involvement that may be missed by conventional methods.[9]

In acute infarction, native T1 and T2 values are elevated due to edema and inflammation, helping distinguish recent from chronic injury and identify the "area at risk." Chronic ischemic damage, by contrast, shows fibrosis and scar formation.[10]

LGE imaging is pivotal in differentiating ischemic from non-ischemic patterns. Ischemic scars typically appear as subendocardial or transmural enhancements conforming to coronary artery territories, while non-ischemic scars often display mid-wall, epicardial, or patchy distributions that do not follow vascular anatomy (Figure 2).[11]

Traditional cardiac MRI techniques used for myocardial tissue characterization have some important drawbacks. Image quality can often be suboptimal, particularly with STIR T2-weighted sequences, especially in patients who have fast or irregular heart rates or who struggle to hold their breath during the scan. In addition, LGE imaging requires contrast administration and may not reliably detect diffuse myocardial involvement, as it is better suited for identifying focal fibrosis rather than widespread subtle changes.[4,12]

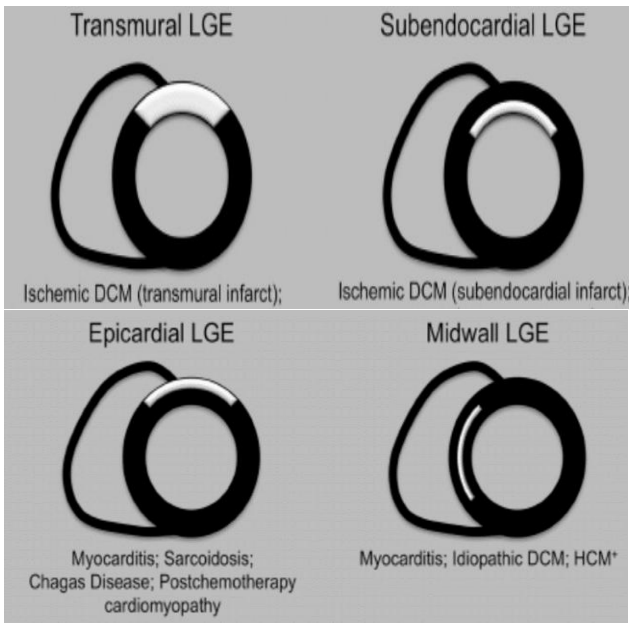

Figure 2: Schematic illustration: LGE patterns in ischemic versus non-ischemic cardiomyopathy.

Stress perfusion CMR: evaluating myocardial ischemia

Stress perfusion CMR provides insight into dynamic ischemia by simulating stress conditions pharmacologically (e.g., adenosine, regadenoson). After vasodilator administration, a gadolinium-based contrast is injected, and first-pass perfusion imaging is performed. Ischemic regions appear as transient perfusion defects during stress, which resolve at rest.[2]

Approximately 10–15 minutes post-contrast, LGE sequences are acquired. If perfusion defects align with areas of LGE, the tissue is considered non-viable. In contrast, viable myocardium lacks LGE despite showing reversible perfusion deficits—information that can guide revascularization decisions.[2,4]

An alternative is dobutamine stress CMR, used when vasodilators are contraindicated. Dobutamine raises heart rate and contractility, and inducible wall motion abnormalities can be visualized using the standard 17-segment model.[2,4]

Numerous studies confirm the high diagnostic yield of stress CMR. The CE-MARC trial, for instance, demonstrated superior sensitivity of stress CMR over single photon emission computed tomography (SPECT) (Table 1).[13] A meta-analysis by Haberkorn et al also found CMR to outperform dobutamine stress echocardiography (DSE) in sensitivity (Table 2).[14]

Table 1: Comparison between the sensitivity and specificity of stress CMR and SPECT in detection of ischemia in patient suspected to have IHD.

Parameter	Stress CMR	SPECT
Sensitivity	85.6%	66.5%
Specificity	Specificity 83·2% (79·1–86·6)	Specificity 82·6% (78·5–86·1)

Table 2: Meta-analysis by Haberkorn comparing the sensitivity and specificity of stress CMR TO DSE in detection of ischemia in patients suspected to have IHD.

Parameter	Stress CMR	DSE
Sensitivity	0.88 (95% CI 0.85-0.9)	0.72 (95% CI 0.61-0.80)
Specificity	0.84 (95% CI 0.81-0.87)	0.89 (95% CI 0.83-0.93)

In recognition of these findings, recent guidelines assigned stress CMR a class IIb recommendation for assessing ischemia and viability prior to revascularization.[15]

Guiding clinical management and prognosis

LGE imaging not only aids in diagnosis but also plays a central role in therapeutic planning and outcome prediction. Areas of LGE reflect myocardial necrosis or fibrosis, representing regions of gadolinium accumulation due to expansion of the extracellular space.[4] The extent and transmurality of LGE are strongly associated with the likelihood of functional recovery following revascularization. Myocardial segments demonstrating more than 50% transmural scar are generally unlikely to recover contractile function, whereas segments with minimal or absent LGE are more likely to demonstrate functional improvement after successful intervention.[4,16]

Myocardial viability assessment plays an important role in the management of ischemic cardiomyopathy by identifying dysfunctional but potentially recoverable myocardium that may benefit from revascularization. Historically, observational studies suggested that revascularization of viable myocardium is associated with improved clinical outcomes. However, contemporary randomized evidence has been less definitive. For example, the STICH trial did not demonstrate a significant interaction between myocardial viability status and survival benefit following coronary artery bypass grafting in patients with ischemic cardiomyopathy. These findings suggest that viability testing should not be used in isolation to determine revascularization strategy but should be integrated into a comprehensive clinical decision-making framework that considers symptom burden, coronary anatomy, ventricular function, comorbidities, and procedural risk.[17]

Beyond viability assessment, LGE provides important prognostic information. Increased total scar burden and the presence of heterogeneous border-zone fibrosis are associated with higher risks of ventricular arrhythmias, sudden cardiac death, and major adverse cardiovascular events. Furthermore, LGE can help predict response to cardiac resynchronization therapy and other device-based management strategies.[18]

In acute coronary syndromes, the combined use of T2-weighted imaging and LGE sequences enables identification of the ischemic “area at risk” and the extent of myocardial salvage after reperfusion, thereby improving prognostic stratification.[4]

More recently, speckle-tracking principles have been applied to CMR using standard cine balanced steady-state free precession (bSSFP) images, a technique known as CMR feature tracking (CMR-FT). This method quantifies myocardial deformation by tracking voxel motion throughout the cardiac cycle, enabling calculation of longitudinal, circumferential, and radial strain without the need for additional imaging sequences or contrast administration. In ICM, CMR-FT has several important roles. First, by objectively identifying reduced global and longitudinal strain, it can detect early subendocardial dysfunction. Second, by quantifying blunted strain augmentation during intermediate- or high-dose dobutamine stress, it improves diagnostic accuracy beyond visual wall-motion analysis for the detection of inducible

ischemia. Third, during low-dose dobutamine stimulation, CMR-FT facilitates the identification of viable but dysfunctional (hibernating or stunned) myocardium by demonstrating improvement in longitudinal or circumferential strain parameters, reflecting contractile reserve. Thus, CMR-FT provides a quantitative, reproducible, and sequence-independent approach for detecting inducible ischemia and assessing myocardial viability in patients with suspected or established ischemic cardiomyopathy.[19,20]

CONCLUSION

While cardiac magnetic resonance imaging does have logistical and patient-related limitations, it remains unmatched in its ability to deliver a comprehensive, non-invasive evaluation of ischemic cardiomyopathy. CMR enables precise quantification of cardiac volumes and function, robust detection of inducible ischemia, and detailed tissue characterization that distinguishes ischemic from non-ischemic pathology. Most notably, LGE imaging offers critical insights that inform treatment decisions and predict long-term outcomes.

From initial diagnosis to therapeutic guidance and prognosis, CMR stands out as an essential tool across the clinical continuum of ischemic cardiomyopathy.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Khair LA. Cardiac magnetic resonance in ischemic cardiomyopathy: from tissue characterization to prognostic stratification. Int J Res Med Sci 2026;14:2162-7.