Interpreting cardiac MRI LGE patterns: a way out of the labyrinth

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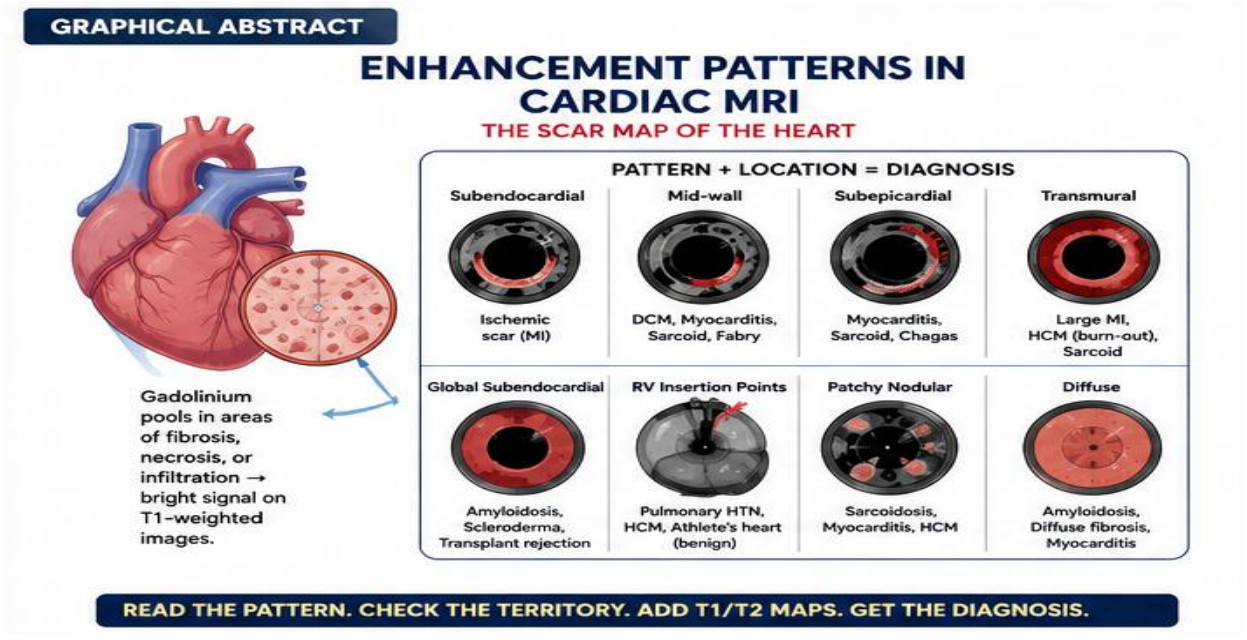
***Abstract*—Late gadolinium enhancement (LGE) in Cardiac Magnetic Resonance (CMR) imaging serves as the myocardial "scar map," utilizing gadolinium pooling in expanded interstitial space (fibrosis, necrosis, or infiltration) to achieve a diagnosis. The primary diagnostic distinction rests on separating ischemic LGE, which must involve the subendocardium and follow a coronary territory, from non-ischemic LGE, which typically spares the subendocardium or begins mid-wall/subepicardially. Core non-ischemic patterns include the mid-wall septal stripe (Dilated Cardiomyopathy), patchy subepicardial involvement (Myocarditis), and global subendocardial enhancement (Amyloidosis). Diagnosis is further refined by pattern-specific "kickers," such as the difficulty in nulling the myocardium in Amyloidosis (blood pool nulls before myocardium) or the patchy nodular lesions favoring the basal septum in Sarcoidosis. Advanced techniques like T1 mapping with Extracellular Volume (ECV) are essential for quantifying diffuse fibrosis (ECV >40% in Amyloid), while T2/STIR imaging helps determine injury chronicity (edema on T2/STIR suggests acute injury). Ultimately, the pattern's location and amount (e.g., >15% LV LGE in DCM/HCM) are crucial prognostic determinants related to outcomes like sudden cardiac death (SCD) risk.**

***Index Terms*—Magnetic Resonance Imaging; Late Gadolinium Enhancement; Cardiac Magnetic Resonance; Myocardial Fibrosis; T1 Mapping; T2 Mapping; Extracellular Volume Fraction; Myocardial Infarction; Myocarditis; Amyloidosis; Sarcoidosis; Fabry Disease; Arrhythmogenic Right Ventricular Cardiomyopathy; Hypertrophic Cardiomyopathy; Dilated Cardiomyopathy; Myocardial Perfusion Imaging; Tissue Characterization; Prognosis.**



I. INTRODUCTION: WHY ENHANCEMENT MATTERS IN CMR

Late gadolinium enhancement LGE and early gadolinium enhancement EGE exploit the wash-in/wash-out kinetics of extracellular gadolinium contrast. Normal myocardium has tight myocytes with little interstitial space, so gadolinium washes out fast. Fibrosis, necrosis, infiltration, or edema expand the interstitial space causing gadolinium to pool. This generates a bright signal on T1-weighted inversion recovery sequences.

Diagnosis is based on a combination of pattern recognition and site of enhancement. The basic interpretation is determined by identifying a "regional differential tissue map or pattern" of the heart.

II. THE CORE ENHANCEMENT PATTERNS

The cornerstone of interpreting late gadolinium enhancement (LGE) in CMR is establishing whether the pattern is ischemic (territorial, subendocardial-involving) or non-ischemic (non-territorial, mid-wall or subepicardial-sparing). The tables below synthesize this differentiation, with the first table outlining the fundamental rules for distinguishing the two main categories, including key prognostic indicators like Microvascular Obstruction (MVO). The second table then organizes the core enhancement patterns, correlating location with specific diseases and highlighting the critical "pearls"—such as the poor nulling characteristic of Amyloidosis or the basal septum involvement in Sarcoidosis—that enable a high-confidence diagnosis.

III. ISCHEMIC LGE

As a rule ischemia always involves subendocardium and follows a coronary territory.

IV. WAVEFRONT PHENOMENON

Progression is as follows (with increasing grades of ischemia): Up to 25% subendocardial, followed by up to 50% subendocardial, up to 75% subendocardial, followed by 100% (i.e. transmural). Sub-endocardial in the early stages is potentially reversible.

V. TERRITORIES

LAD corresponds to anterior/septum/apex.

RCA corresponds to inferior wall, inferoseptum and RV.

LCx corresponds to lateral wall.

Microvascular obstruction or MVO corresponds to dark core within bright infarct. This implies worse prognosis in acute infarcts. A thrombus represented on imaging by a non-enhancing dark mass usually at LV apex also portends a grave prognosis.

Ischemic and Non-Ischemic LGE Differentiation – first port of call

Feature	Ischemic LGE (MI Scar)	Non-Ischemic LGE
Location Rule	Always involves the subendocardium, progressing transmural	Spares the subendocardium, or starts mid-wall/epicardial
Territory	Follows a specific coronary artery territory	Doesn't respect coronary territories
Progression	Wavefront phenomenon (20% subendo → 100% transmural as infarct ages)	Linear (DCM), Patchy (Myocarditis), or Multifocal (Sarcoid)
Prognosis Marker	Microvascular obstruction (MVO) is a dark core within bright infarct, signaling bad prognosis	Amount of LGE (>15% in DCM or HCM) indicates a higher SCD risk

VI. NON-ISCHEMIC LGE

Rule: Spares subendocardium, or starts mid-wall/epicardial. Doesn't necessarily follow coronary territories. Most important patterns are myocarditis, HCM and DCM.

1. Myocarditis: Subepicardial and inferolateral more commonly than septal.

Lake Louise Criteria: LGE with T2 edema and hyperemia (on perfusion imaging).
2. HCM: Patchy mid-wall at RV insertion points with anywhere hypertrophied. Amount of LGE corresponds to sudden cardiac demise risk.
3. DCM: Mid-wall septal stripe (linear and non-nodular).

Characteristic "tissue signature" can help discern between common diagnoses. Recognition of these characteristic distributions, combined with T1/T2 mapping and functional assessment, enables accurate differentiation of infiltrative, inflammatory, genetic, and arrhythmogenic cardiomyopathies. As in other scenarios, endo-myocardial biopsy remains the reference standard in all non-ischemic conditions (provided the myocardial thickness remains adequate). Amyloidosis typically produces diffuse subendocardial or transmural enhancement with abnormal myocardial nulling, while sarcoidosis manifests as patchy multifocal scars involving the basal septum and lateral wall. Myocarditis characteristically demonstrates subepicardial or mid-wall enhancement in the inferolateral wall, particularly when associated with myocardial edema. Anderson–Fabry disease shows a distinctive basal inferolateral mid-wall pattern accompanied by low native T1 values, whereas ARVC primarily affects the RV free wall with fibro-fatty replacement and LGE.

Disease-Specific Late Gadolinium Enhancement (LGE) Patterns in Cardiac MRI

Disease	Typical LGE Pattern	Predilection Site	Key Imaging Pearls
Amyloidosis	Global subendocardial enhancement progressing to diffuse transmural enhancement in advanced disease	Circumferential LV involvement; atrial walls often enhance	Difficult myocardial nulling on inversion recovery imaging; blood pool nulls before myocardium (classic sign); markedly elevated extracellular volume (ECV >40%)
Cardiac Sarcoidosis	Patchy, multifocal, nodular ("punched-out") enhancement	Basal interventricular septum and lateral LV wall; RV involvement common	Active disease shows LGE with concomitant T2 edema; often corresponds with increased FDG uptake on PET imaging
Myocarditis	Subepicardial or mid-wall enhancement, non-coronary distribution	Inferolateral wall most common, followed by septum	Acute myocarditis demonstrates LGE plus myocardial edema on T2/STIR imaging; chronic myocarditis shows persistent LGE without edema
Anderson–Fabry Disease	Mid-wall enhancement	Basal inferolateral LV wall	Characteristically low native T1 values before contrast due to sphingolipid accumulation; presence of LGE indicates advanced myocardial involvement
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)	LGE involving RV free wall with fibro-fatty replacement	RV free wall; LV involvement may occur in left-dominant variants	Diagnosis strengthened by combination of regional wall-motion abnormalities, fat signal on T1-

			weighted imaging, and LGE; high specificity when fat and fibrosis coexist

VII. BEYOND LGE

While late gadolinium enhancement (LGE) remains the cornerstone of myocardial tissue characterization, several complementary CMR techniques provide additional diagnostic and prognostic information. Early gadolinium enhancement (EGE) performed 1–3 minutes after contrast administration helps differentiate avascular thrombus from vascularized masses and identifies microvascular obstruction, with thrombi remaining characteristically non-enhancing. T1 mapping and extracellular volume (ECV) quantification detect diffuse interstitial expansion and fibrosis that may be invisible on conventional LGE imaging, making them particularly valuable in infiltrative cardiomyopathies such as amyloidosis. T2 mapping and STIR imaging assess myocardial edema, allowing distinction between acute and chronic injury; the coexistence of LGE and elevated T2 signal indicates active inflammation or recent injury, whereas isolated LGE usually reflects chronic fibrosis or scar. First-pass perfusion imaging, especially during pharmacologic stress, evaluates myocardial blood flow and can identify inducible subendocardial perfusion defects indicative of ischemia even in the absence of infarction. Together, these techniques provide a comprehensive assessment of myocardial structure, inflammation, fibrosis, viability, and perfusion.

Tabulated Précis of Complementary CMR Enhancement Techniques

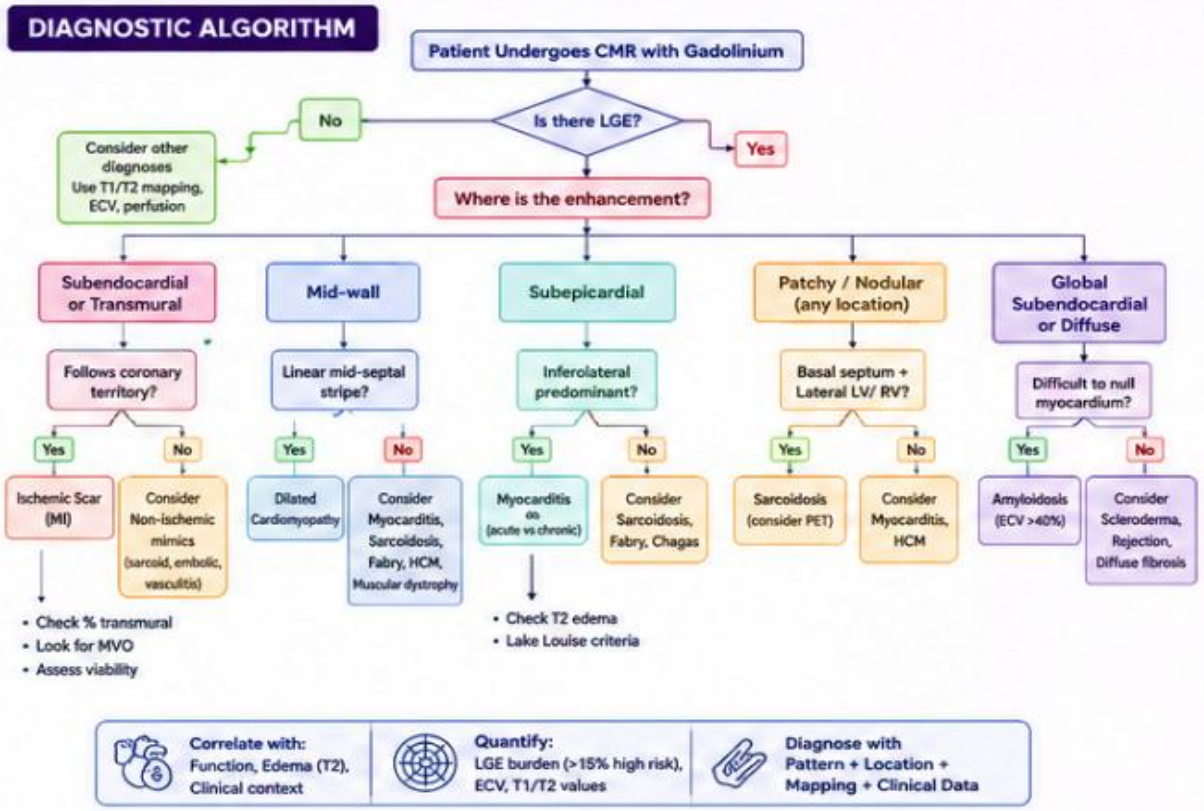
Technique	Timing / Principle	Primary Role	Characteristic Findings	Clinical Applications
Early Gadolinium Enhancement (EGE)	1–3 minutes after contrast administration	Assessment of vascularity and microvascular integrity	Thrombus remains dark; tumors enhance; microvascular obstruction is hypoenhancing	Differentiation of thrombus from tumor; detection of MVO after myocardial infarction
T1 Mapping + Extracellular Volume (ECV)	Quantitative tissue characterization before and after contrast	Detection of diffuse fibrosis and infiltration	Normal ECV: 25–28%; diffuse fibrosis: 30–35%; amyloidosis: >40%	Amyloidosis, diffuse fibrosis, cardiomyopathies, longitudinal disease monitoring
T2 Mapping / STIR Imaging	Quantitative or qualitative edema imaging	Detection of myocardial edema and active inflammation	Bright T2 signal indicates edema; LGE + T2 = acute injury; LGE without T2 = chronic scar	Acute myocarditis, acute MI, sarcoidosis activity assessment, inflammatory cardiomyopathies
First-Pass Perfusion Imaging	Dynamic imaging during contrast transit, often with stress	Assessment of myocardial perfusion	Reversible subendocardial perfusion defect indicates inducible ischemia	Coronary artery disease, ischemia evaluation, microvascular dysfunction, viability assessment

Figures depicting the patterns and a diagnostic algorithm is presented in the following page.

TYPES AND CAUSES

TYPE OF ENHANCEMENT	CMR IMAGE (Short-axis)	TYPICAL LOCATION	MAJOR CAUSES / ASSOCIATED DISEASES	KEY FEATURES
Subendocardial		Subendocardium → progresses transmural	Ischemic heart disease, Myocardial infarction	Follows coronary artery territory. Wavefront phenomenon. MVO = dark core.
Mid-wall		Mid-myocardium, spares endo/epi	Dilated cardiomyopathy, Myocarditis, Sarcoid, Fabry, Muscular dystrophy	Septal "mid-wall stripe" = classic DCM. Linear, not territorial.
Subepicardial		Outer 1/3 (subepicardial)	Myocarditis, Sarcoid, Anderson-Fabry disease, Chagas disease	Often lateral/inferolateral wall in myocarditis. Patchy.
Transmural		Full thickness (endo to epi)	Large MI, HCM (burn-out), Sarcoidosis	>50% wall thickness = likely non-viable post-MI.
Global Subendocardial		Circumferential subendocardium	Amyloidosis, Systemic sclerosis, Transplant rejection	Amyloid: poor nulling, blood pool dark, myocardium brighter.
RV Insertion Points		Anterior / Inferior RV-LV junction	Pulmonary hypertension, HCM, Athlete's heart	Mechanical stress, not necrosis. Benign if isolated.
Patchy Nodular		Random, often RV too	Sarcoidosis, Myocarditis, HCM	"Hot spots". Basal septum + lateral LV common in sarcoid.
Diffuse		Entire myocardium	Amyloidosis, Diffuse fibrosis, Myocarditis	Best quantified with T1 mapping / ECV, not just LGE.

DIAGNOSTIC ALGORITHM

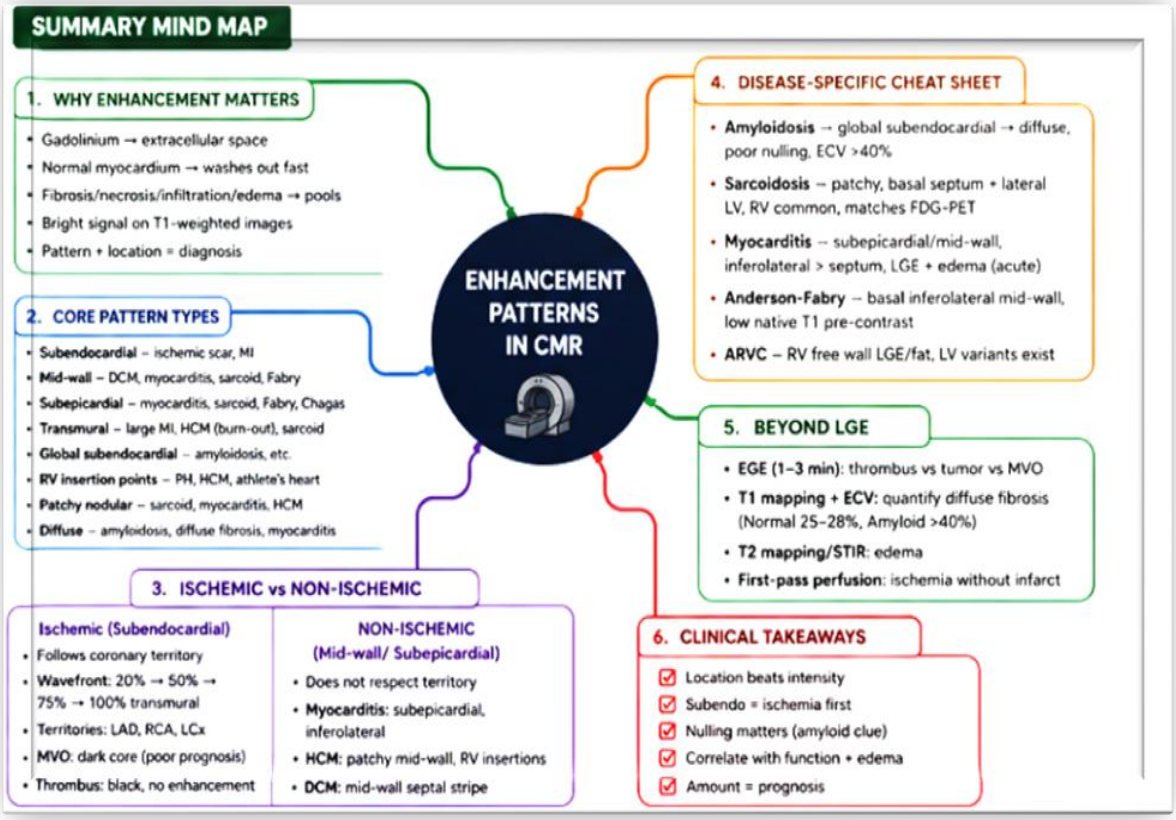


VIII. CLINICAL TAKEAWAYS

1. Location beats intensity: Subendo corresponds to think ischemia first. Mid-wall/epi corresponds to think non-ischemic.
2. Nulling matters: If you can't null the myocardium, think amyloid.
3. Correlate with function with edema: LGE with hypokinetic with no edema corresponds to chronic MI. LGE with edema with normal motion corresponds to myocarditis.
4. Amount corresponds to prognosis: >15% LV LGE in DCM or HCM corresponds to higher SCD risk. Transmural MI corresponds to non-viable.

IX. CONCLUSION

Enhancement patterns in CMR are the fingerprint of myocardial disease. The rule of thumb remains: Read the pattern, check the territory, add T1/T2 maps, and you've possibly gotten a biopsy without being cardio-invasive. A summary mind map is presented in the following figure.



ACKNOWLEDGEMENTS

Sri Siba Prasad Patra and Smt Ruma Das for unwavering support. Our team member who has taken a sabbatical Mrs. Sima Patra (Bhandari) for suggestions over the phone.

AI was used exhaustively for compilation of this manuscript although the finer points were abstracted and made presentable by the authors.
Meta AI was used for a preliminary framework while ChatGPT was used for compilation of conceptual images. Gemini 3.1 flash (Google, ABC Inc.) was used for all the editing and simplification to enable mass comprehension. Initial edits were performed on

Google Docs. Final document compilation was performed on M365 platform.

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