Dijlah Journal of Medical Sciences (DJMS) Vol. 3, No. 3, September, 2026, pp. 57-69
P-ISSN: 3078-3178, E-ISSN: 3078-8625, paper ID: 06

ORIGINAL ARTICLE

Between Stenosis and Scar: A 1.5T CMR Study Using MOLLI T1 Mapping and PSIR-LGE in Iraqi CAD Patients

Zamazam Hussein Shummar*, Mariwan Jalal-Tawfeek and Imad Kh. Resen
Department of Radiology Technologies, College of Health and Medical Technologies, Dijlah University, Baghdad, Iraq

Article Info

Article history:

Received June, 01, 2026
Revised June, 23, 2026
Accepted June, 30, 2026

Keywords:

Cardiac Magnetic Resonance Imaging,
MOLLI,
PSIR-LGE,
T1 Mapping,
Extracellular Volume,
Myocardial Scar,
Coronary Artery Disease,
1.5 Tesla

ABSTRACT

This study aimed to assess the agreement between MOLLI T1 mapping and PSIR-LGE for myocardial scar assessment at 1.5T in Iraqi patients with coronary artery disease confirmed by coronary CT angiography. This prospective pilot study included 33 adult patients with significant coronary stenosis or complete occlusion on CCTA. All patients underwent cardiac MRI at 1.5T. The protocol included cine imaging, native and post-contrast MOLLI T1 mapping, extracellular volume calculation, and PSIR-LGE. MOLLI was considered abnormal when native T1, ECV, or both were elevated. Agreement between MOLLI and PSIR-LGE was assessed at patient level and segment level using the AHA 17-segment model. CMR showed myocardial scar in 26 of 33 patients. Seven patients had no visible scar on PSIR-LGE, despite having significant coronary disease on CCTA. PSIR-LGE identified scar in all 26 scar-positive patients, while MOLLI showed abnormal native T1 and/or ECV in 25 of them. Patient-level agreement was strong, with $\kappa = 0.85$, $p < 0.001$. At segment level, PSIR-LGE identified 84 scar-positive segments, and MOLLI showed corresponding abnormality in 81 segments, with $\kappa = 0.82$, $p < 0.001$. In the scar-negative subgroup, mean native T1 was 982 ± 24 ms and mean ECV was 27.1 ± 2.4%. The best visual myocardial nulling on PSIR-LGE was observed at inversion times of about 300–320 ms. MOLLI T1 mapping showed strong agreement with PSIR-LGE for myocardial scar assessment at 1.5T. PSIR-LGE remains more suitable for direct visual scar detection, while MOLLI adds quantitative tissue information through native T1 and ECV. The findings also show that significant coronary stenosis on CCTA does not always mean visible myocardial scar on CMR. Larger local studies are needed to validate Iraqi 1.5T T1 and ECV values and to refine PSIR-LGE acquisition practice.

Corresponding Author:

* Zamazam Hussein Shummar
Department of Radiology Technologies, College of Health and Medical Technologies, Dijlah University, Baghdad, Iraq
Email: zamzamsh91@gmail.com

1- INTRODUCTION

Cardiac magnetic resonance imaging (CMR) has become an important method for myocardial tissue characterization, not only because it shows cardiac anatomy and function, but because it can also describe the tissue itself. This point is important in coronary artery disease (CAD), where the anatomical information alone may not be enough. Coronary CT angiography (CCTA) can show coronary stenosis, vessel narrowing, plaque, and luminal

morphology in a very detailed way. Still, it cannot always answer the more difficult clinical question: did the downstream myocardium already develop irreversible injury, or not? CMR gives this missing part by assessing myocardial scar, fibrosis, and interstitial expansion using late gadolinium enhancement and quantitative mapping techniques [1]. The Society for Cardiovascular Magnetic Resonance describes myocardial T1 mapping and extracellular volume (ECV) measurement as valuable methods for myocardial tissue evaluation, especially when traditional imaging are insufficient to elucidate the underlying disease process. [2]. Later clinical recommendations also emphasized that T1 mapping, T2 mapping, T2* mapping, and ECV should not be treated as simple image sequences only. They are quantitative measurements, and for this reason they need careful acquisition, interpretation, and local validation [3]. This is not a small technical detail. In practice, the measured value can change because of the field strength, pulse sequence, scanner vendor, reconstruction method, and even patient-related factors. So, using one universal normal value for all centers is not safe.

T1 mapping makes the technical problem obvious. Kellman and Hansen noted that accuracy and precision in cardiac T1 mapping are impacted by various sequence-related parameters, including heart rate sensitivity, magnetisation transmission, inversion efficiency, and mobility. [4]. Because of this, normal cardiac T1 levels should be taken with caution. International multicenter research has demonstrated that reference values differ between locations and techniques, even when healthy people are investigated [5]. This was also shown at 3T, where myocardial T1 and T2 values were influenced by technical and biological factors [6]. More recent studies continued to show the same concern: normal values are not completely transferable from one population, scanner, or protocol to another [7–9]. For a country such as Iraq, where 1.5T systems are commonly used but local CMR mapping data are still limited, this problem becomes more practical, not just theoretical. Late gadolinium enhancement remains the clinically accepted approach for diagnosing focal myocardial scar. The famous work of Kim et al. revealed that contrast-enhanced MRI may diagnose irreversible cardiac damage and aid differentiates viable from non-viable myocardium [10]. Simonetti et al. then improved infarct visualization using an inversion-recovery technique that increased contrast between scarred and normal myocardium [11]. Phase-sensitive inversion recovery late gadolinium enhancement (PSIR-LGE) further strengthened this approach, because it reduced the dependence on perfect inversion time selection and made scar detection more reliable in routine clinical imaging [12]. This is why PSIR-LGE is still considered the reference standard for focal myocardial scar assessment. Nonetheless, PSIR-LGE isn't flawless. It works quite well for focused scars, particularly ischaemic scars, although it is primarily visual or semi-quantitative. It may overlook diffuse myocardial involvement because diffuse disease may not distinguish between aberrant and distant myocardium. MOLLI T1 mapping provides a different sort of information. It generates pixel-level quantitative T1 values before and after contrast. ECV may be determined by combining post-contrast T1 and haematocrit measurements. ECV represents enlargement of the myocardial extracellular space, which may occur with fibrosis, infarction, edema-related alterations, or other cardiac pathologies based on the clinical situation [13].
So, PSIR-LGE and MOLLI should not be seen as two competing methods in a simple way. PSIR-LGE answers one strong question: is there a visible focal scar? MOLLI and ECV answer a different question: is the tissue quantitatively abnormal, and how much interstitial expansion may be present? In CAD patients, both questions matter. A patient may have significant coronary stenosis on CCTA but no visible scar on PSIR-LGE. Another patient may show scar clearly, while MOLLI provides additional quantitative information about the involved and remote myocardium. This combination may be useful, especially in centers trying to build their own 1.5T CMR reference experience.

The present pilot study was therefore designed to evaluate the agreement between MOLLI-derived T1/ECV abnormalities and PSIR-LGE scar detection at 1.5T in Iraqi patients with CCTA-confirmed CAD. The study also aimed to generate preliminary local T1 and ECV values in scar-negative CAD patients and to assess whether MOLLI can serve as an adjunctive quantitative tool beside PSIR-LGE in routine local CMR practice.

2- MATERIALS AND METHODS

2.1 Study design

This prospective pilot observational research was conducted in the hospitals of Baghdad Medical City, Baghdad, Iraq, between July 2025 and March 2026. The institutional review board provided ethical approval, and the study followed the principles of the Declaration of Helsinki. All individuals provided written informed permission before to inclusion. A total of 33 adult patients with known coronary artery disease (CAD) were prospectively recruited. In this investigation, CAD was defined as the presence of hemodynamically substantial coronary stenosis or full coronary blockage on coronary CT angiography (CCTA) conducted during the preceding

four weeks. CCTA was used only as an anatomical inclusion criterion. The actual myocardial tissue assessment was done by cardiac magnetic resonance imaging (CMR), not by CCTA. Patients were excluded if they had standard contraindications to MRI, severe claustrophobia, markedly impaired renal function, non-diagnostic CMR image quality, or refusal to provide informed consent. Markedly impaired renal function was defined as an estimated glomerular filtration rate (eGFR) below 30 mL/min/1.73 m², in line with commonly used contrast safety thresholds for patients at higher risk when gadolinium-based contrast administration is considered [14]. Baseline clinical data and left ventricular ejection fraction (LVEF) were recorded for all enrolled patients.

2.2 CMR Protocol and T1 Mapping

Cardiac MRI was accomplished on a 1.5 Tesla scanner utilizing a customized phased array cardiac coil. The procedure includes cine balanced steady state free precession (bSSFP) pictures to evaluate ventricular function, native MOLLI T1 mapping, post-contrast MOLLI T1 mapping and phase sensitive inversion recovery late gadolinium enhancement (PSIR-LGE). These sequences are chosen because they are frequently utilized in clinical CMR for evaluation of function, T1-tissue characterization, and myocardial scar [2,3]. Gadolinium-based contrast was given intravenously at a dose of 0. 1 mmol/kg, after renal function had been checked. Patients with eGFR below 30 mL/min/1.73 m² were not included, according to contrast safety recommendations used for higher-risk patients [14]. PSIR-LGE images were acquired about 10–15 minutes after contrast injection. Post-contrast T1 mapping was performed at approximately 15 minutes, using the same short-axis levels as far as possible. For MOLLI T1 mapping, the 5(3)3 scheme was used in most patients, as this approach has been reported to provide reliable T1 measurements at 1.5T [4,7]. In some patients, especially when rhythm or heart-rate variation affected image stability, a 4(1)3(1)2 scheme was used instead. The main acquisition parameters were: TR/TE 2.8/1.1 ms, flip angle 35°, slice thickness 8 mm, and receiver bandwidth 930 Hz/pixel. PSIR-LGE was used for scar assessment because it is less dependent on exact inversion-time selection than conventional inversion recovery imaging and is well established for detecting myocardial infarction and focal scar [10,12].

2.3 Image and Segmental Analysis

Two radiologists experienced in cardiac MRI assessed the studies independently. The PSIR-LGE and MOLLI datasets were initially read independently as much as the local process permitted. At this point each reader was kept blind to the result of the other series. This was done to minimize direct visual bias, even if total blinding was not always straightforward in a small clinical sample. Segmental myocardial examination was done according to the American Heart Association 17-segment model [15]. Segments having substantial motion artefact, inadequate ECG triggering or non-diagnostic picture quality were omitted from segment-level analysis. PSIR-LGE was employed as the reference technique for localised myocardial scar. A segment was defined as scar-positive if there was localised myocardial hyperenhancement in a coronary arterial area, rather than a random or nonischemic pattern. This criterion was based on the known usage of LGE and PSIR-LGE for detection of infarction and focal scar [10–12]. Also, myocardial nulling was examined in an exploratory manner. PSIR pictures were evaluated at many inversion times between 200 and 350 ms. This component was not an individual diagnostic end point. It was added largely to see how inversion-time variation affects visual scar contrast and myocardial background suppression in our local 1.5T context. For quantitative analysis, regions of interest were placed manually on the MOLLI maps. ROIs were drawn in the interventricular septum and, when present, in LGE-positive myocardial regions. Care was taken to avoid the blood pool, papillary muscles, trabeculations, and obvious partial-volume areas. Native T1 and post-contrast T1 values were recorded for myocardium and blood pool. ECV was then calculated using myocardial and blood T1 values together with the same-day hematocrit value, following the standard approach described for CMR-based ECV assessment [1,2,13]. Only seven patients were scar-negative. T1 and ECV levels in this subgroup are thus considered as preliminary local values, not as definitive diagnostic cutoffs. This is relevant because one cannot safely create a normal reference range from a tiny scar-negative population. Inter-reader agreement was tested using Cohen’s kappa coefficient for categorical scar detection and intraclass correlation coefficients for quantitative T1 and ECV measures [16,17].

2.4 Statistical Analysis

Continuous variables were tested for normality using the Shapiro–Wilk test. Data are provided as mean ± SD if normally distributed, or median (interquartile range) if not normally distributed. Categorical variables are presented as frequency and percentage. Cohen's kappa was used to evaluate the agreement between MOLLI T1 mapping and PSIR-LGE at the patient and segment levels. MOLLI's diagnostic performance indices (sensitivity, specificity, positive and negative predictive value) were calculated using the 2x2 contingency tables. The discriminating ability was further characterised by receiver operating characteristic (ROC) curve analysis. Potential

confounds of heart rate and body mass index on map quality were also examined. Statistical significance was determined as a two-sided p-value <0.05. All analyses were performed using SPSS version 27.0.

3- RESULTS AND DISCUSSION

3.1 Baseline Characteristics

Thirty-three persons with coronary artery disease verified by CCTA were included in the final analysis. The average age was 59.3 ± 8.7 years (range, 45 to 74 years). There were twenty-two participants, 66.7% of them were male. Common cardiovascular risk factors were hypertension in 18 individuals (54.5%), diabetes mellitus in 14 participants (42.4%), dyslipidaemia in 19 participants (57.6%), and active or past smoking in 10 people (30.3%). Nine patients (27.3%) had a history of prior myocardial infarction. The mean body mass index was 29.4 ± 3.8 kg/m2 showed a substantial number of individuals were overweight or obese. Mild to severe impairment of left ventricular systolic function was noted with a mean left ventricular ejection fraction of 47.8 ± 8.1%. Overall, the trial group constituted a clinically relevant CAD cohort with a significant burden of metabolic and cardiovascular risk factors, rather than a low-risk screening population. The whole baseline profile is provided in Table 1.

Table (1): Baseline characteristics of the study population

Variable	Value
Number of participants	33
Age, years	59.3 ± 8.7
Age range, years	45–74
Male sex	22 (66.7%)
Hypertension	18 (54.5%)
Diabetes mellitus	14 (42.4%)
Dyslipidemia	19 (57.6%)
Smoking	10 (30.3%)
Previous myocardial infarction	9 (27.3%)
Body mass index, kg/m²	29.4 ± 3.8
BMI range, kg/m²	23.1–36.8
Left ventricular ejection fraction, %	47.8 ± 8.1
LVEF range, %	30–60

Values are presented as mean ± standard deviation or number (%)

3.2 CCTA Stenosis versus CMR-Detected Myocardial Scar

All 33 enrolled participants demonstrated significant coronary arterial stenosis or occlusion on CCTA, thereby fulfilling the predefined anatomical criterion for CMR tissue characterization. Myocardial scarring was identified in 26 participants (78.8%) on the basis of PSIR-LGE findings and/or concordant MOLLI parametric abnormalities. Conversely, the remaining 7 participants (21.2%) exhibited no CMR evidence of myocardial scar, notwithstanding the presence of anatomically confirmed coronary stenosis on CCTA. This mismatch between anatomical and tissue findings is clinically important. CCTA can show the luminal narrowing in the epicardial coronary arteries, but CMR gives another type of information, which is the myocardial tissue response after this narrowing. Therefore, the absence of scar in these seven patients does not mean that CCTA was false positive. It may simply mean that the stenosis did not lead to irreversible myocardial damage. This can be explained by collateral perfusion, non-significant hemodynamic effect of the lesion, or previous revascularization. So, the two techniques should not be seen as duplicated tests. In practice, they answer different questions, and their combination gives a more complete assessment of CAD.

Table (2): Relationship between CCTA-confirmed CAD and CMR-detected myocardial scar

Finding	Number of participants	Percentage
CCTA-confirmed coronary stenosis or occlusion	33/33	100%
CMR-detected myocardial scar	26/33	78.8%
CCTA stenosis without detectable CMR scar	7/33	21.2%

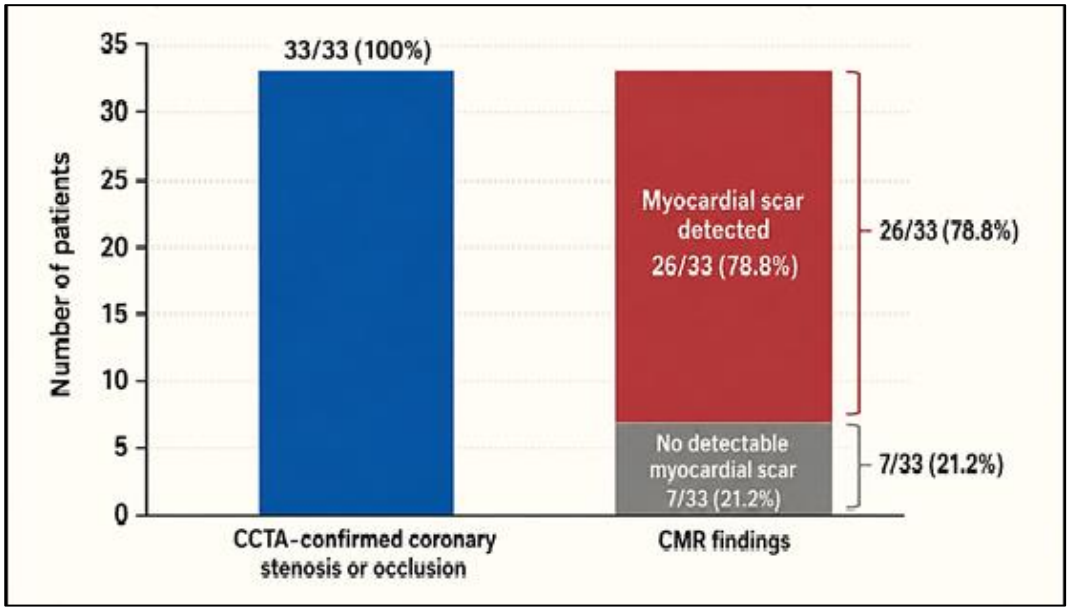

Fig (1): CCTA stenosis versus CMR-Detected Myocardial Scar

All patients had significant coronary stenosis or occlusion on CCTA; CMR detected Myocardial Scar in 26 patients and no detectable scar in 7 patients.

3.3 Patient-Level Agreement between MOLLI and PSIR-LGE

Among the 26 participants in whom CMR identified myocardial scarring, PSIR-LGE detected the scar in all 26 cases, while MOLLI T1 mapping revealed abnormal native T1 and/or ECV values in 25 of these individuals. The single discordant case — PSIR-LGE positive with normal MOLLI parameters — most plausibly reflects either a limited scar volume falling below the sensitivity threshold of quantitative mapping or subendocardial partial-volume averaging inherent to the spatial resolution of the technique. At the patient level, concordance between MOLLI and PSIR-LGE was strong, yielding a Cohen's kappa of 0.85 ($p < 0.001$). With PSIR-LGE serving as the CMR reference standard for focal scar detection, MOLLI demonstrated near-complete agreement for scar-positive classification. It is nonetheless important to contextualize this finding appropriately: the observed concordance reflects strong methodological agreement rather than diagnostic equivalence, given that PSIR-LGE functioned as the comparator modality rather than an independent biological or histopathological gold standard.

Table (3): Patient-level comparison between MOLLI and PSIR-LGE

Parameter	**Value**
PSIR-LGE scar-positive participants	26
MOLLI-positive among PSIR-positive participants	25
MOLLI-negative among PSIR-positive participants	1
Cohen’s kappa	0.85
p-value	<0.001

PSIR-LGE was used as the reference CMR method for focal scar detection

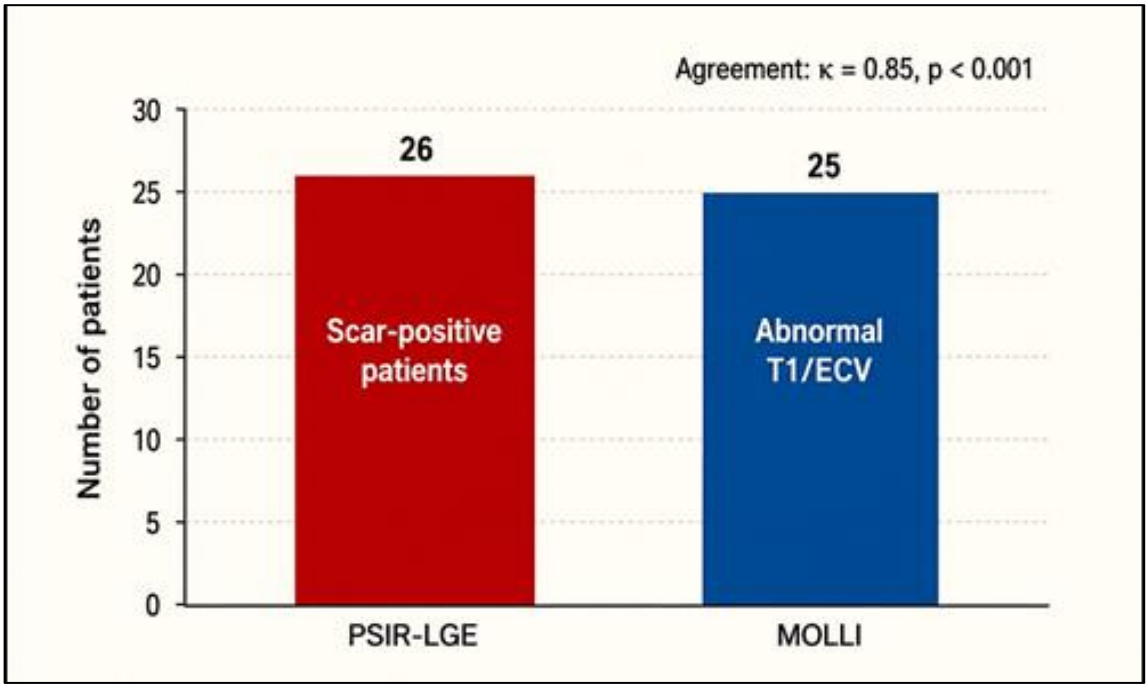

Fig (2): Patient Level Agreement between MOLLI and PSIR-LGE

PSIR-LGE detected myocardial scar in 26 patients, while MOLLI showed corresponding abnormal T1/ECV values in 25 patients.

3.4 Segment-Level Agreement

For the segment analysis, we used the AHA 17-segment left ventricular model. In all the analyzed heart segments, PSIR-LGE found 84 scar-positive segments. MOLLI showed abnormal native T1 or ECV values in 81 of these positive segments. The other three segments that were positive on PSIR-LGE did not show clear abnormalities on MOLLI. At the segment level, the agreement between MOLLI and PSIR-LGE was also very strong, with a Cohen's kappa value of 0.82. ($p < 0.001$). This finding suggests that MOLLI abnormalities were spatially concordant with focal scar distribution on PSIR-LGE in an appreciable amount affected myocardial segments. The small number of discordant segments may reflect limited scar size, subendocardial partial-volume effects, motion-related misregistration, or threshold limitations in quantitative mapping.

Table (4): Segment-level comparison between MOLLI and PSIR-LGE

Analysis level	PSIR-LGE positive segments	MOLLI-positive corresponding segments	Agreement κ	p-value
AHA 17-segment model	84	81	0.82	<0.001

Segments with severe artifact or inadequate myocardial coverage should be excluded and reported separately if present.

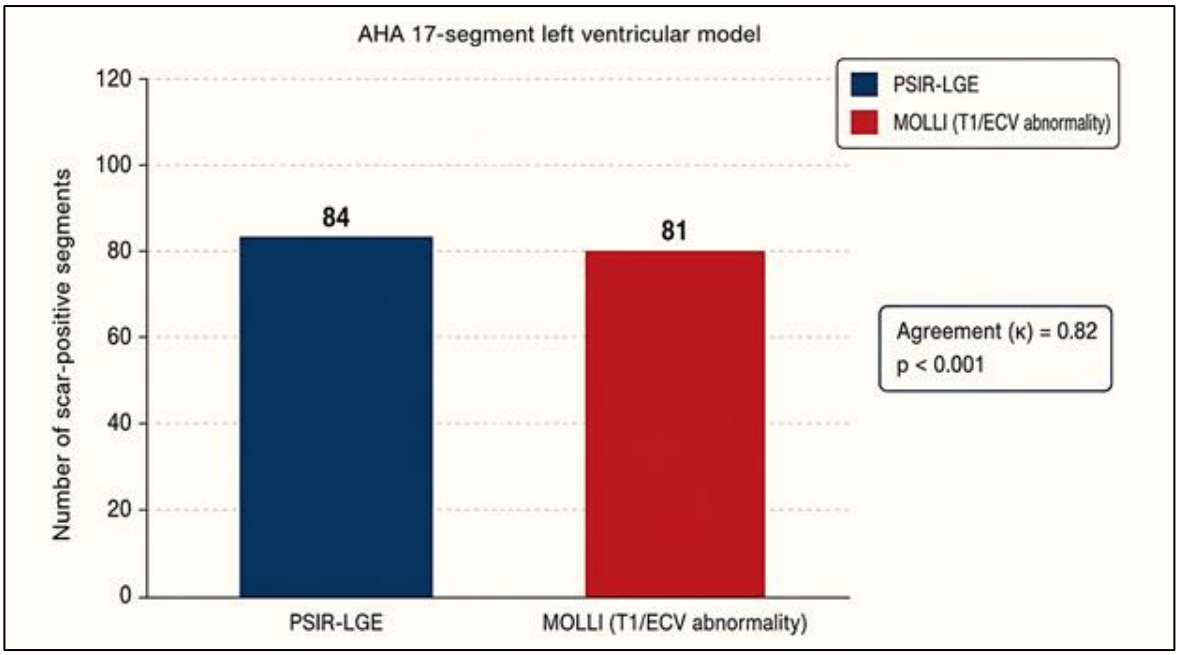

Fig (3): Segment-Level Agreement between MOLLI and PSIR-LGE

PSIR-LGE identified 84 scar-positive segments. MOLLI demonstrated abnormal native T1 and/or ECV values in 81 corresponding segments. Agreement between the two methods was high (K=0.82, P<0.001). analysis performed using the AHA 17-segment left ventricular model.

3.5 Preliminary Native T1 and ECV Estimates in Scar-Negative CAD Participants

In the seven participants without detectable myocardial scar on CMR, MOLLI-derived values were relatively consistent. The mean native myocardial T1 value was 982 ± 24 ms, with a range of 950–1018 ms. The mean post-contrast T1 value performed the function of 457 ± 21 ms, with a range of 430–489 ms. The mean ECV was 27.1 ± 2.4%, with a range of 24.5–30.3%. These values should be interpreted as preliminary local estimates in scar-negative Iraqi CAD participants rather than definitive population reference ranges. The sample was small, and the participants were not healthy volunteers. Still, these values provide a useful starting point for local 1.5T CMR interpretation and future validation studies.

Table (5): Preliminary MOLLI T1 and ECV estimates in scar-negative CAD participants

Parameter	**Mean ± SD**	**Range**
Native T1, ms	982 ± 24	950–1018
Post-contrast T1, ms	457 ± 21	430–489
ECV, %	27.1 ± 2.4	24.5–30.3

Values are derived from seven scar-negative CAD participants when viewed in conjunction with should not be interpreted as definitive normal reference ranges.

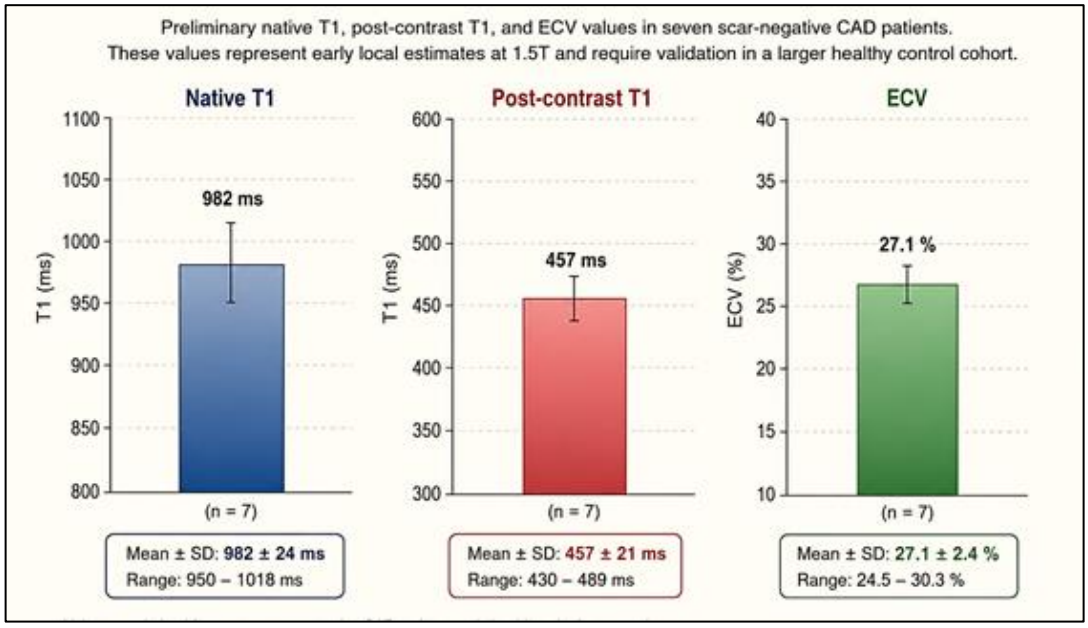

Fig (4): Preliminary MOLLI T1 and ECV Estimates in Scar-Negative CAD Patients

Values are derived from seven scar-negative CAD patients and should not be interpreted as definitive normal reference ranges.

3.6 Exploratory TI Optimization Finding

Exploratory PSIR-LGE acquisitions at a variety of inversion periods demonstrated a consistent link between TI choice and quality of myocardial nulling in this group. Shorter TI values of 200 and 250 ms were linked with partial suppression of seemingly normal myocardium and reduced scar-to-myocardium contrast, therefore limiting reliable scar delineation. TI values of around 300–320 ms consistently resulted in optimal myocardial nulling and more robust scar conspicuousness. Acquisitions beyond this range, at around 350 ms, showed slight over-nulling artefacts in certain situations. Taken together, our findings show that TI selection may require site-specific tuning in 1.5T CMR practice, particularly for persons with high BMI or changed myocardial relaxation qualities due to underlying disease. However, these data should be considered hypothesis-generating and taken with appropriate caution, because this investigation was exploratory rather than a fully powered technical validation trial.

Table (6): Exploratory PSIR inversion time assessment

TI tested, ms	Visual image quality	Myocardial nulling	Interpretation
200	Poor	Incomplete	Residual normal myocardial signal
250	Moderate	Acceptable	Usable but suboptimal contrast
300	Good	Optimal	Improved scar-to-myocardium contrast
320	Very good	Optimal	Best visual nulling in this cohort
350	Good	Mild over-nulling	Possible reduction in tissue contrast

Image quality was assessed visually as part of exploratory protocol optimization

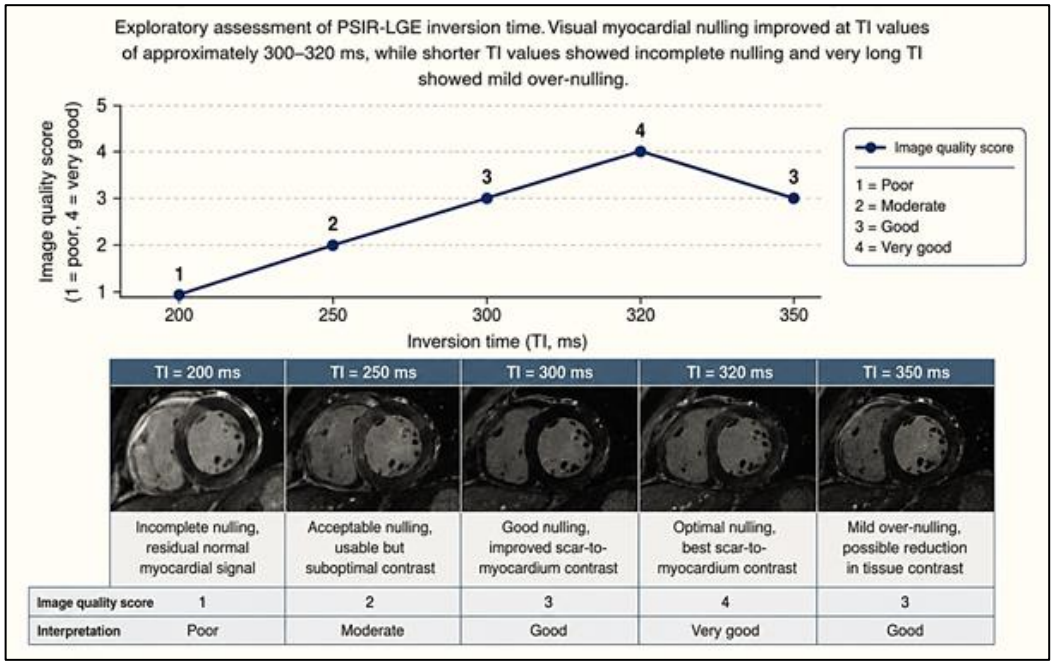

Fig (5): Effect of Inversion Time on PSIR-LGE Image Quality

In this prospective pilot study, we tried to see how much MOLLI T1 mapping agrees with PSIR-LGE for myocardial scar assessment at 1.5T in Iraqi patients who already had CAD confirmed by CCTA. The results showed a few points, and maybe the first one is the most clinically important. Significant coronary stenosis on CCTA did not always mean that scar was present on CMR. This is not strange. CCTA shows the artery. It tells us about stenosis, occlusion, plaque, and the lumen. But it does not directly tell us what happened to the myocardium after that narrowing. In our study, all patients had significant stenosis or occlusion, but only 26 of 33 patients showed myocardial scar by CMR. This gap between coronary anatomy and myocardial tissue finding is important. Previous work in CAD has shown that native T1 and ECV of even non-infarcted myocardium may carry prognostic and pathophysiological information, beyond the visible LGE scar itself [18].

PSIR-LGE was used as the reference method for focal scar in this study. This is reasonable because LGE is still the strongest visual method for replacement fibrosis and myocardial infarction scar. In our results, PSIR-LGE identified scar in 26 patients. The value of this technique is that it shows focal hyperenhancement, usually following coronary arterial distribution in ischemic injury. It is easy to understand clinically. There is scar, or no scar. There is subendocardial involvement, maybe transmural, maybe not. And this matters for viability and revascularization decisions. Still, even with its power, LGE is essentially a visual or semi-quantitative approach. That depends on the contrast difference between aberrant and distant myocardium. In case of diffuse illness the contrast gap may shrink.
Additional information was obtained using MOLLI T1 mapping. Of the 26 PSIR-LGE-positive individuals in this investigation, 25 had native T1 and/or ECV abnormalities by MOLLI. Agreement was good both at patient level ($\kappa = 0.85$) and at segment level ($\kappa = 0.82$). That suggests the aberrant parametric values were not just floating about as random integers on the map. They mainly corresponded to the same regions of scar seen by PSIR-LGE. This is consistent with earlier studies that have demonstrated that native T1 mapping may identify infarcted myocardium and correlate with infarct size, however its performance may change between acute and chronic infarction. [19,20].
The one discordant case, where PSIR-LGE was positive but MOLLI did not show clear abnormality, should not be over-read. In small studies, one case can look dramatic, but clinically it may be explained by a simple technical reason. The scar may have been too small. It may have been subendocardial and affected by partial-volume averaging. Or the MOLLI slice may not have matched the PSIR-LGE slice exactly. Motion and triggering problems can also reduce confidence in T1 maps. This single mismatch does not make MOLLI weak. It only reminds us that MOLLI is not a replacement for visual scar imaging. It is a quantitative adjunct. The practical value of MOLLI is that it gives numbers. Native T1 and ECV can describe myocardial tissue in a way that visual LGE cannot always do. In infarction, ECV has been shown to add useful information to LGE for predicting functional recovery after acute myocardial infarction [21]. This matters because scar is not only a shape on the image. It is tissue injury,

extracellular expansion, fibrosis, and sometimes edema or microvascular injury depending on timing. In reperfused acute infarction, T1 mapping has also been studied in relation to microvascular obstruction and LGE findings, which again supports the idea that mapping and LGE are connected but not identical methods [22]. This is why the result should be read carefully. A high agreement between MOLLI and PSIR-LGE is encouraging, but it does not mean both techniques are the same. PSIR-LGE is better when the question is focal replacement scar. MOLLI is better when the question is quantitative tissue change. In many CAD patients, both questions exist together. A patient can have a visible scar and also abnormal remote myocardium. Another patient can have no visible scar but still have mildly abnormal T1 or ECV, especially if there is hypertension, diabetes, obesity, chronic ischemic exposure, or diffuse fibrosis. Studies of remote myocardium after infarction support that non-infarcted tissue may also change and may relate to remodeling [23,24].

Shorter TI values gave incomplete nulling, and longer values sometimes produced mild over-nulling. This is useful for local protocol adjustment, but it is not enough to create a fixed recommendation. It was a visual assessment, and the sample was small. A proper TI optimization study would need blinded scoring, contrast-to-noise measurements, and probably repeated testing across different heart rates and contrast timing. Still, as a practical observation, 300–320 ms may be a reasonable starting range for our local 1.5T setting. Another interesting point is that newer work has tried to generate synthetic LGE-like images from mapping data, and this supports the broader movement toward quantitative scar imaging [25]. But this does not remove the need for real PSIR-LGE, especially in a clinical CAD population. Native T1 mapping and ECV are powerful, but they have limits. They can be affected by technical noise, ROI placement, blood pool contamination, and partial-volume averaging. Meta-analytic data also support the value of T1 mapping for myocardial fibrosis, but they show variation across methods and disease groups [26,27]. So, in daily clinical work, the safest interpretation is combined reading: PSIR-LGE for scar pattern, MOLLI/ECV for quantitative tissue characterization. Our scar-negative category consisted of just seven individuals. Mean native T1 was 982 ± 24 ms and mean ECV was 27.1 ± 2.4% These figures should not be regarded as usual Iraqi reference values. That would be too strong, and not right, honestly. They're CAD sufferers, not healthy volunteers. Also, lack of LGE does not imply absence of entire cardiac abnormalities. So the appropriate description is preliminary local data from scar-negative CAD patients. Small yet necessary. It's a beginning point. It does not provide a diagnostic cut-off. This local issue is important in Iraq. Most CMR work is done on 1.5T systems, and local mapping validation is still limited. T1 and ECV values change with scanner type, field strength, sequence design, timing after contrast, heart rate, and analysis method. Even ECV calculation can be influenced by the contrast protocol, including bolus-only versus infusion approaches [28].

This study has certain drawbacks. First of all, it was a preliminary trial with just 33 individuals. This is sufficient to demonstrate feasibility and early agreement, but not to construct final reference values or strong diagnostic cutoffs. Secondly, it was performed at one single local site and on one 1.5T scanner. The results may not be immediately applicable to different scanners, manufacturers or mapping methods. Third, PSIR-LGE was employed as the CMR reference technique, but no histology, radionuclide viability imaging or long term clinical outcome follow-up was available. This is a frequent drawback in CMR scar investigations, but it does matter. Fourth, ROI was manually placed on the MOLLI maps. Small defects can be made with care, especially around the blood pool, papillary muscles, trabeculations, and thin subendocardial scars. The slice mismatch between PSIR-LGE and MOLLI may also impair segmentation agreement. The group without scars was relatively small, comprising 7 patients. Thus their T1 and ECV values should be seen as early local estimations rather than typical values. Also these patients still had CAD. They were not controls. Finally, the TI analysis was exploratory and mostly focused on visual quality, will have to be tested later with objective measures of contrast, and larger numbers.

4- CONCLUSION

MOLLI T1 mapping showed strong agreement with PSIR-LGE for myocardial scar assessment at 1.5T in Iraqi patients with CCTA-confirmed CAD. PSIR-LGE remains the main method for identifying focal ischemic scar, especially when the scar pattern and transmural extent are clinically important. MOLLI adds a different value. It gives quantitative native T1 and ECV data, which may reflect tissue abnormality beyond what visual LGE alone can show. The study also shown that CCTA-defined coronary stenosis does not always indicate myocardial scar seen by CMR. This supports the role of CMR as a tissue level test after anatomical coronary imaging. Preliminary scar-negative T1 and ECV values may be useful for future local work but should not be utilized as final reference ranges. Larger multicenter Iraqi trials, including healthy controls, standardized MOLLI collection, and outcome follow-up and more formal PSIR-LGE optimization are needed.

REFERENCES

[1] Vaikom House, A., and Grosse-Wortmann, L. (2025). Cardiac magnetic resonance imaging assessment of myocardial disease in children and adolescents. Cardiovascular Diagnosis and Therapy, 15(4), 888–897. https://doi.org/10.21037/cdt-24-502

[2] Messroghli, D. R., Moon, J. C., Ferreira, V. M., Grosse-Wortmann, L., He, T., Kellman, P., et al. (2017). Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2 and extracellular volume: A consensus statement by the Society for Cardiovascular Magnetic Resonance endorsed by the European Association for Cardiovascular Imaging. *Journal of Cardiovascular Magnetic Resonance, 19*, 75. https://doi.org/10.1186/s12968-017-0389-8

[3] Kellman, P., and Hansen, M. S. (2014). T1-mapping in the heart: Accuracy and precision. *Journal of Cardiovascular Magnetic Resonance, 16*, 2. https://doi.org/10.1186/1532-429X-16-2

[4] Dabir, D., Child, N., Kalra, A., Rogers, T., Gebker, R., Jabbour, A., et al. (2014). Reference values for healthy human myocardium using a T1 mapping methodology: Results from the International T1 Multicenter Cardiovascular Magnetic Resonance Study. *Journal of Cardiovascular Magnetic Resonance, 16*, 69. https://doi.org/10.1186/s12968-014-0069-x

[5] Gottbrecht, M., Kramer, C. M., and Salerno, M. (2019). Native T1 and extracellular volume measurements by cardiac MRI in healthy adults: A meta-analysis. *Radiology, 290*(2), 317–326. https://doi.org/10.1148/radiol.2018180226

[6] von Knobelsdorff-Brenkenhoff, F., Prothmann, M., Dieringer, M. A., Wassmuth, R., Greiser, A., Schwenke, C., et al. (2013). Myocardial T1 and T2 mapping at 3 T: Reference values, influencing factors and implications. *Journal of Cardiovascular Magnetic Resonance, 15*, 53. https://doi.org/10.1186/1532-429X-15-53

[7] Krumm, P., Nikolaou, K., Bamberg, F., Klumpp, B., Fenchel, M., Mangold, S., et al. (2022). T1 mapping MOLLI 5(3)3 acquisition scheme yields high accuracy in 1.5T cardiac magnetic resonance. *Diagnostics, 12*(11), 2729. https://doi.org/10.3390/diagnostics12112729

[8] Xu, Z., Li, W., Huang, Y., Liu, X., Zhang, Q., Jiang, L., et al. (2023). Reference ranges of myocardial T1 and T2 mapping in healthy Chinese adults: A multicenter 3T cardiovascular magnetic resonance study. *Journal of Cardiovascular Magnetic Resonance, 25*, 69. https://doi.org/10.1186/s12968-023-00974-5

[9] Gonçalves, A., Moutinho, E., Santos, A., and Teixeira, T. (2024). Myocardial native T1 mapping at 3T cardiac magnetic resonance: Closing the full-vendor reporting cycle of normal values. *Clinical Radiology, 79*, 473–478. https://doi.org/10.1016/j.crad.2024.02.013

[10] Kim, R. J., Wu, E., Rafael, A., Chen, E. L., Parker, M. A., Simonetti, O. P., et al. (2000). The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *New England Journal of Medicine, 343*(20), 1445–1453. https://doi.org/10.1056/NEJM200011163432003

[11] Simonetti, O. P., Kim, R. J., Fieno, D. S., Hillenbrand, H. B., Wu, E., Bundy, J. M., et al. (2001). An improved MR imaging technique for the visualization of myocardial infarction. *Radiology, 218*(1), 215–223. https://doi.org/10.1148/radiology.218.1.r01ja50215

[12] Kellman, P., Arai, A. E., McVeigh, E. R., and Aletras, A. H. (2002). Phase-sensitive inversion recovery for detecting myocardial infarction using gadolinium-delayed hyperenhancement. *Magnetic Resonance in Medicine, 47*(2), 372–383. https://doi.org/10.1002/mrm.10051

[13] Ugander, M., Oki, A. J., Hsu, L. Y., Kellman, P., Greiser, A., Aletras, A. H., et al. (2012). Extracellular volume imaging by magnetic resonance imaging provides insights into overt and subclinical myocardial pathology. *European Heart Journal, 33*(10), 1268–1278. https://doi.org/10.1093/eurheartj/ehr481

[14] Weinreb, J. C., Rodby, R. A., Yee, J., Wang, C. L., Fine, D., McDonald, R. J., et al. (2021). Use of intravenous gadolinium-based contrast media in patients with kidney disease: Consensus statements from

the American College of Radiology and the National Kidney Foundation. *Radiology, 298*(1), 28–35. https://doi.org/10.1148/radiol.2020202903

[15] Cerqueira, M. D., Weissman, N. J., Dilsizian, V., Jacobs, A. K., Kaul, S., Laskey, W. K., et al. (2002). Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation, 105*(4), 539–542. https://doi.org/10.1161/hc0402.102975

[16] McHugh, M. L. (2012). Interrater reliability: The kappa statistic. *Biochemia Medica, 22*(3), 276–282.

[17] Koo, T. K., and Li, M. Y. (2016). A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *Journal of Chiropractic Medicine, 15*(2), 155–163. https://doi.org/10.1016/j.jcm.2016.02.012

[18] Puntmann, V. O., Carr-White, G., Jabbour, A., Yu, C. Y., Gebker, R., Kelle, S., et al. (2018). Native T1 and ECV of noninfarcted myocardium and outcome in patients with coronary artery disease. *Journal of the American College of Cardiology, 71*(7), 766–778. https://doi.org/10.1016/j.jacc.2017.12.020

[19] Dastidar, A. G., Harries, I., Pontecorboli, G., Bruno, V. D., De Garate, E., Moret, C., et al. (2019). Native T1 mapping to detect extent of acute and chronic myocardial infarction: Comparison with late gadolinium enhancement technique. *International Journal of Cardiovascular Imaging, 35*(3), 517–527. https://doi.org/10.1007/s10554-018-1467-1

[20] Aquaro, G. D., Monastero, S., Todiere, G., Barison, A., De Gori, C., Grigoratos, C., et al. (2023). Diagnostic Role of Native T1 Mapping Compared to Conventional Magnetic Resonance Techniques in Cardiac Disease in a Real-Life Cohort. *Diagnostics (Basel, Switzerland)*, 13(14), 2461. https://doi.org/10.3390/diagnostics13142461

[21] Kidambi, A., Motwani, M., Uddin, A., Ripley, D. P., McDiarmid, A. K., Swoboda, P. P., et al. (2017). Myocardial extracellular volume estimation by CMR predicts functional recovery following acute MI. *JACC: Cardiovascular Imaging, 10*(9), 989–999. https://doi.org/10.1016/j.jcmg.2016.06.015

[22] Shin, J. M., Choi, E. Y., Park, C. H., Han, K., and Kim, T. H. (2020). Quantitative T1 mapping for detecting microvascular obstruction in reperfused acute myocardial infarction: Comparison with late gadolinium enhancement imaging. *Korean Journal of Radiology, 21*(8), 978–986. https://doi.org/10.3348/kjr.2019.0736

[23] Biesbroek, P. S., Amier, R. P., Teunissen, P. F. A., Hofman, M. B. M., Robbers, L. F. H. J., van de Ven, P. M., et al. (2017). Changes in remote myocardial tissue after acute myocardial infarction and its relation to cardiac remodeling: A CMR T1 mapping study. *PLoS ONE, 12*(6), e0180115. https://doi.org/10.1371/journal.pone.0180115

[24] Robbers, L. F. H. J., Nijveldt, R., Beek, A. M., Teunissen, P. F. A., Hollander, M. R., Biesbroek, P. S., et al. (2018). The influence of microvascular injury on native T1 and T2* relaxation values after acute myocardial infarction: Implications for non-contrast-enhanced infarct assessment. *European Radiology, 28*(2), 824–832. https://doi.org/10.1007/s00330-017-5010-x

[25] Abdula, G., Nickander, J., Sörensson, P., Lundin, M., Kellman, P., Sigfridsson, A., et al. (2018). Synthetic late gadolinium enhancement cardiac magnetic resonance for diagnosing myocardial scar. *Scandinavian Cardiovascular Journal, 52*(3), 127–132. https://doi.org/10.1080/14017431.2018.1449960

[26] Kaolawanich, Y., Boonyasirinant, T., Limprasert, N., and Krittayaphong, R. (2022). Native T1 mapping for the diagnosis of myocardial fibrosis in patients with chronic myocardial infarction. *JACC: Cardiovascular Imaging, 15*(12), 2127–2139. https://doi.org/10.1016/j.jcmg.2022.09.011

[27] Marques, M. D., Weinberg, R., Kapoor, S., Ostovaneh, M. R., Ambale-Venkatesh, B., Lima, J. A. C., et al. (2022). Myocardial fibrosis by T1 mapping magnetic resonance imaging: A meta-analysis. *European Heart Journal – Cardiovascular Imaging, 23*(10), 1407–1416. https://doi.org/10.1093/ehjci/jeac010

[28] White, S. K., Sado, D. M., Fontana, M., Banypersad, S. M., Maestrini, V., Flett, A. S., et al. (2013). T1 mapping for myocardial extracellular volume measurement by CMR: Bolus only versus primed infusion technique. *JACC: Cardiovascular Imaging, 6*(9), 955–962. https://doi.org/10.1016/j.jcmg.2013.01.011