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Late Gadolinium Enhancement Granularity Score in Patients Presenting with Heart Failure and Unrecognised Myocardial Infarction

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Abstract

Background/Objectives: We aimed to determine if the concept of a late gadolinium enhancement (LGE) granularity score can be used to help predict outcomes in patients presenting with heart failure of presumed non-ischaemic aetiology. The secondary aim was to discover the prevalence of unrecognised myocardial infarction (MI) within this cohort and assess its association with adverse events. **Methods:** We prospectively recruited patients who had been diagnosed with heart failure (left ventricular ejection fraction (LVEF) <50%) and referred for cardiac magnetic resonance (CMR) assessment. Patients with an established diagnosis of coronary artery disease or known structural or congenital heart disease were excluded. A previously published LGE granularity score was calculated for each patient. **Results:** A total of 737 patients were included. One hundred and forty (19%) were found to have ischaemic LGE and were defined as unrecognised MI. Over a median of 3.4 years of follow-up, patients with unrecognised MI exhibited a higher composite all-cause mortality and heart failure hospitalisation (27.9%) than those with non-ischaemic heart failure (12.9%). Patients with unrecognised MI and a granularity score of greater than seven had the highest risk compared to those with non-ischaemic heart failure even after adjustment for age, sex and LVEF (adjusted hazard ratio 2.85 (1.61–5.05, $p < 0.001$). **Conclusions:** Heart failure patients with unrecognised MI and a higher LGE granularity score had the greatest risk of all-cause mortality or heart failure hospitalisation, independently of age, sex, and LVEF. These findings suggest that the LGE granularity score may improve long-term risk stratification and support individualised management in patients with heart failure.

Keywords: late gadolinium enhancement; granularity; unrecognised myocardial infarction; CMR

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1. Introduction

Cardiovascular magnetic resonance (CMR) is recommended in international guidelines and is utilised widely for the comprehensive assessment of patients presenting with heart failure. In addition to assessing cardiac anatomy and function, CMR allows for tissue characterisation, precise volumetric assessment and the detection of prior myocardial infarction (MI) to understand the underlying aetiology, guide management and assist in patient prognosis [1,2].

Late gadolinium enhancement (LGE) is often used as a prognostic marker, with recent European Society of Cardiology (ESC) guidelines establishing LGE characteristics for several cardiomyopathy phenotypes [3,4]. The presence of LGE has been shown to be a predictor of several adverse outcomes [5–8].

The presence of additional non-ischaemic LGE has been demonstrated to be associated with a higher all-cause mortality in patients with ischaemic cardiomyopathy [9]. In patients with non-ischaemic heart failure, non-ischaemic LGE has been shown to be strongly associated with developing ventricular arrhythmias and sudden cardiac death [10,11].

Recent suggestions have hypothesised that characterising LGE quantitatively may better predict prognosis in patients with ischaemic cardiomyopathy [12]. Unger et al. devised a granularity LGE score that is calculated by using LGE extent, location, transmurality and the presence of additional non-ischaemic LGE in patients with coronary artery disease and left ventricular ejection fraction (LVEF) of under 50% [13]. It has been shown in this cohort of patients to be predictive of all-cause mortality, with the scoring model also exhibiting a higher prognostic value over traditional methods [13].

Unrecognised myocardial infarction (MI), identified on CMR by an ischaemic pattern of LGE, may be detected in patients with heart failure who have no previous clinical diagnosis of myocardial infarction or coronary artery disease. In such patients, unrecognised MI may reveal a previously unsuspected ischaemic contribution to the aetiology of heart failure. Although the LGE granularity score was developed for patients with ischaemic cardiomyopathy, its prognostic value in patients with clinically unrecognised infarction remains uncertain.

We therefore aimed to determine whether the LGE granularity score was associated with clinical outcomes among patients presenting with heart failure who were subsequently found to have MI on CMR. We also compared their outcomes with those of patients in whom CMR demonstrated a non-ischaemic pattern or absence of LGE.

2. Materials and Methods

2.1. Study Population

Participants from the MATCH (MyocArdial Tissue CHaracteristics in patients with heart failure) heart failure registry were prospectively enrolled [14,15]. These patients had been diagnosed with heart failure (ejection fraction $< 50\%$) and referred for CMR assessment, which was performed between February 2018 and April 2024.

Patients were excluded from the initial recruitment if they had a known diagnosis of coronary artery disease (defined as >70% stenosis in a major coronary artery or >50% stenosis of the left main stem on coronary angiogram, previous myocardial infarction (MI), history of coronary revascularisation or typical angina symptoms), structural heart disease (hypertrophic cardiomyopathy, amyloidosis), suspected myocarditis, congenital heart disease or a contraindication to CMR or gadolinium-based contrast.

The study protocol was approved by the National Research Ethics Committee (17/YH/0300 & 20/NW/0326). All patients gave written informed consent.

2.2. Patient Characteristics

Patients underwent clinical assessment on the same day as their CMR. This included confirmation of co-morbidities, current medical therapy, and New York Heart Association (NYHA) functional status.

2.3. Follow-Up

Patients were followed up by review of electronic clinical records. The primary outcome was defined as a composite of all-cause mortality and heart-failure hospitalisation (HFH),

based on the timing of the first event. The two secondary outcomes included the occurrence of HFH or all-cause mortality. Time to endpoint was defined from the date of the CMR study until the occurrence of a primary endpoint or censoring at the end of the follow-up period. Patients without events were censored at the end of the follow-up period. HFH was defined as admission requiring treatment with intravenous diuretics ± intensification of medical therapy.

2.4. CMR Protocol

All CMR studies were conducted on a 3T system (Siemens Magnetom Prisma, Erlangen, Germany).

Patients were advised to avoid caffeine for at least 24 h prior to the study. The study protocol included the following acquisitions: (i) long- and short-axis cine sequences; (ii) motion-corrected bright-blood LGE with phase-sensitive inversion-recovery sequences in both long- and short-axis orientation, with additional dark-blood LGE images in cases where necessary if the presence of a subendocardial scar was uncertain.

A total contrast dose of Gadovist® (Bayer Pharma, Berlin, Germany) was given intravenously at 0.15 mmol/kg.

2.5. Image Analysis

Image processing and analysis were performed using CVI42 software (Version 4.1.2, Circle Cardiovascular Imaging, Calgary, AB, Canada). Manual contouring of endocardial and epicardial borders was performed to obtain volumetric data, and values were indexed to body surface area (BSA). LGE was defined by visual enhancement in at least one segment. Subendocardial enhancement was categorised as ischaemic LGE, and non-ischaemic LGE when the subendocardium was not involved.

2.6. Late Gadolinium Enhancement Score

The previously published granularity scoring system is summarised in Table 1 [13]. The LGE granularity scores were calculated for each patient with unrecognised MI and used a combination of the extent (the number of segments affected as per the AHA 16-segment model), transmurality (visually assessed as either subendocardial ≤ 50%, subendocardial > 50% to <75% or transmural ≥ 75%), the location of LGE as well as the presence of non-ischaemic LGE [13]. Patients were then categorised into the following groups: those with non-ischaemic heart failure without non-ischaemic LGE (NI-LGE-ve), non-ischaemic heart failure with non-ischaemic LGE (NI-LGE+ve), unrecognised MI with a LGE granularity score of 7 or less (Isch-Low), and unrecognised myocardial infarction with a LGE granularity score of greater than 7 (Isch-High) (Figure 1). The threshold of 7 was prespecified based on a previous study that identified this value as the optimal cut-off for prognostic risk stratification, and above this score was defined as intermediate or high [13].

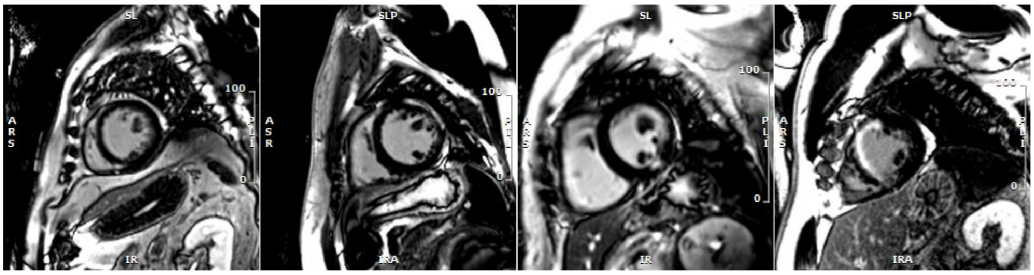


Figure 1. CMR images showing (from left to right)—no LGE, non-ischaemic LGE, low burden of ischaemic LGE, high burden of ischaemic LGE.

Table 1. LGE granularity score.

Ischaemic LGE Extent	+ Number of Segment(s) × 1
Ischaemic LGE Transmurality	
Subendocardial ≤50%	+0
Subendocardial >50% and <75%	+2
Transmural ≥75%	+3
Ischaemic LGE Location	
Posterior, lateral and/or apical	+0
Anterior without septal involvement	+5
Septal	+6
Presence of non-ischaemic-LGE	+2

2.7. Statistical Analysis

Continuous variables were presented as the median with interquartile range and were evaluated with the Mann–Whitney U test. Categorical variables are expressed as frequency (percentage) and compared using the chi-square test. Univariable Cox proportional hazards models were used to assess associations between CMR parameters and the primary and secondary endpoints. Multivariable models were adjusted for age, sex, and baseline LVEF (Model 1), with further adjustment for diabetes, hypertension, NYHA class, and the use of diuretics, SGLT2 inhibitors, and antiplatelet agents (Model 2). Results were reported as hazard ratios with 95% confidence intervals. The proportional hazards assumption was assessed using tests based on scaled Schoenfeld residuals for individual model terms and the overall model.

Kaplan–Meier curves with log-rank test were used to estimate event-free rates for the primary and secondary endpoints. An alternative LGE granularity score cutoff was derived using ROC analysis and the Youden index for an exploratory sensitivity analysis.

Incremental prognostic value was assessed separately for the primary endpoint and all-cause mortality using three nested Cox models containing LVEF alone, LVEF plus unrecognised MI, and LVEF plus unrecognised MI and the continuous LGE granularity score. Models were compared within the same complete-case dataset using likelihood-ratio chi-square tests and Harrell's C-index. Five-year reclassification was assessed using continuous net reclassification improvement (NRI) and integrated discrimination improvement (IDI). Confidence intervals and *p*-values for NRI and IDI were obtained by perturbation resampling.

Univariate and multivariate logistic regressions were used to compare LVEF status (LVEF < 45%) at the time of CMR. Multivariate model 1 was adjusted for age and sex; Model 2 included the same additional clinical covariates as the extended Cox model. Results were reported as odds ratios with 95% confidence intervals.

Each analysis used complete cases for the required variables, without imputation. A *p*-value of <0.05 was considered significant. All analyses were performed using the JMP data analysis software (JMP Version 14.2.0, SAS Institute Inc., NC, USA) and R version 4.5.0 (R Foundation for Statistical Computing, Vienna, Austria) using the survival and survIDINRI packages.

3. Results

3.1. Study Population

A total of 737 patients were included in the analysis. Eight patients were excluded due to amyloidosis ($n = 5$), cardiac sarcoidosis ($n = 2$), and hypertrophic cardiomyopathy ($n = 1$). 140 patients (19%) were found to have ischaemic LGE. The remaining 597 patients (81%) made up the non-ischaemic heart failure group, consisting of 205 with non-ischaemic LGE and 392 without non-ischaemic LGE (Figure 2). Follow-up was complete for all patients, with no patients lost to follow-up.

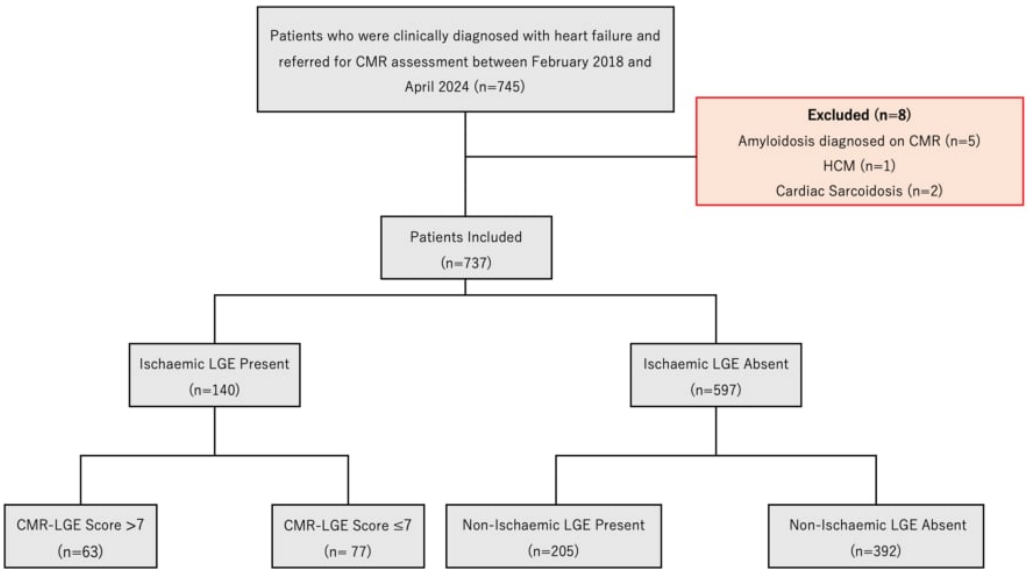


Figure 2. CONSORT diagram.

3.2. Clinical Characteristics and Medication

Baseline characteristics are displayed in Table 2. The median age was 63.3 years (55.0–72.6), with 474 (64.3%) males. Patients with ischaemic LGE were older (68.2 [62.3–75.6] vs. 62.0 [54.1–72.0], $p < 0.001$) and had a higher proportion of males (77.1% vs. 61.3%, $p < 0.001$).

Table 2. Baseline characteristics.

Variable Median [IQR] or *n* (%)	All (*n* = 737)	Unrecognised MI (*n* = 140)	Non-Ischaemic HF (*n* = 597)	*p*
Clinical Parameters				
Age (years)	63.3 (55.0–72.6)	68.2 (62.3–75.6)	62.0 (54.1–72.0)	<0.001
Sex (Male)	474 (64.3)	108 (77.1)	366 (61.3)	<0.001
BMI (kg/m^2)	28.4 (24.7–32.4)	28.4 (24.0–32.0)	28.4 (24.7–32.4)	0.510
Co-morbidities				
Hypercholesterolemia, n (%)	217 (29.5)	47 (33.6)	170 (28.5)	0.239
Diabetes, n (%)	127 (17.2)	33 (23.6)	94 (15.8)	0.027
Hypertension, n (%)	329 (44.7)	76 (54.3)	253 (42.5)	0.011
Current smoking, n (%)	128 (17.4)	31 (22.1)	97 (16.3)	0.103

Table 2. *Cont.*

Variable Median [IQR] or *n* (%)	All (*n* = 737)	Unrecognised MI (*n* = 140)	Non-Ischaemic HF (*n* = 597)	*p*
Medications				
Betablocker, n (%)	606 (82.8)	124 (88.6)	482 (81.4)	0.044
ACEi/ARB, n (%)	562 (76.8)	107 (76.4)	455 (76.9)	0.914
MRA, n (%)	302 (41.3)	63 (45.0)	239 (40.4)	0.317
ARNI, n (%)	97 (13.2)	25 (17.9)	72 (12.1)	0.073
Diuretic, n (%)	315 (43.1)	71 (51.1)	244 (41.2)	0.035
SGLT2i, n (%)	120 (16.4)	31 (22.1)	89 (15.0)	0.039
Anticoagulant, n (%)	236 (32.3)	46 (32.9)	190 (32.2)	0.872
Antiplatelet, n (%)	140 (19.1)	42 (30.0)	98 (16.6)	<0.001
NYHA classification				0.120
1	373 (50.6)	63 (45.0)	310 (51.9)	
2	297 (40.3)	67 (47.9)	230 (38.5)	
3	67 (9.1)	10 (7.1)	57 (9.6)	
NT-proBNP (pg/mL)	574 (164–1651)	1201 (397–2826)	487 (132–1418)	<0.001

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor–neprilysin inhibitor; BMI, body mass index; HF, heart failure; IQR, interquartile range; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association; SGLT2i, sodium-glucose cotransporter 2 inhibitor.

Those with ischaemic LGE had comparable co-morbidities and cardiovascular risk factors compared to the non-ischaemic LGE group, apart from hypertension, which was more common in the ischaemic LGE group (54.3% vs. 42.5%, $p = 0.011$).

Regarding guideline directed medical therapy (GDMT), beta blockers (88.6% vs. 81.4%, $p = 0.044$), diuretics (51.1% vs. 41.2%, $p = 0.035$), sodium-glucose co-transporter 2 inhibitors (SGLT2i) (22.1% vs. 15%, $p = 0.039$) and antiplatelets (30% vs. 16.6%, $p < 0.001$) were more commonly prescribed in the ischaemic LGE group.

3.3. CMR Evaluation

CMR data is summarised in Table 3. Patients with ischaemic LGE had larger left ventricular dimensions indexed left ventricular end diastolic volume (LVEDVi): 113.3 [92.5–145.7] vs. 100.3 [83.4–123.4] mL/m^2, $p < 0.001$; indexed left ventricular end systolic volume (LVESVi): 77.2 [53.2–110.9] vs. 58.4 [42.0–85.0] mL/m^2, $p < 0.001$); higher LV mass index (69.8 [58.8–86.2] vs. 63.1 [52.7–77.2] g/m^2, $p < 0.001$); and a lower LVEF (34 [25.5–44.1] vs. 41.9 [31.5–50]%, $p < 0.001$). Right ventricular end systolic volume (RVESVi) was larger (40.1 (31.3–52.5) vs. 36.7 (27.6–47.9) $p = 0.023$) and RVEF was lower (48.9 (38.2–55.0) vs. 50.6 (42.0–58.6) $p = 0.007$).

3.4. Differences in Clinical Outcomes

Patients were followed up for a median duration of 3.4 years. Overall, there were 116 instances of the primary endpoint (composite of death or heart failure hospitalisation)—with 77 events in patients with non-ischaemic heart failure (12.9%) and 39 events in those with unrecognised myocardial infarction (27.9%).

Table 3. Clinical CMR characteristics.

Variable	All (n = 737)	Unrecognised MI (n = 140)	Non-Ischaemic HF (n = 597)	p
LV EDVi (mL/m^2)	102.5 (84.9–128.2)	113.3 (92.5–145.7)	100.3 (83.4–123.4)	<0.001
LV ESVi (mL/m^2)	60.6 (43.9–86.8)	77.2 (53.2–100.9)	58.4 (42.0–85.0)	<0.001
LVEF (%)	40.5 (30.1–48.9)	34.0 (25.5–44.1)	41.9 (31.5–50.0)	<0.001
LV Mass Index (g/m^2)	64.7 (53.9–78.8)	69.8 (58.8–86.2)	63.1 (52.7–77.2)	<0.001
RV EDVi (mL/m^2)	75.8 (63.3–90.3)	78.5 (65.4–92.1)	75.5 (63.0–89.9)	0.228
RV ESVi (mL/m^2)	37.3 (28.4–48.7)	40.1 (31.3–52.5)	36.7 (27.6–47.9)	0.023
RVEF (%)	50.3 (41.2–58.2)	48.9 (38.2–55.0)	50.6 (42.0–58.6)	0.007
Ischaemic LGE (%)	140 (19.0)	140 (100)	0 (0)	<0.001
Non-ischaemic LGE (%)	221 (30.0)	16 (11.4)	205 (34.3)	<0.001
Native T1 (ms)	1322 (1291–1355)	1326 (1277–1365)	1321 (1293–1354)	0.747
ECV (%)	25.6 (23.3–28.0)	26.0 (23.3–28.1)	25.4 (23.3–28.0)	0.394

Abbreviations: ECV—extracellular volume; EDVi—end-diastolic volume index; ESVi—end-systolic volume index; HF, heart failure, LGE—late gadolinium enhancement; LV—left ventricular; RV—right ventricular.

Kaplan–Meier survival analysis for the different groups aresplayed in Figures 3 and 4. The unrecognised MI group had lower event-free survival compared to the non-ischaemic heart failure group (Figure 3). Unrecognised MI was associated with higher risks of all three outcomes in unadjusted analyses (all $p < 0.001$). These associations persisted after adjustment for age, sex, and baseline LVEF; the HR was 2.01 (95% CI, 1.35–3.02; $p < 0.001$) for the composite of all-cause mortality and HFH, for all-cause mortality, 2.29 (95% CI, 1.42–3.69; $p < 0.001$) and 2.10 (95% CI, 1.15–3.84; $p = 0.016$) for HFH.

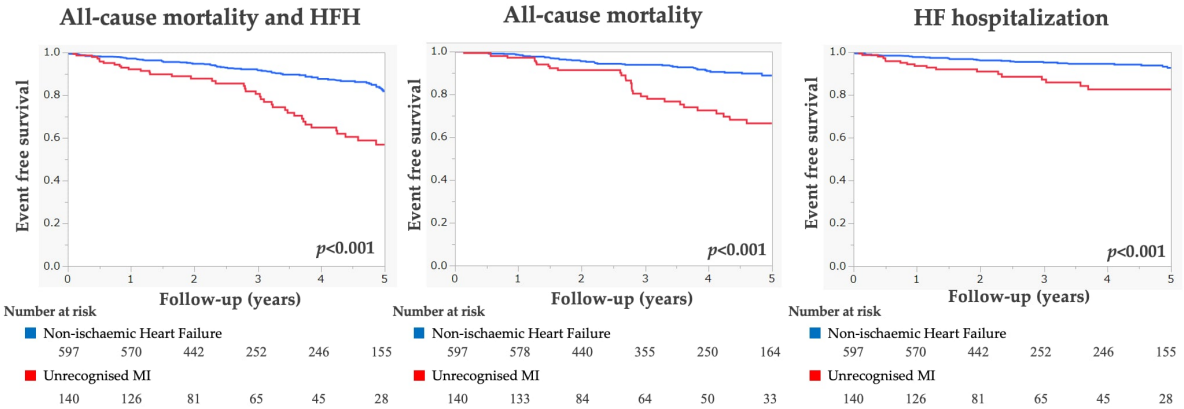


Figure 3. Kaplan–Meier analysis of event-free survival according to the presence or absence of unrecognised myocardial infarction. The unrecognised MI group showed lower event-free survival compared with the non-ischaemic heart failure group.

The LGE granularity score was associated with all three outcomes after adjustment for age, sex, and baseline LVEF (Model 1). After further adjustment for clinical covariates (Model 2), each one-point increase in the score remained associated with the primary endpoint (HR, 1.08; 95% CI, 1.04–1.12; $p < 0.001$) and all-cause mortality (HR, 1.11; 95% CI, 1.06–1.16; $p < 0.001$). The association with HFH was attenuated and was no longer statistically significant (HR, 1.05; 95% CI, 0.99–1.11; $p = 0.101$) (Table 4).

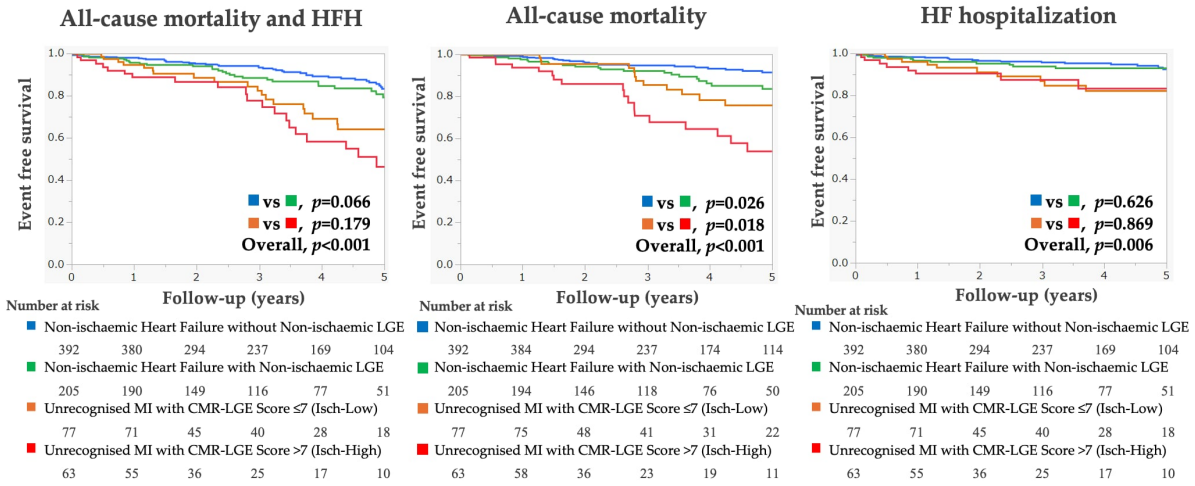


Figure 4. Kaplan–Meier analysis of event-free survival across four groups defined by LGE pattern and granularity score. Patients were stratified into the NI-LGE−, NI-LGE+, Isch-Low, and Isch-High groups. Patients with unrecognised MI and a granularity score >7 (Isch-High) had significantly lower event-free survival, with approximately 50% remaining event-free during follow-up ($p < 0.001$).

Table 4. Cox regression analyses of the association between the continuous LGE granularity score and clinical outcomes.

			Model 1		Model 2	
Predictor	**Unadjusted HR (95% CI)**	***p***	**Adjusted HR * (95% CI)**	***p***	**Adjusted HR * (95% CI)**	***p***
Composite of all-cause mortality and Heart failure hospitalization						
LGE granularity score	1.11 (1.07–1.14)	<0.001	1.09 (1.05–1.13)	<0.001	1.08 (1.04–1.12)	<0.001
All-cause mortality						
LGE granularity score	1.12 (1.08–1.17)	<0.001	1.12 (1.07–1.16)	<0.001	1.11 (1.06–1.16)	<0.001
Heart failure hospitalization						
LGE granularity score	1.09 (1.03–1.17)	0.001	1.07 (1.00–1.13)	0.022	1.05 (0.99–1.11)	0.101

* Hazard ratios are reported per one-point increase in the LGE granularity score. Model 1 was adjusted for age, sex, and baseline LVEF. Model 2 was additionally adjusted for diabetes, hypertension, NYHA class, and the use of diuretics, SGLT2 inhibitors, and antiplatelet agents.

When looking at the four groups—NI-LGE-ve, NI-LGE +ve, Isch-Low, Isch-High—those with unrecognised MI and an LGE granularity score of >7 (Isch-High) had significantly higher rates of the primary outcome—with approximately 50% of patients exhibiting event-free survival ($p < 0.001$) (Figure 4).

Exploratory ROC analysis using the Youden index identified an alternative LGE granularity score cutoff of 13. Kaplan–Meier analyses showed significant differences between patients with scores ≥13 and <13 in survival free from the primary endpoint and in overall survival. Heart failure hospitalisation-free survival did not differ significantly between the groups (Figure 5).

Within the unrecognised MI group, LGE granularity score >7 was associated with higher all-cause mortality in both Model 1 (HR, 2.41; 95% CI, 1.15–5.08; $p = 0.020$) and Model 2 (HR, 2.17; 95% CI, 1.03–4.59; $p = 0.042$). However, the differences in the primary endpoint (HR, 1.54; 95% CI, 0.81–2.92; $p = 0.188$) and HFH (HR, 0.99; 95% CI, 0.38–2.56; $p = 0.977$) were not statistically significant in Model 2 (Table 5).

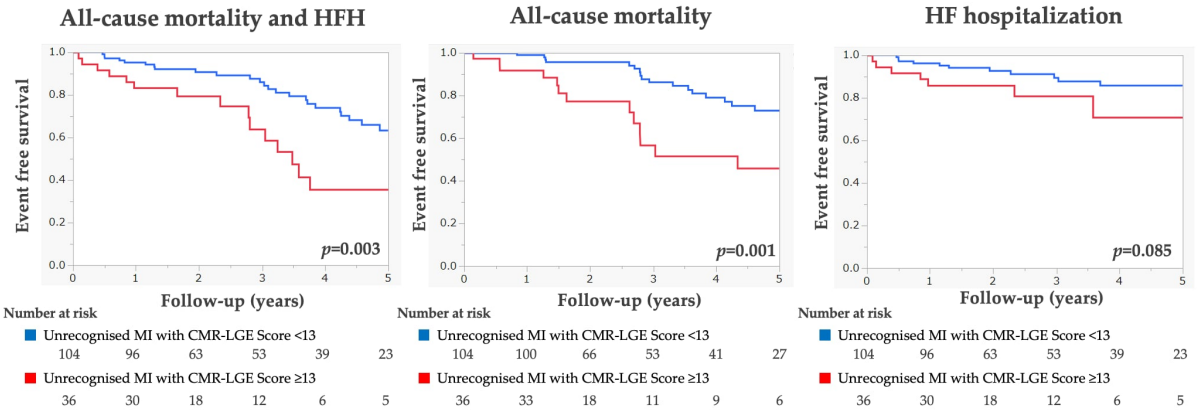


Figure 5. Kaplan–Meier analyses using an exploratory LGE granularity score cutoff of 13. Kaplan–Meier curves for the primary endpoint, all-cause mortality, and heart failure hospitalisation according to LGE granularity scores ≥13 vs. <13. Differences were statistically significant for the primary endpoint and all-cause mortality, but not for heart failure hospitalisation.

Table 5. Cox regression for LGE granularity and the primary and secondary endpoint.

			Model 1		Model 2	
Predictor	**Unadjusted HR (95% CI)**	***p***	**Adjusted HR (95% CI)**	***p***	**Adjusted HR (95% CI)**	***p***
Primary endpoint: composite of all-cause mortality and Heart failure hospitalization						
Non-ischaemic HF w/o non-ischaemic LGE	1.00	Ref.	1.00	Ref.	1.00	Ref.
Non-ischaemic HF w non-ischaemic LGE	1.53 (0.97–2.40)	0.066	1.25 (0.77–2.02)	0.371	1.20 (0.74–1.96)	0.453
Unrecognised MI with LGE granularity score ≤ 7	1.00	Ref.	1.00	Ref.	1.00	Ref.
Unrecognised MI with LGE granularity score > 7	1.53 (0.82–2.87)	0.183	1.55 (0.82–2.93)	0.180	1.54 (0.81–2.92)	0.188
All-cause mortality						
Non-ischaemic HF w/o non-ischaemic LGE	1.00	Ref.	1.00	Ref.	1.00	Ref.
Non-ischaemic HF w non-ischaemic LGE	1.85 (1.06–3.20)	0.029	1.54 (0.86–2.77)	0.150	1.57 (0.87–2.82)	0.135
Unrecognised MI with LGE granularity score ≤ 7	1.00	Ref.	1.00	Ref.	1.00	Ref.
Unrecognised MI with LGE granularity score > 7	2.25 (1.08–4.68)	0.030	2.41 (1.15–5.08)	0.020	2.17 (1.03–4.59)	0.042
Heart failure hospitalization						
Non-ischaemic HF w/o non-ischaemic LGE	1.00	Ref.	1.00	Ref.	1.00	Ref.
Non-ischaemic HF w non-ischaemic LGE	1.20 (0.58–2.45)	0.624	1.02 (0.48–2.18)	0.951	0.93 (0.44–1.97)	0.842
Unrecognised MI with LGE granularity score ≤ 7	1.00	Ref.	1.00	Ref.	1.00	Ref.
Unrecognised MI with LGE granularity score > 7	1.07 (0.42–2.72)	0.880	0.96 (0.38–2.46)	0.938	0.99 (0.38–2.56)	0.977

Model 1 was adjusted for age, sex, and baseline LVEF. Model 2 was additionally adjusted for diabetes, hypertension, NYHA class, and the use of diuretics, SGLT2 inhibitors, and antiplatelet agents. The non-ischaemic group with non-ischaemic LGE was compared with the non-ischaemic group without non-ischaemic LGE as the reference. The unrecognised MI group with an LGE granularity score >7 was compared with the unrecognised MI group with a score ≤7 as the reference. Reference groups are indicated by "Ref.".

Among non-ischaemic patients, those with non-ischaemic LGE were compared with those without non-ischaemic LGE as the reference. In Model 2, there were no statistically significant differences in the primary endpoint (HR, 1.20; 95% CI, 0.74–1.96; $p = 0.453$), all-cause mortality (HR, 1.57; 95% CI, 0.87–2.82; $p = 0.135$), or HFH (HR, 0.93; 95% CI, 0.344–1.97; $p = 0.842$). The unadjusted association between non-ischaemic LGE and all-cause mortality was attenuated after adjustment.

Tests based on scaled Schoenfeld residuals did not indicate violations of the proportional hazards assumption for the continuous LGE granularity score or group classification in any model, and all global tests were nonsignificant. However, NYHA class showed evidence of non-proportional hazards in the extended-adjusted HFH models containing the continuous score ($p = 0.035$) or group classification ($p = 0.032$).

For the primary endpoint, adding unrecognised MI to LVEF improved model fit (likelihood-ratio $\chi^2 = 15.95$, $p < 0.001$) and increased the C-index from 0.583 (95% CI, 0.519–0.646) to 0.625 (95% CI, 0.564–0.686). Adding the continuous LGE granularity score further improved model fit ($\chi^2 = 4.73$, $p = 0.030$), with a C-index of 0.635 (95% CI, 0.576–0.694). The increase in the C-index was 0.010 (95% CI, −0.004 to 0.024). At 5 years, the IDI was 0.011 (95% CI, 0.0002–0.043; $p = 0.044$), whereas the continuous NRI was 0.060 (95% CI, −0.160 to 0.349; $p = 0.384$).

For all-cause mortality, adding unrecognised MI to LVEF improved model fit ($\chi^2 = 16.30$, $p < 0.001$) and increased the C-index from 0.557 (95% CI, 0.489–0.624) to 0.615 (95% CI, 0.545–0.685). Adding the score further improved model fit ($\chi^2 = 8.45$, $p = 0.004$), with a C-index of 0.655 (95% CI, 0.593–0.718). The increase in the C-index was 0.040 (95% CI, −0.005 to 0.085). At 5 years, the IDI was 0.025 (95% CI, 0.004–0.078; $p = 0.006$), whereas the continuous NRI was 0.237 (95% CI, −0.090 to 0.584; $p = 0.132$).

Within the unrecognised MI group, patients with an LGE granularity score >7 had higher odds of LVEF <45% than those with a score ≤7 in both Model 1 (OR, 3.13; 95% CI, 1.22–8.04; $p = 0.018$) and Model 2 (OR, 2.99; 95% CI, 1.12–7.97; $p = 0.029$) (Table 6).

Table 6. Logistic regression analyses of the associations of LGE-based groups with LVEF <45% at CMR scan.

			Model 1		Model 2	
Predictor	**Unadjusted OR (95% CI)**	***p***	**Adjusted OR (95% CI)**	***p***	**Adjusted OR (95% CI)**	***p***
Non-ischaemic HF w /o non-ischaemic LGE	1.00	Ref.	1.00	Ref.	1.00	Ref.
Non-ischaemic HF w non-ischaemic LGE	2.01 (1.41–2.86)	<0.001	1.54 (1.06–2.24)	0.024	1.35 (0.91–1.99)	0.135
Unrecognised MI with LGE granularity score ≤ 7	1.00	Ref.	1.00	Ref.	1.00	Ref.
Unrecognised MI with LGE granularity score > 7	3.00 (1.18–7.62)	0.021	3.13 (1.22–8.04)	0.018	2.99 (1.12–7.97)	0.029

The binary outcome was LVEF < 45% at CMR scan. Model 1 was adjusted for age and sex. Model 2 was additionally adjusted for diabetes, hypertension, NYHA class, and the use of diuretics, SGLT2 inhibitors, and antiplatelet agents. The non-ischaemic group with non-ischaemic LGE was compared with the non-ischaemic group without non-ischaemic LGE as the reference. The unrecognised MI group with an LGE granularity score >7 was compared with the unrecognised MI group with a score ≤7 as the reference. Reference groups are indicated by "Ref.".

In the non-ischaemic group, the presence of non-ischaemic LGE was associated with higher odds of LVEF <45% at the time of CMR after adjustment for age and sex (OR, 1.54; 95% CI, 1.06–2.24; $p = 0.024$). This association was attenuated after additional adjustment in Model 2 (OR, 1.35; 95% CI, 0.91–1.99; $p = 0.135$).

4. Discussion

In this cohort of patients investigated for aetiology of heart failure of presumed non-ischaemic aetiology, those with unrecognised MI were found to have a worse prognosis than those with non-ischaemic heart failure. Amongst patients with unrecognised MI, those with a high granularity score had the worst outcomes in terms of all-cause mortality even after adjustment for baseline risk and clinical covariates. This supports the use of a granularity score for LGE assessment for risk stratification of patients with unrecognised MI.

In a recent meta-analysis of 10 studies assessing those with or at risk of cardiovascular disease, unrecognised MI detected by CMR had a prevalence of 22.5% [16]. Though the prevalence of unrecognised MI has not been previously assessed in an undifferentiated heart failure population, it is comparable to 19% in our study. When studies assessed general populations, the prevalence was lower (10.8%) [16]. Meijs et al. demonstrated that as cardiovascular risk factors and disease accrue, the risk of unrecognised MI expectedly rises, with a prevalence of 26% in the presence of more than four risk factors [17].

In ICELAND MI, in a similar-sized observational population-based study of 935 patients, unrecognised MI had an equivalent 10-year mortality risk as clinically recognised MI [18]. Unrecognised MI was shown to have significant all-cause and cardiovascular risk compared with the normal population (adjusted HR range: 1.52–1.61). Additionally, unrecognised MI was compared to general population participants rather than those with elevated risk (non-ischaemic heart failure in our study). Unrecognised MI was associated with increased risk of future heart failure, carrying approximately a 50% increased risk compared to patients with no MI (HR 1.52 [1.09–2.14]) [18]. Similar findings were reported in the ARIC study, where normal populations were compared to those with unrecognised MI detected via electrocardiogram; unrecognised MI was associated with increased risk of heart failure hospitalisation (adjusted HR 1.85 (1.40–2.44) $p < 0.001$) [18,19].

Our findings show that unrecognised MI also carries prognostic information among patients undergoing assessment for heart failure aetiology. Compared with the non-ischaemic heart failure group, patients with unrecognised MI had higher risks of the primary endpoint, all-cause mortality, and HFH after adjustment for age, sex, and baseline LVEF. The adjusted HRs were 2.29 for all-cause mortality and 2.10 for HFH. Unlike population-based studies assessing incident heart failure, our study examined outcomes in patients already referred for heart failure assessment, using non-ischaemic heart failure as the comparator. Differences in populations, MI detection methods, endpoints, and follow-up preclude direct comparisons of effect sizes. Nevertheless, the persistent associations after adjustment for LVEF support the relevance of identifying previously unrecognised ischaemic injury during the assessment of heart failure aetiology.

Previous studies have only described unrecognised MI in binary terms in its presence or absence. MI characterisation allows clinicians to more easily define subsequent risk. Components of the granularity score have been used to predict risk separately, but using a composite score of the extent, transmurality, location and presence of additional non-ischaemic LGE can help assess the risk to an individual patient in more detail. A modified version of the granularity score proposed by Unger et al. has been shown to improve risk stratification in patients with ischaemic, non-ischaemic and hypertrophic cardiomyopathy [20,21].

The extent of myocardial infarction has been long established to have an association with an increased rate of adverse events. A meta-analysis of studies measuring infarct size in ST-elevation myocardial infarction (STEMI) patients demonstrated that for every 5% increase in infarct size, the risk of all-cause mortality or heart failure hospitalisation increased by 20% within one year of primary percutaneous coronary intervention (PCI) [22]. The extent of LGE, in terms of both the number of segments and mass, have demonstrated

an increased risk of arrhythmic events [23]. Specifically, in patients with unrecognised MI, left ventricular mass of LGE has been shown to correlate with major cardiac events, with every 1% and 10% increase associated with a 9% and 77% increase, respectively [16].

The granularity score used is heavily weighted towards left anterior descending (LAD) infarcts, with anterior MIs having long been associated with an increased incidence of adverse outcomes. In terms of LGE location, anterior LGE has been shown to be the strongest predictor of developing ventricular tachycardia [10]. There is ongoing discussion around the relationship between infarct location, myocardial insult and long-term clinical outcomes, with evidence suggesting that worse outcomes in patients with an anterior ST-elevation MI are due to the extent of myocardial and microvascular damage rather than the location of the infarct [24]. CMR studies have shown that even though anterior MI is associated with an increased degree of this remodelling, it is in fact infarct size that confers heightened risk due to a greater degree of myocardial damage rather than infarct location [25,26]. In our study, the continuous LGE granularity score remained associated with the primary endpoint and all-cause mortality after adjustment for clinical covariates, whereas its association with HFH was attenuated. The score may therefore capture aspects of myocardial damage that are not fully reflected by LVEF or other clinical information.

The association between the score and LV systolic function may also help explain its association with outcomes. Within the unrecognised MI group, patients with an LGE granularity score > 7 had higher odds of LVEF $< 45\%$ at CMR than those with a score ≤ 7, even after adjustment for clinical covariates. This finding is consistent with a relationship between the LGE characteristics captured by the score and impaired LV systolic function. However, the absence of a significant adjusted association with HFH also limits conclusions about the relationship between the score, ventricular remodelling, and subsequent heart failure hospitalisation.

Non-ischaemic LGE is also associated with adverse outcomes in non-ischaemic heart failure. In dilated cardiomyopathy (DCM), its presence is associated with a 9-fold increased risk of sudden cardiac death and aborted sudden cardiac death [11]. In patients with ischaemic heart failure, the presence of non-ischaemic LGE has been associated with almost a five-fold increased risk of major adverse cardiovascular events (MACE) [27]. Unger et al. showed that concomitant non-ischaemic LGE almost doubles mortality risk, even after adjustment for ischaemic LGE [13].

In our cohort, non-ischaemic LGE was associated with all-cause mortality in unadjusted analysis, but this association was attenuated after adjustment for clinical covariates. No statistically significant associations with the primary endpoint or HFH were observed in the fully adjusted model. These findings suggest that the prognostic value of non-ischaemic LGE may be limited after accounting for clinical characteristics in this cohort.

Investigation into the aetiology of heart failure is one of the most common indications for CMR and remains a class 1 indication in the latest ESC heart failure guidelines [28,29]. The clinical use of CMR has increased substantially over the past 15 years, reflecting its expanding role in the investigation of heart failure aetiology [30]. By considering the specific LGE extent, transmurality, distribution and location, the use of an LGE granularity score gives CMR reporters the opportunity to help guide clinicians in assessing a patient's individual risk profile for future events. In our study, adding the score improved model fit and 5-year IDI for both the primary endpoint and all-cause mortality. However, the increases in the C-index were modest and continuous NRI was not statistically significant for either endpoint. These results suggest additional prognostic information, although its value for clinical risk classification remains uncertain. The score may inform discussions about further assessment and management, but our findings do not establish that score-guided

changes in medical or device therapy improve outcomes. Validation in independent cohorts is needed before the score can be used to guide treatment decisions.

There are limitations to this study. First, this is a single-centre study with a relatively small number of patients with unrecognised MI. The limited number of events in subgroup and secondary outcome analyses may also have reduced statistical power and increased the risk of overfitting, particularly in models with multiple covariates. Second, patients recruited to this study were real-world patients with heart failure and may not have been on optimal guideline-directed medical therapy (GDMT) at the time of CMR. Furthermore, during the recruitment phase, some current GDMT recommendations were not yet endorsed. The majority of patients were receiving therapy with ACE inhibitors and beta blockers; however, under half of patients were receiving mineralocorticoid receptor antagonists, and only approximately one in six patients were receiving a SGLT2i. Patients were referred for aetiology assessment at unspecified time points after their initial heart failure diagnosis. As a result, subsequent LV functional assessment showed improvement in over one-third of patients. The decision for referral to the specialist centre was at the discretion of local referring cardiologists; therefore, although representative of real-world behaviour, it may include inherent bias in patient selection, such as age and availability of MRI. Third, data on subsequent echocardiography, coronary investigations and their findings, revascularisation, ICD/CRT implantation, and changes in GDMT after CMR were unavailable. We therefore could not assess how subsequent investigations and treatment influenced outcomes or contributed to the observed associations between LGE findings and prognosis. Fourth, renal function data were unavailable, and NT-proBNP measurements were incomplete. Neither variable was included in the multivariable models, leaving the possibility of residual confounding by renal dysfunction and heart failure severity. Finally, information on cardiovascular causes of death was also unavailable, preventing separate assessment of cardiovascular mortality. Fifthly, patients with left bundle branch block were not excluded from the study. Inclusion of this group of patients may have led to the detection of pseudo-infarction patterns on LGE imaging. The recommendations for GDMT within this cohort differ from those who have a higher degree of symptoms or have an LVEF <40%.

5. Conclusions

Patients presenting with heart failure who were subsequently discovered to have evidence of an unrecognised MI had worse prognosis than those with non-ischaemic heart failure. Patients with evidence of an unrecognised MI with a calculated granularity score of greater than seven had the highest risk. The use of a CMR LGE granularity score, which takes into account the extent, location and distribution of scar, provides the opportunity to risk-stratify patients, highlighting to clinicians patients at high risk of adverse events to ensure optimal medical and device therapy. This CMR LGE granularity score relies purely on visual assessment, rather than requiring dedicated software, allowing it to be easily adopted by clinicians.

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Abbreviations

The following abbreviations are used in this manuscript:

ACEi	Angiotensin converting enzyme inhibitor
ARB	Angiotensin receptor blocker
ARNI	Angiotensin receptor–neprilysin inhibitor
BMI	Body mass index
BSA	Body surface area
CI	Confidence interval
CMR	Cardiovascular magnetic resonance
DCM	Dilated cardiomyopathy
ECV	Extracellular volume
EDVi	End-diastolic volume index
EF	Ejection fraction
ESC	European Society of Cardiology
ESVi	End-systolic volume index
GDMT	Guideline-directed medical therapy
HF	Heart failure
HFH	Heart failure hospitalisation
HR	Hazard ratio
IQR	Interquartile range
LGE	Late gadolinium enhancement
LV	Left ventricle
LVEF	Left ventricular ejection fraction
MRA	Mineralocorticoid receptor antagonist
MATCH	MyocArdial Tissue CHaracteristics in patients with heart failure
MI	Myocardial infarction
NYHA	New York Heart Association
SGLT2i	Sodium-glucose cotransporter 2 inhibitor
RV	Right ventricle
RVEF	Right ventricular ejection fraction

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