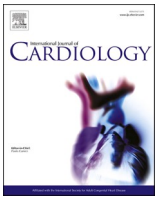
International Journal of Cardiology 460 (2026) 134617

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard



Clinical significance of incidental pericardial late gadolinium enhancement on cardiac magnetic resonance

Veronica Lepre [a], Nicole Moretto [b], Maria De Martino [b,c,e], Valentino Collini [a,b], Mariacristina Tomat [b], Francesco Venturelli [a,f], Laura Cescon [a,f], Cosimo Vittorio Agrimi [a,f], Michela Puppato [d], Andrea Pellegrin [d], Gaia Zussino [d], Laura Zuliani [d], Miriam Isola [b,c], Massimo Imazio [a,b,*,1]

[a] Cardiothoracic Department, University Hospital Santa Maria della Misericordia, Udine, Italy
[b] Department of Medicine, University of Udine, Udine, Italy
[c] Biostatistic Unit, University of Udine, Udine, Italy
[d] CMR Unit, Radiology Department, University Hospital Santa Maria della Misericordia, Udine, Italy
[e] Aging Research Center, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet and Stockholm University, Stockholm, Sweden
[f] Center for Diagnosis and Treatment of Cardiomyopathies, Cardiovascular Department, Azienda Sanitaria Universitaria Giuliano-Isontina (ASUGI), University of Trieste, Italy

ARTICLE INFO

Keywords:
Pericardial late gadolinium enhancement
Cardiac magnetic resonance
Pericarditis
Constrictive pericarditis
Pericardial inflammation
Prognosis

ABSTRACT

Background: Pericardial late gadolinium enhancement (LGE) on cardiac magnetic resonance (CMR) is typically interpreted as a marker of active pericardial inflammation. However, it may also be detected incidentally during CMR performed for unrelated indications, and its clinical significance remains unclear. Whether incidental pericardial LGE reflects persistent subclinical inflammation associated with adverse long-term outcomes is unknown.

Methods: We conducted a retrospective single-center study including consecutive patients undergoing clinically indicated CMR. Patients with incidentally detected pericardial LGE were identified and compared with a control population without LGE. Clinical characteristics, CMR findings, and long-term outcomes were collected. The primary endpoint was the development of constrictive pericarditis (CP) during follow-up.

Results: Among 590 patients, incidental pericardial LGE was identified in 31 (5.3%), while 559 patients had no LGE. During a median follow-up of 72 months, CP occurred in 2 patients (6.5%) with LGE and in none of the patients without LGE ($p = 0.003$). Patients with LGE more frequently had a history of pericarditis and showed higher prevalence of pericardial effusion and increased pericardial thickness on CMR.

Conclusions: Incidental pericardial LGE on CMR was associated with a history of prior pericardial and myocardial inflammation, and CP occurred only among patients with LGE during follow-up. However, these findings should be interpreted cautiously, as LGE may represent a surrogate marker of an underlying inflammatory pericardial substrate rather than an independent prognostic marker. Larger prospective studies are needed to determine whether incidental pericardial LGE provides prognostic information beyond clinical history.

1. Introduction

Pericardial diseases encompass a broad spectrum of inflammatory and structural conditions affecting the pericardium, ranging from acute pericarditis to chronic constrictive pericarditis. Multimodality imaging plays a fundamental role in their diagnosis and management, with cardiac magnetic resonance (CMR) emerging as a particularly valuable technique for assessing both the structural and inflammatory components of pericardial disease. [1–3]

Late gadolinium enhancement (LGE) imaging is widely used in CMR to identify myocardial fibrosis and inflammation, but it also provides important insights into pericardial pathology. In the setting of

* Corresponding author at: Piazzale Santa Maria della Misericordia 15, 33100 Udine, Italy.
E-mail address: massimo.imazio@uniud.it (M. Imazio).
[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

https://doi.org/10.1016/j.ijcard.2026.134617
Received 31 March 2026; Received in revised form 7 May 2026; Accepted 8 June 2026
Available online 9 June 2026
0167-5273/© 2026 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

pericardial inflammation, gadolinium contrast accumulates within the pericardial layers because of increased vascular permeability, neovascularization, and inflammatory cell infiltration. These processes result in delayed contrast washout and therefore visible enhancement on LGE sequences. [3]

Importantly, pericardial LGE should not be interpreted solely as a marker of fibrotic remodelling. Instead, it frequently reflects an active inflammatory process involving the pericardial layers. Experimental and clinical data suggest that persistent inflammation may lead to progressive pericardial thickening and fibrosis, ultimately contributing to the development of constrictive physiology. [1,3]

While pericardial LGE is commonly observed in patients with clinically apparent pericarditis, it may also be detected incidentally during CMR examinations performed for other indications such as cardiomyopathy, ischemic heart disease, or myocarditis. In these circumstances, the prognostic significance of this imaging finding remains unclear. The presence of LGE could represent residual inflammatory activity from prior pericardial injury or may reflect ongoing subclinical inflammation.

Several studies have suggested that CMR markers of pericardial inflammation may have prognostic value. For example, pericardial LGE has been associated with disease activity in recurrent pericarditis and may help identify patients at increased risk of recurrence [4] [5]. In addition, pericardial LGE combined with elevated inflammatory biomarkers has been shown to predict reversibility of constrictive pericarditis with anti-inflammatory therapy [6].

More recently, Pavon and colleagues demonstrated that pericardial LGE may persist for many years after cardiac surgery and is associated histologically with chronic low-grade inflammation of the pericardium [7]. These findings highlight the possibility that pericardial LGE may represent a marker of ongoing inflammatory remodelling even in the absence of overt clinical symptoms.

Despite these observations, the long-term clinical implications of incidentally detected pericardial LGE remain poorly defined. It is unknown whether this finding identifies patients at increased risk of developing constrictive pericarditis.

The aim of the present study was therefore to evaluate the prognostic significance of incidental pericardial LGE detected on CMR and to assess whether this imaging finding is associated with an increased risk of constrictive pericardial disease during long-term follow-up.

2. Materials and methods

2.1. Study design and population

This retrospective observational study included consecutive patients who underwent CMR for any clinically indicated reason other than suspected pericarditis, at our tertiary referral centre between January 2016 and December 2019. Symptoms present at the time of CMR were not related to active pericarditis or constrictive physiology, and CMR was performed for indications other than pericarditis.

Exclusion criteria were known history of constrictive pericarditis, cardiac transplantation, insufficient clinical data for analysis (primarily patients referred externally and not followed at our centre), and CMR examinations performed without gadolinium contrast. For patients who underwent more than one CMR during the study period, only the earliest examination was included. Of 989 patients screened, 590 were eligible and included in the final analysis.

CMR scans were screened for the presence of pericardial LGE on standard LGE sequences. Patients demonstrating incidental pericardial LGE constituted the study group. In the present study, 'incidental pericardial LGE' was defined as pericardial enhancement detected on a CMR examination performed for an indication other than suspected active pericarditis or known constrictive pericarditis. Thus, the term 'incidental' refers to the reason for CMR referral and not necessarily to the absence of previous inflammatory cardiac disease.

The control population consisted of patients undergoing CMR during the same period without evidence of pericardial LGE.

Clinical history, CMR parameters, and follow-up data were obtained from institutional electronic medical records. Inflammatory biomarkers, including C-reactive protein, were not systematically available at the time of CMR and therefore were not included in the main analysis. Follow-up information included clinical visits, hospitalizations, and imaging studies. Diagnostic criteria of myocarditis and pericarditis and diagnostic criteria for cardiomyopathies followed respectively 2025 and 2023 corresponding ESC guidelines.

The primary endpoint of the study was development of constrictive pericarditis during follow-up. Secondary outcomes included occurrence of pericarditis and all-cause mortality.

Constrictive pericarditis was diagnosed by the presence of compatible clinical features of right-sided heart failure or impaired diastolic filling together with imaging evidence of constrictive physiology on echocardiography, CMR, CT, and confirmation at cardiac catheterization.

2.2. Cardiac magnetic resonance protocol

All CMR examinations were performed using 1.5 T magnetic resonance scanner (Siemens Aerea). Studies were performed using dedicated cardiac software and ECG-gating. Cine short axis images were acquired in a stack of contiguous 8 mm-thick slices from cardiac base to apex. Approximately ten minutes after intravenous administration of 0.2 mmol/kg gadolinium-based contrast agent, LGE images were acquired using segmented T1-weighted gradient-echo inversion-recovery and phase sensitive gradient-echo inversion-recovery sequences in the same views used for cine images. When available, T1-weighted morphological imaging and STIR T2-weighted were also examined.

LV and RV volumes and function and LV myocardial mass were measured using the standard volumetric technique from the cine short axis stack.

Pericardial LGE was considered present only when enhancement was visible in the expected anatomical location of the pericardium on at least two contiguous images and orthogonal views, and was distinguishable from blood-pool signal, respiratory motion, partial-volume effects, epicardial fat, and adjacent pleural enhancement. Pericardial enhancement was evaluated visually by two experienced observers. Interobserver agreement for the presence of pericardial LGE was good, with a Cohen's kappa of 0.80; discordant cases were resolved by consensus with a third experienced observer.

Pericardial LGE severity was graded according to the 2025 ACC Expert Consensus Statement on the Diagnosis and Management of Pericarditis criteria using three parameters: 1) number of short-axis levels involved, assessed at basal, mid-ventricular, and apical slices; 2) circumferential extent of enhancement on each slice, using a 50% threshold; and 3) maximal pericardial LGE thickness, using a 3-mm threshold. The presence of LGE was classified as mild when enhancement involved $\geq$50% of the circumference in fewer than three slices with thickness $\leq$ 3 mm, or $<$ 50% of the circumference in fewer than three slices with thickness $>$ 3 mm; moderate when enhancement involved $\geq$50% of the circumference in all three slices or thickness $>$ 3 mm, but not both, or when $<$50% circumferential enhancement involved all three slices with thickness $>$ 3 mm; and severe when enhancement involved $\geq$50% of the circumference in all three slices and thickness was $>$3 mm [2].

Pericardial thickness and the presence of pericardial effusion were also systematically evaluated in T1- or STIR T2-weighted when available, or in their absence in cine sequences.

2.3. Statistical analysis

Continuous variables are reported as median and interquartile range (IQR) or as mean $\pm$ standard deviation when normally distributed. Categorical variables are presented as counts and percentages. Group

comparisons for continuous variables were performed using the Mann–Whitney *U* test or Student's *t*-test, depending on the distribution of the data, and the chi-square or Fisher's exact test for categorical variables, as appropriate.

To evaluate independent predictors of pericarditis recurrence, univariable and multivariable logistic regression analyses were performed. Variables included in the multivariable model were selected based on clinical relevance and included prior history of pericarditis and prior history of myocarditis. Results are expressed as odds ratios (OR) with 95% confidence intervals (CI).

The cumulative incidence of pericardial constriction was estimated accounting for the competing risk of death. Given the very small number of events, time-to-event analyses are presented descriptively.

A two-sided *p*-value <0.05 was considered statistically significant. All analyses were performed using R software (version 4.5.1, R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Study population

A total of 590 patients were included in the analysis. Among these, 31 patients (5.3%) demonstrated pericardial LGE. The control group consisted of 559 patients without evidence of pericardial LGE. A representative CMR example of incidental pericardial LGE is illustrated in Fig. 1.

3.2. Characteristics of pericardial LGE

Among patients with pericardial LGE, enhancement intensity was lower than the blood pool in 61.3% and greater than the blood pool in 38.7%. In most cases (87.1%), enhancement involved more than 50% of the pericardial surface at least in one axial level.

Pericardial enhancement was observed in a mean of 2.45 axial levels per patient, indicating that in most cases the process involved multiple pericardial regions.

Pericardial LGE was graded as mild in most patients (48.4%), moderate in 29.0%, and severe in the remaining 22.6%.

3.3. Clinical characteristics

Patients with pericardial LGE had a significantly higher prevalence of prior pericarditis compared with those without LGE (48.4% vs 7%, $p < 0.001$). They also more frequently had a history of myocarditis (41.9% vs 10.2%, $p < 0.001$). These baseline differences indicate that patients with incidental pericardial LGE represented a clinically distinct subgroup with a substantially higher burden of previous inflammatory cardiac disease. Therefore, subsequent associations between LGE and follow-up pericardial events may be confounded by prior pericarditis or myocarditis.

Although prior cardiac surgery was numerically more frequent among patients with LGE, this difference did not reach statistical significance. The baseline features of patients with pericardial LGE are summarized in Table 1. A comparison of the clinical features of patients with or without pericardial LGE is reported in Tables 2 and Table 3.

3.4. Associated CMR findings

Pericardial effusion was significantly more frequent in patients with pericardial LGE (22.6% vs 4.7%, $p < 0.001$). Pericardial thickness was also significantly greater among patients with LGE compared with those without enhancement (2.4 mm vs 1.8 mm, $p < 0.001$). Left ventricular ejection fraction and right ventricular function were similar between groups. Left ventricular end diastolic volume was significantly greater than in the non-LGE group, due to the greater prevalence of ischemic and non-ischemic cardiomyopathy (17.4% and 30.1% respectively).

3.5. CMR diagnosis

CMR final diagnosis in the pericardial LGE group was myocardial or pericardial inflammatory syndrome in 74.2% of cases. In the remaining patients, the main diagnosis was ischemic cardiomyopathy ($n = 4$,

Table 1
Characteristics of patients with pericardial late gadolinium enhancement (LGE).

Pericardial LGE patients ($n = 31$)	
LGE grading – Intensity	
Less than blood pool	19 (61,3%)
More than blood pool	12 (38,7%)
LGE diffusion	
< 50%	4 (12,9%)
> 50%	27 (87,1%)
Pericardial axial levels (base, medium, apex)	
Mean	Mean 2,45 (± 0,77 DS)
One level	16,1%
Two levels	22,6%
Three levels	61,3%
Pericardial LGE severity (2025 ACC expert consensus)	
Mild	15 (48,4%)
Moderate	9 (29,0%)
Severe	7 (22,6%)

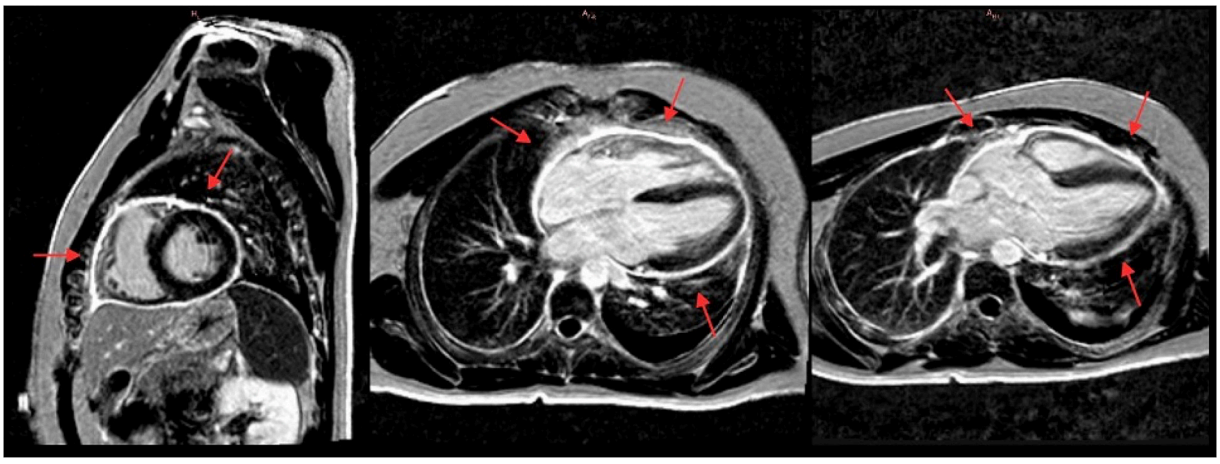


Fig. 1. Representative CMR image showing severe pericardial late gadolinium enhancement (LGE) indicated by red arrows and consistent with pericardial inflammatory involvement. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 2
Comparison between patients with and without pericardial LGE in terms of anamnestic data, CMR data and follow-up results.

Variable	LGE – (n° 559) (follow up available in 541 pts)	LGE + (n° 31) (follow up available in 31 pts)	P_value
Clinical data			
Age (years)	54 [42–66]	46 [35.5–60]	0.078
Male sex	180 (32.2%)	8 (25.8%)	0.585
History of pericarditis	39 (7%)	15 (48.4%)	**<0.001**
Type of pericarditis			
Acute	32 (82.1%)	9 (60%)	
Recurrent	5 (12.8%)	4 (26.7%)	
Incessant	2 (5.1%)	1 (6.7%)	
Chronic	0 (0%)	1 (6.7)	
History of myocarditis	57 (10.2%)	13 (41.9%)	**<0.001**
Previous cardiac surgery	18 (3.2%)	3 (9.7%)	0.092
CABG	8	1	
Aortic surgery	1	0	
Mixed	2	1	
Congenital	8	1	
Early pericardial complications			
None	17 (94.4%)	2 (66.7%)	
Pericarditis	0	0	
Pericardial effusion	1 (5.6%)	1 (33.3%)	
Revision surgery	0	0	
Late reinterventions	1 (5.6%)	0	
CMR			
LVEF (%)	58 [45–67]	60 [52.25–65]	0.611
LVEDVi (ml/m2)	85 [72–106]	78 [67.5–87.25]	0.024
RVEF (%)	63 [57–69]	63 [58.25–66.5]	0.728
RVEDVi (ml/m2)	73 [62–86]	71.5 [62.5–79.75]	0.459
Pericardial effusion	26 (4.7%)	7 (22.6%)	**<0.001**
Circumferential pericardial effusion	19 (76%)	4 (57.1%)	0.370
Pericardial effusion, width (mm)	9 [7–17]	7 [5.5–10]	0.170
Pericardial thickness (mm)	1.8 [1.7–2]	2.4 [2.1–3.1]	**<0.001**
Follow-up			
Length of follow up (months)	61 [36–76.75]	72 [43.5–88.5]	0.036
Pericarditis relapse	8 (1.5%)	5 (16.1%)	**<0.001**
Pericardial constriction	0 (0%)	2 (6.5%)	**0.003**
Death (all causes)	62 (11.2%)	2 (6.5%)	0.562
CV death	24 (44.4%)	1 (100%)	
CMR diagnosis			
Normal	202 (36.1%)	0 (%)	
Ischaemic	97 (17.4%)	4 (12.9%)	
Non-ischaemic cardiomyopathy	168 (30.1%)	2 (6.5%)	
Pericarditis or myocarditis	53 (9.5%)	21 (67.7%)	
Valvular disease	18 (3.2%)	0 (%)	
Congenital	10 (1.8%)	0 (%)	
Other/mixed	11 (2%)	4 (12.9%)	

Values are presented as number (%) or median (interquartile range) as appropriate.
CABG: coronary artery bypass grafting; LVEF: left ventricular ejection fraction; LVEDVi: left ventricular end-diastolic volume indexed; RVEF: right ventricular ejection fraction; RVEDVi: right ventricular end-diastolic volume indexed.

12.9%), non-ischemic cardiomyopathy (n = 2, 6.5%) and mixed phenotype (n = 4, 12.9%). In the context of ischemic cardiomyopathy, the finding of pericardial LGE was associated with inflammatory involvement of the pericardial layers in the early phase of an acute coronary syndrome. The two cases in non-ischemic cardiomyopathy were classified both as non-dilated left ventricular cardiomyopathy, one of them with history of systemic sclerosis. The mixed phenotype group included patients with overlapping ischemic and inflammatory features, post-surgical pericardial involvement, and connective tissue disease.

Table 3
Univariable and multivariable logistic regression model for pericarditis recurrence.

Variable	Univariable OR (95% CI)	p-value	Multivariable OR (95% CI)†	p-value
Pericardial LGE	12.81 (3.66–41.23)	<0.001	2.933 (0.71–11.461)	0.1249
Prior pericarditis	–	–	37.52 (7.189–297.647)	**<0.001**
Prior myocarditis	–	–	1.72 (0.433–7.389)	0.4496

†OR: odds ratio; CI: confidence interval.

3.6. Follow-up outcomes

The median follow-up duration was 72 months in patients with LGE and 61 months in those without LGE.

On univariable analysis, incidental pericardial LGE was significantly associated with pericarditis occurrence (16.1% vs 1.5%, OR 12.8, 95% CI 3.66–41.2, $p < 0.001$). However, after adjustment for prior history of pericarditis, LGE was no longer independently associated with recurrence (OR 2.93, 95% CI 0.71–11.46, $p = 0.12$), while prior pericarditis emerged as the dominant predictor (OR 37.52, 95% CI 7.19–297, $p < 0.001$). These findings suggest that the association between incidental pericardial LGE and pericarditis may be largely mediated by the underlying inflammatory history of the patient.

During follow-up, constrictive pericarditis developed in two patients with pericardial LGE and in none of the patients without LGE. Given the very small number of events and the absence of events in the control group, no multivariable time-to-event analysis was feasible, therefore, findings are presented descriptively and should not be interpreted as establishing independent prognostic value. The cumulative incidence of pericardial constriction in patients with LGE was 3.6% at 36 months and 8.5% at 60 months, whereas no events were observed in patients without LGE (Fig. 2). The time-to-event curve is shown for descriptive purposes only.

All-cause and cardiovascular mortality did not differ significantly between groups (Fig. 3). Both patients with constrictive pericarditis underwent pericardiectomy and are still living at follow up visits.

4. Discussion

The present study provides preliminary observational data on the long-term clinical course of patients with incidentally detected pericardial LGE. Although constrictive pericarditis occurred only in patients with pericardial LGE, this finding was based on only two events and should therefore be considered exploratory and hypothesis-generating rather than definitive evidence of prognostic value. This observation supports the concept that pericardial LGE may represent an imaging marker of persistent inflammatory activity within the pericardium. Chronic inflammation of the pericardial layers can lead to progressive fibrotic remodelling and thickening, ultimately resulting in impaired diastolic filling due to constrictive physiology.

The pathophysiologic mechanisms underlying pericardial enhancement on LGE imaging are complex. Inflammatory processes increase vascular permeability and promote neovascularization within the pericardial tissue. These changes allow accumulation of gadolinium contrast within the extracellular space, producing delayed enhancement on CMR imaging. [3]

Our findings are consistent with previous studies demonstrating the role of CMR in characterizing inflammatory activity in pericardial disease. In patients with recurrent pericarditis, CMR markers such as pericardial oedema and LGE have been shown to correlate with disease activity and may provide incremental diagnostic and prognostic information [4] [5].

In addition, Feng et al. demonstrated that pericardial LGE in patients

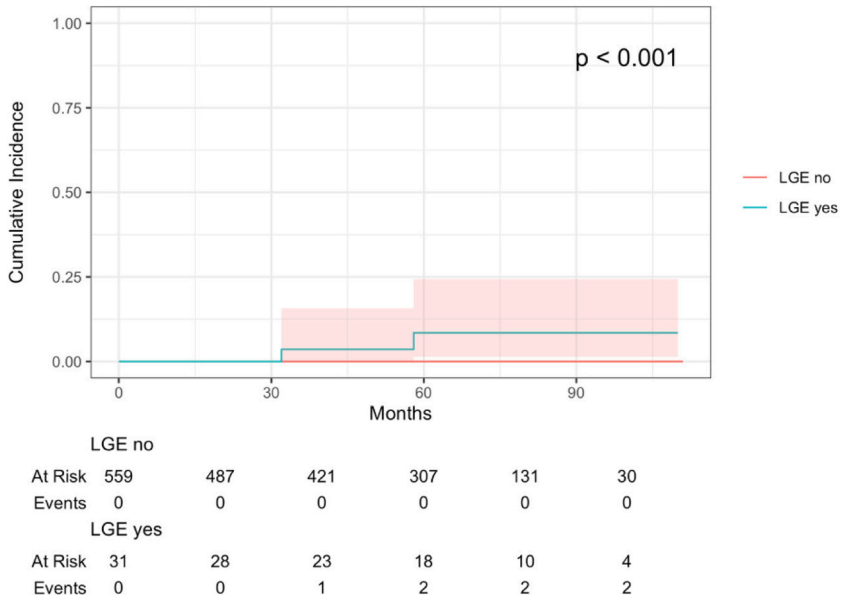


Fig. 2. Cumulative incidence of constrictive pericarditis in patients with and without incidental pericardial late gadolinium enhancement (LGE) on cardiac magnetic resonance. Shaded areas represent 95% confidence intervals. Numbers at risk are shown below the x-axis. Gray's test $p < 0.001$.

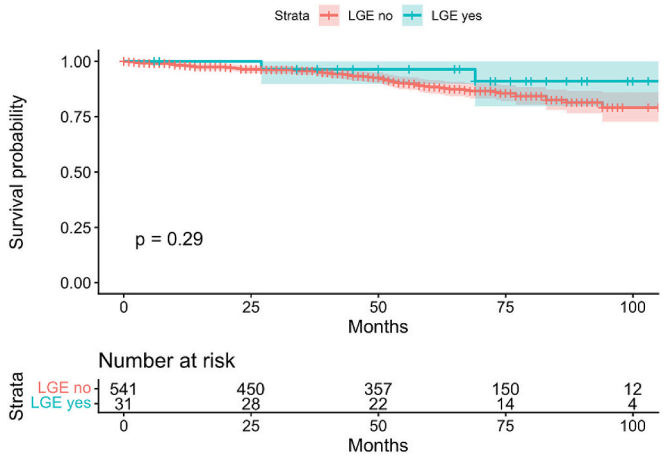


Fig. 3. Kaplan-Meier curves for all-cause mortality in patients with and without incidental pericardial LGE. Log-rank $p = 0.29$.

with constrictive physiology may identify those with active inflammatory disease who are more likely to respond to anti-inflammatory therapy. [6] These observations further emphasize the importance of inflammatory processes in the pathogenesis of constrictive pericarditis.

Our results also align with recent findings reported by Pavon et al., who observed that pericardial LGE may persist many years after cardiac surgery and corresponds histologically to chronic subclinical inflammation [7]. In that study, low-grade inflammatory infiltrates were detected in biopsy specimens from patients with circumferential pericardial enhancement, suggesting that LGE may reflect ongoing inflammatory remodelling even in clinically asymptomatic individuals.

Notably, on multivariable logistic regression analysis, the association between pericardial LGE and pericarditis recurrence was attenuated and no longer statistically significant after adjustment for prior history of pericarditis, which emerged as the dominant independent predictor of recurrence. This finding suggests that, at least for the recurrence endpoint, pericardial LGE may serve as a surrogate marker of an underlying inflammatory substrate rather than an independent prognostic driver. Patients with incidental pericardial LGE frequently carry a history of prior pericardial disease, and it is this inflammatory background — rather than the imaging finding per se — that appears to drive the risk of recurrence. Whether pericardial LGE provides additional prognostic information beyond clinical history in patients without prior pericardial disease remains an open question that warrants investigation in larger prospective cohorts.

Taken together, these data suggest that incidental pericardial LGE may represent a spectrum of chronic inflammatory pericardial injury. While many patients remain clinically stable, a subset may progress toward constrictive disease over time.

From a clinical standpoint, recognition of incidental pericardial LGE may justify careful clinical correlation, particularly in patients with prior pericarditis, pericardial thickening, or pericardial effusion. However, the present data are insufficient to define a specific surveillance strategy or to recommend management changes based solely on this imaging finding. The presence of additional features such as pericardial thickening or effusion may further increase suspicion for persistent inflammatory activity.

4.1. Study limitations

Some limitations of the present study should be acknowledged. A major limitation of the present study is the very small number of constrictive pericarditis events. Only two cases occurred, both in the pericardial LGE group, while no events were observed among controls. Consequently, the statistical comparison is driven by extremely low event counts, and no multivariable time-to-event analysis could be performed because of complete separation. The observed association should therefore be interpreted descriptively and as hypothesis-generating rather than as evidence that incidental pericardial LGE is an independent prognostic marker. The retrospective single-centre design introduces potential selection and referral bias. Patients referred externally and not followed at our institution were excluded because of insufficient follow-up data, which may have selected a population with more complete local clinical information. Moreover, as a tertiary referral centre, our cohort may not be fully representative of patients undergoing CMR in other clinical settings.

Finally, inflammatory biomarkers such as C-reactive protein were not systematically collected, preventing correlation between imaging findings and serological markers of disease activity. Future studies should prospectively collect clinical, laboratory, and imaging data in

larger multicentre cohorts to better define the prognostic significance of incidental pericardial LGE.

5. Conclusions

In this retrospective single-centre cohort, incidental pericardial LGE was identified in a small proportion of patients undergoing clinically indicated CMR, and constrictive pericarditis occurred only among patients with LGE. Because this observation was based on only two events, the findings should be interpreted as exploratory and hypothesis-generating. Larger prospective studies are required to determine whether incidental pericardial LGE provides independent prognostic information and whether it should influence follow-up strategies.

CRediT authorship contribution statement

Veronica Lepre: Writing – review & editing, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Nicole Moretto:** Writing – review & editing, Methodology, Investigation, Data curation. **Maria De Martino:** Writing – review & editing, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Valentino Collini:** Writing – review & editing, Writing – original draft, Validation, Data curation, Conceptualization. **Mariacristina Tomat:** Writing – review & editing, Visualization, Validation, Data curation. **Francesco Venturelli:** Writing – review & editing, Validation, Data curation, Conceptualization. **Laura Cescon:** Writing – review & editing, Supervision, Data curation. **Cosimo Vittorio Agrimi:** Writing – original draft, Validation, Methodology, Data curation. **Michela Puppato:** Writing – original draft, Validation, Data curation, Conceptualization. **Andrea Pellegrin:** Writing – review & editing, Validation, Data curation, Conceptualization. **Gaia Zussino:** Writing – original draft, Visualization, Supervision, Data curation, Conceptualization. **Laura Zuliani:** Writing – review & editing, Visualization, Validation, Data curation, Conceptualization. **Miriam Isola:** Writing – review & editing, Visualization, Validation, Supervision, Software, Formal analysis, Data curation, Conceptualization. **Massimo Imazio:** Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

References

[1] J. Schulz-Menger, V. Collini, J. Gröschel, Y. Adler, A. Brucato, V. Christian, V. M. Ferreira, E. Gandjbakhch, B. Heidecker, M. Kerneis, A.L. Klein, K. Klingel, G. Lazaros, R. Lorusso, E.G. Nesukay, K. Rahimi, A.D. Ristić, M. Rucinski, L.E. Sade, H. Schaubroeck, A.G. Semb, G. Sinagra, J.J. Thune, Imazio M; ESC scientific document group. 2025 ESC guidelines for the management of myocarditis and pericarditis, Eur. Heart J. 46 (40) (2025 Oct 22) 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192. PMID: 40878297.

[2] T.K.M. Wang, A.L. Klein, P.C. Cremer, M. Imazio, S. Kohnstamm, S.A. Luis, V. Mardigyan, M. Mukherjee, K. Ordovas, S. Vakamudi, G.F. Wohlford, 2025 concise clinical guidance: an ACC expert consensus statement on the diagnosis and Management of Pericarditis: a report of the American College of Cardiology Solution set Oversight Committee, J. Am. Coll. Cardiol. 86 (25) (2025) 2691–2719, https://doi.org/10.1016/j.jacc.2025.05.023. Epub 2025 Aug 6. PMID: 40767817.

[3] A.L. Klein, T.K.M. Wang, P.C. Cremer, A. Abbate, Y. Adler, C. Asher, A. Brucato, M. Chetrit, B. Hoit, C.L. Jellis, D.H. Kwon, M. LeWinter, D. Lin, S.A. Luis, V. Mardigyan, J.K. Oh, K.G. Ordovas, E.R. Rodriugez, A.L. Schenone, C.D. Tan, B. Weber, M. Imazio, Pericardial diseases: international position statement on new concepts and advances in multimodality cardiac imaging, JACC Cardiovasc. Imaging 17 (8) (2024) 937–988, https://doi.org/10.1016/j.jcmg.2024.04.010. PMID: 39111992; PMCID: PMC12156183.

[4] M. Imazio, E. Pivetta, S. Palacio Restrepo, P. Sormani, P. Pedrotti, G. Quarta, A. Brucato, E. Bubbico, M. Dal Corso, A. Milazzo, G. Quattrocchi, M. Andriani, L. Lobetti Bodoni, O. Davini, S. Sironi, C. Giannattasio, C. Giustetto, J. Bogaert, Y. Adler, C. Bucciarelli Ducci, G.M. De Ferrari, Usefulness of cardiac magnetic resonance for recurrent pericarditis, Am. J. Cardiol. 125 (1) (2020) 146–151, https://doi.org/10.1016/j.amjcard.2019.09.026. Epub 2019 Oct 11. PMID: 31711636.

[5] E. Conte, C. Agalbato, G. Lauri, S. Mushtaq, A.D. Cia, A. Bonomi, M. Guglielmo, A. Baggiano, M. Gaudenzi-Asinelli, G. Colombo, C. Sforza, P. Agostoni, G. Tamborini, E. Assanelli, G. Pontone, M. Pepi, A. Brucato, D. Andreini, Cardiac MRI after first episode of acute pericarditis: a pilot study for better identification of high risk patients, Int. J. Cardiol. 354 (2022) 63–67, https://doi.org/10.1016/j.ijcard.2022.03.007. Epub 2022 Mar 12. PMID: 35288198.

[6] D. Feng, J. Glockner, K. Kim, M. Martinez, I.S. Syed, P. Araoz, J. Breen, R. E. Espinosa, T. Sundt, H.V. Schaff, J.K. Oh, Cardiac magnetic resonance imaging pericardial late gadolinium enhancement and elevated inflammatory markers can predict the reversibility of constrictive pericarditis after antiinflammatory medical therapy: a pilot study, Circulation 124 (17) (2011) 1830–1837, https://doi.org/10.1161/CIRCULATIONAHA.111.026070. Epub 2011 Oct 3. PMID: 21969014.

[7] A.G. Pavon, R. Martinez Fernandez, D. Arangalage, L. Bergamaschi, N. Maurizi, S. Colombier, S. Rotman, A. Nowacka, J. Bouchardy, J. Schwitter, M. Kirsch, P. Monney, T. Rutz, Prevalence of pericardial late gadolinium enhancement in patients after cardiac surgery: clinical and histological correlations, Circ. Cardiovasc. Imaging 16 (11) (2023) e015606, https://doi.org/10.1161/CIRCIMAGING.123.015606 (Epub 2023 Nov 21. PMID: 37988447).