European Journal of Radiology 195 (2026) 112543

Contents lists available at ScienceDirect

European Journal of Radiology

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

Post-myocardial infarction pericarditis: insight from a cardiovascular magnetic resonance study

Riccardo Cau [a,b,*], Luigi Natale [c], Filippo Cademartiri [d], Giuseppe Falconi [b], Jasjit S Suri [e,f,g,h,i], Antonio Esposito [j], Luca Saba [a,b]

[a] Department of Radiology, Azienda Ospedaliero-Universitaria (A.O.U.) di Cagliari, Cagliari, Italy
[b] University of Cagliari, Cagliari, Italy
[c] Department of Radiological Sciences - Institute of Radiology, Catholic University of Rome, "A. Gemelli" University Hospital, Rome, Italy
[d] IRCCS SYNLAB SDN, Naples, Italy
[e] Department of ECE, Idaho State University, Pocatello, ID 83209, USA
[f] Department of CE, Graphics Era Deemed to be University, 248002 Dehradun, India
[g] University Center for Research & Development, Chandigarh University, Mohali, India
[h] Symbiosis Institute of Technology, Nagpur Campus, Symbiosis International (Deemed University), Pune, India
[i] Stroke Diagnostic and Monitoring Division, AtheroPoint™ , Roseville, CA 95661, USA
[j] IRCCS San Raffaele Scientific Institute, Milano, Lombardia, Italy

ARTICLE INFO

Keywords:
STEMI
Pericarditis
Epistenocardiac Pericarditis
Dressler pericarditis
CMR

ABSTRACT

Objective: This study aims to investigate the demographic, laboratory, clinical, and cardiovascular magnetic resonance (CMR) correlates of post-myocardial infarction pericarditis (PMIP), as well as its impact on outcomes in patients with ST-segment elevation myocardial infarction (STEMI)

Method: This retrospective study included CMR scans of 122 consecutive patients with STEMI (92 males, mean age 64.16 ± 10.35 years). Among them, 33 (26 males, mean age 60.81 ± 11.27 years) exhibited PMIP, defined by the presence of pericardial enhancement on T2-STIR and/or late gadolinium enhancement (LGE) sequences.

Results: Patients with PMIP had a lower left ventricular ejection fraction ($p = 0.017$) and a higher indexed right ventricular end-systolic volume ($p = 0.025$) compared to those without PMIP.

Patients with PMIP exhibited more impaired atrial reservoir strain, global radial strain, and global longitudinal strain, as well as a greater extent of LGE and papillary muscle involvement compared to those without PMIP ($p = 0.001$; $p = 0.002$; $p = 0.012$; $p = 0.001$; $p = 0.001$, respectively). On multivariate analysis, atrial reservoir strain and global longitudinal strain were independently associated with PMIP ($\beta = -2.803$, $p = 0.009$; $\beta = 2.475$, $p = 0.013$). However, the presence of PMIP was not associated with a higher incidence of adverse cardiac events during follow-up.

Conclusion: PMIP is a well-known complications of STEMI patients and is associated with greater cardiac dysfunction, as well as more extensive myocardial damage. Despite these myocardial alterations, PMIP did not result in a higher incidence of adverse cardiac events during follow-up.

1. Introduction

Post-myocardial infarction pericarditis (PMIP) is a recognized complication of ST-segment elevation myocardial infarction (STEMI), with potential implications for cardiac function as a marker of more extensive myocardial damage [1]. PMIP can be classified into two distinct forms: early (epistenocardic) pericarditis, which develops within days of infarction due to local inflammatory responses, and late pericarditis (Dressler's syndrome), which occurs as an autoimmune reaction [2]. The pathophysiological mechanisms underlying PMIP

Abbreviations: CMR, cardiovascular magnetic resonance; LGE, late gadolinium enhancement; LV, left ventricle; PMIP, post-myocardial infarction pericarditis; STEMI, non-anterior ST-segment elevation myocardial infarction.

* Corresponding author at: Department of Radiology, Azienda Ospedaliero Universitaria (A.O.U.), di Cagliari – Polo di Monserrato s.s. 554 Monserrato (Cagliari) 09045, Italy.

E-mail address: riccardo.cau@unica.it (R. Cau).

https://doi.org/10.1016/j.ejrad.2025.112543
Received 1 April 2025; Received in revised form 25 September 2025; Accepted 16 November 2025
Available online 17 November 2025

involve pericardial irritation due to myocardial necrosis, inflammation, and immune-mediated processes [2]. Historically, the incidence of pericarditis was significantly higher in the prethrombolytic era [3]. The diagnosis of PMIP remains challenging due to the paucity of objective clinical manifestations and the absence of universally accepted diagnostic criteria [2]. However, advances in cardiovascular magnetic resonance (CMR) have improved the detection and characterization of myocardial and pericardial involvement [1,4–11]. Specifically, PMIP can be identified by pericardial enhancement on T2-weighted imaging and late gadolinium enhancement (LGE) sequences [1,6]. Despite being a well-recognized complication of myocardial infarction, data on the association between PMIP and clinical outcomes remain limited and controversial [1,3,12–13]. Understanding whether PMIP contributes to adverse cardiac events is crucial for optimizing management and follow-up strategies. In this study, we aim to investigate the demographic, laboratory, clinical, and CMR correlates of PMIP. Furthermore, we seek to explore its impact on long-term clinical outcomes.

2. Material and Method

2.1. Study population

In this retrospective, cross-sectional, observational, single-center study, all consecutive patients with NA-STEMI who underwent CMR between March 3rd 2019, and December 31st 2023 were included.

STEMI was defined according to the ESC/ACCF/AHA/WHF consensus document criteria, which include typical chest pain lasting more than 30 min, persistent ST-segment elevation of at least 1.0 mm in two or more contiguous ECG leads, and elevated cardiac enzyme levels [14].

PMIP was defined as pericardial inflammation detected on T2-weighted and/or LGE images [1]. Fig. 1.

PMIP includes both early infarct-associated pericarditis (e.g., Epistenocardiac Pericarditis) and late post-myocardial infarction pericarditis (e.g., Dressler syndrome). The former occurs within five days of symptom onset, while the latter develops at least two weeks after symptom onset [2].

The presence of PPM infarction was defined as LGE in a papillary muscle head in 2 contiguous LGE CMR slices, and its presence needed to be confirmed on the long-axis LGE CMR slices [15].

Exclusion criteria included: subjects < 18 years; previous myocardial infarction and pericarditis; pre-existing cardiomyopathy; known valvular diseases, and CMR not performed until 30 days after symptom onset.

Cardiovascular risk factors were collected from medical records. Hypertension was defined as a systolic blood pressure of ≥ 140 mmHg or a diastolic blood pressure of ≥ 90 mmHg at rest on more than two occasions, or the use of antihypertensive drugs [16]. Smoking status was defined as current smokers or never smokers. Cholesterol laboratory analyses were conducted following the standard in-house protocol. Diabetes status was assessed using the World Health Organization criteria [17] or an established diagnosis of type 2 diabetes. Obesity was defined as a BMI > 30, as defined by the World Health Organization criteria [18].

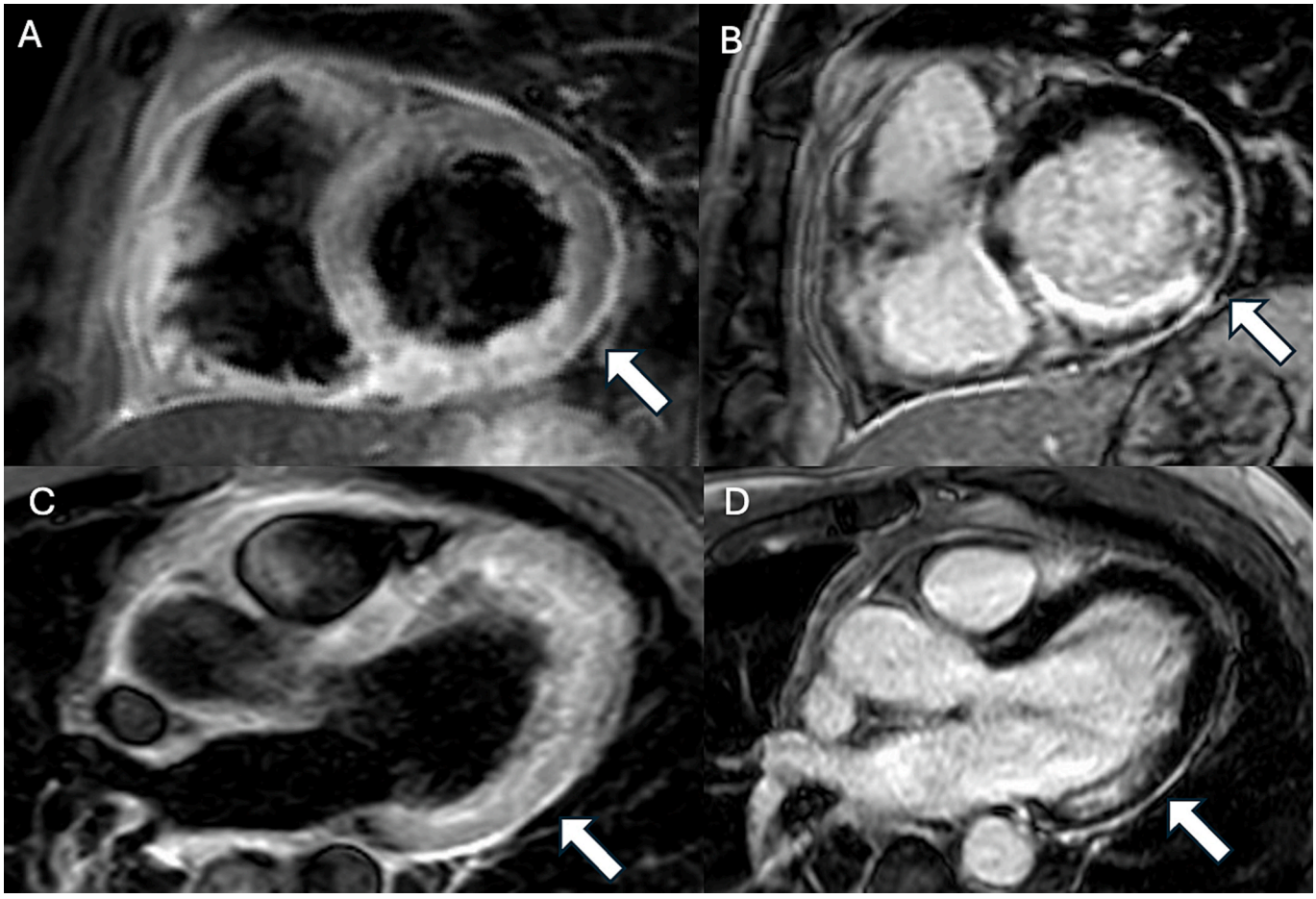

Fig. 1. An example of post-myocardial infarction pericarditis in a patient with ST-segment elevation myocardial infarction. Panels A and B show short-axis late gadolinium enhancement and T2-short tau inversion recovery images of an inferior infarction with post-myocardial infarction pericarditis (arrow in Panel A and B). Panels C and D present long-axis three-chamber view late gadolinium enhancement and T2-short tau inversion recovery images of the same patient, with pericardial enhancement highlighted by an arrow.

All patients were followed up through clinical visits after the CMR examinations, and hospital records were reviewed for clinical events. The primary endpoint was a composite of adverse cardiac events, including nonfatal reinfarction, rehospitalization for heart failure, ventricular arrhythmia, and ischemic stroke. Recurrent acute myocardial infarction was diagnosed according to current guidelines of the European Society of Cardiology [14]. Stroke was defined as an ischemic cerebral infarction resulting from embolic or thrombotic occlusion of a major intracranial artery. Rehospitalization for heart failure was assessed and classified based on the latest guidelines of the European Society of Cardiology [19]. Ventricular arrhythmia was defined as the presence of nonsustained ventricular tachycardia, sustained ventricular tachycardia, or aborted sudden cardiac death.

The Institutional Review Board approval for this retrospective, cross-sectional study was obtained, and patient's consent was waived because of the retrospective nature.

A flowchart demonstrating the application of inclusion and exclusion criteria is provided in Fig. 2.

2.2. CMR acquisition

CMR scans were performed at 16.2 ± 10.5 days after coronary revascularization by using a Philips Achieva dStream 1.5 T scanner system (*Philips Healthcare, Best, The Netherlands*). Anterior coil arrays were used. All cine-images were acquired using a balanced steady-state free precession and retrospective gating during an expiratory breath-hold manoeuvres (TE: 1.7mssec; TR: 3.4msec /flip-angle: 45°, section thickness = 8 mm) in both long-axis (two-, three- and four-chamber view) as well as short-axis plane with whole ventricular coverage from left ventricle (LV) base to apex.

T2-short tau inversion recovery (T2-STIR) images were obtained using triple inversion recovery T2-weighted pulse sequence (TR = 2 RR, TE ≈ 70 msec; flip-angle: 45°, section thickness = 8 mm, FOV 300 × 300 mm2) in long-axis (2-, 3- and 4-chamber view) and short-axis plane with whole ventricular coverage from base to apex.

LGE imaging was performed in both long- and short- axis slices 10–12 min after contrast media injection (Gadovist, Bayer Healthcare) with a dose of 0.15 ml per kg body weight using phase-sensitive inversion recovery sequences (PSIR) (TE: 2.0 ms; TR: 3.4 ms; flip angle: 20°, section thickness = 8 mm) with an inversion time determined using the Look-Locker technique.

2.3. CMR image post-processing

We used the commercially available software system Circle CVI42 (*CVI42, Circle Cardiovascular Imaging Inc., Calgary, Canada*) for CMR-FT data analysis. LV volumes and function were determined on short-axis cine images by manually tracing contours. LV end-diastolic volume and end-systolic volume were obtained from the cine images. The LV ejection fraction (EF) was calculated using the formula: (LV end-diastolic volume – LV end-systolic volume) / LV end-diastolic volume * 100 %.

Offline CMR feature tracking analyses were performed to evaluate peak global longitudinal strain, global radial strain, and global circumferential strain using a 16-segment, software-generated 2D model. Longitudinal strain data were derived from two-, three-, and

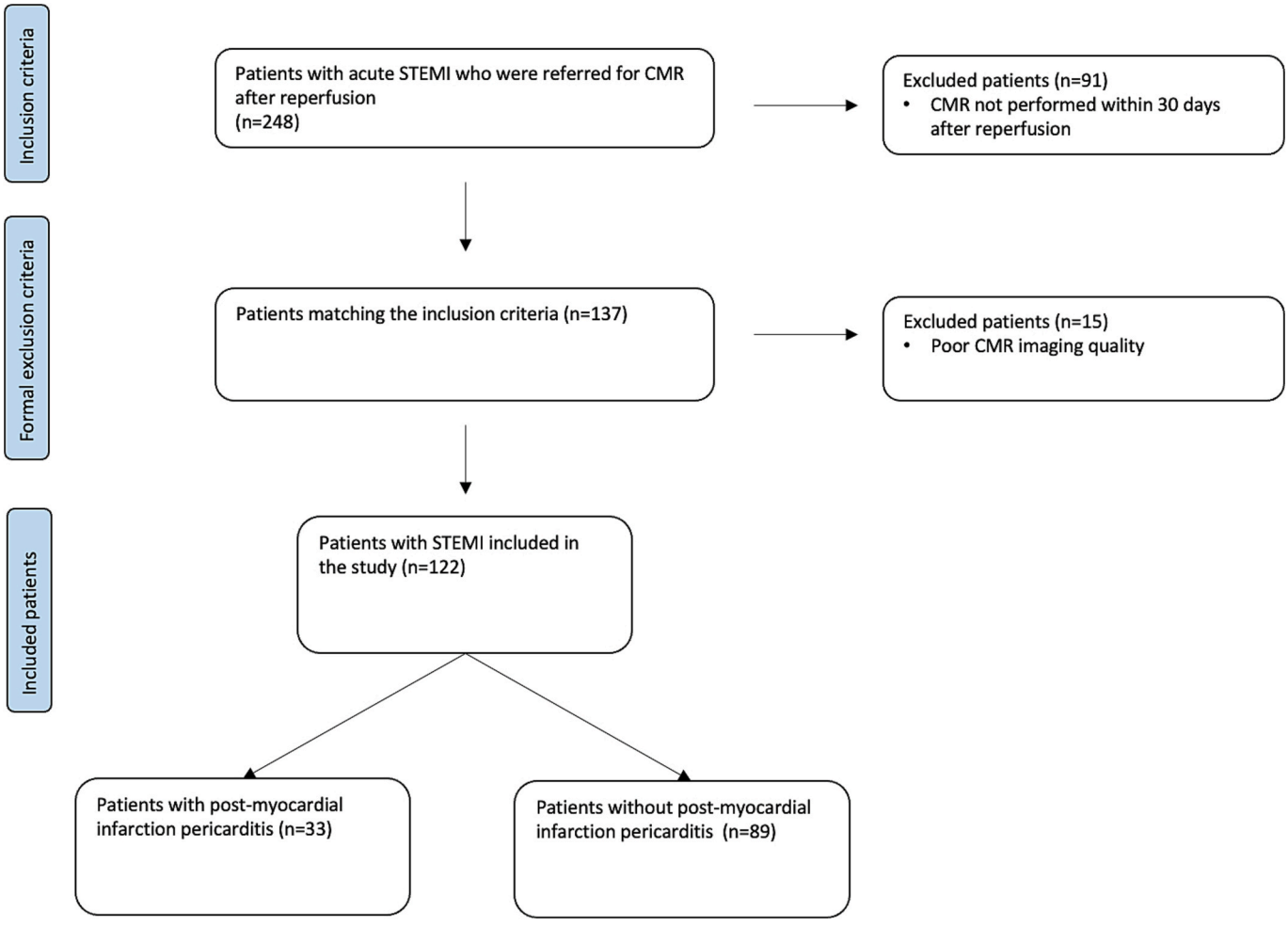

Fig. 2. Flowchart of patients enrolled.

four-chamber long-axis views, while radial and circumferential strain data were obtained from apical, mid-ventricular, and basal short-axis views in all patients. The epi- and endocardial borders were traced in end-diastole on all images, after which an automatic computation was initiated to outline the borders throughout the cardiac cycle. The quality of tracking and contouring was visually validated and manually corrected as needed.

CMR feature tracking analyses of atrial deformation were also conducted offline. The LA endocardial borders were manually traced on long-axis views of the cine images when the atrium was at its minimum volume. Specifically, the four-, three-, and two-chamber views were used to derive LA longitudinal strain, excluding the LA appendage and pulmonary veins. After manual segmentation, the software automatically tracked the myocardial borders throughout the entire cardiac cycle. The quality of tracking and contouring was visually validated and manually corrected by a radiologist with three years of experience in cardiac imaging. The strain curve included three peaks: reservoir, conduit, and booster strain (Fig. 3).

2.4. Statistical analysis

Continuous variables were presented as mean ± standard deviation (SD), while categorical variables were expressed as frequency and percentage (%). Differences between continuous variables were assessed using Welch's *t*-test, and the Kolmogorov–Smirnov test was applied to evaluate the normality of residuals. Categorical variables were analyzed using either the chi-square test or Fisher's exact test, as appropriate.

The association between pericardial involvement and CMR features was analyzed using multivariable linear regression. Variables that reached statistical significance ($p < 0.05$) in the univariable analysis were further examined through multivariable linear regression, adjusting for all statistically significant factors from the univariable analysis.

Collinearity among variables in the multivariable model was assessed using the variance inflation factor (VIF), with values > 5 indicating collinearity, and the tolerance statistic, with values < 0.20 suggesting collinearity. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant. Statistical analyses were performed using JASP software.

3. Results

3.1. Baseline characteristics

A total of 122 patients with STEMI were included, comprising 92 males (75 %) and 30 females (25 %), with a mean age of 64.16 ± 10.35 years. The enrolled patients were divided into two distinct groups based on the presence of PMIP as assessed by CMR. One group consisted of 33 patients with PMIP (26 males, 79 %), with a mean age of 60.81 ± 11.27 years, while the other group comprised 89 patients without PMIP (66 males, 74 %), with a mean age of 65.40 ± 9.77 years.

Among the enrolled PMIP patients, 29 (20 %) presented with early post-infarct pericarditis, while 4 (3 %) had late post-infarct pericarditis.

No significant differences in sex distribution, age range, or cardiovascular risk factors were observed between the groups.

In the PMIP group, the infarct location was anterior in 16 patients (48 %) and non-anterior in 17 patients (51 %). In STEMI patients without PMIP, the infarct location was anterior in 56 patients (63 %) and non-anterior in 33 patients (37 %). No significant differences in infarct location were observed between patients with and without PMIP.

During a median follow-up of 25 months (IQR 12–60), 43 (30 %) patients experienced adverse cardiac events, including 5 cases of nonfatal reinfarction, 25 cases of rehospitalization for heart failure, 8 cases of ventricular arrythmia, and 5 cases of ischemic stroke. A total of 98 patients (69 %) completed the follow-up period without events.

Table 1 presents the comparison between the enrolled groups.

3.2. CMR features in STEMI patients

CMR characteristics of the enrolled patients are summarized in Table 2. STEMI patients with PMIP showed a lower LV ejection fraction (33.90 ± 8.93 % vs. 39.75 ± 12.51 %, $p = 0.017$) and higher end-systolic indexed right ventricular volumes (37.15 ± 18.63 mL/m^2 vs. 28.96 ± 13.79 mL/m^2, $p = 0.025$). No other significant differences were found in left and right ventricular volumes and function. Patients with PMIP demonstrated lower left atrial reservoir strain parameters (14.82 ± 4.9 % vs. 22.13 ± 10.62 %, $p = 0.001$). Regarding ventricular strain parameters, both GLS and GRS were more impaired in STEMI patients with PMIP compared to those without PMIP (GLS: −6.32 ± 3.64 % vs. −9.42 ± 3.82 %, $p = 0.002$; GRS: 11.43 ± 4.87 % vs. 15.86 ± 9.43 %, p

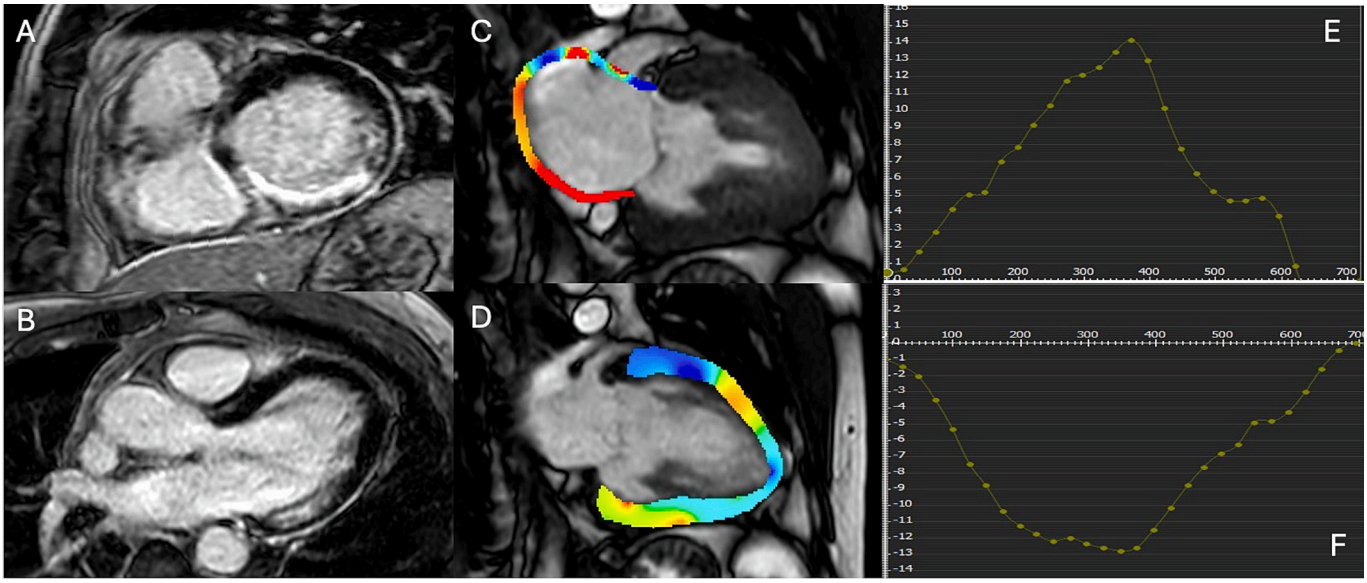

Fig. 3. Representative impairment of atrial and ventricular strain parameters in patients with ST-segment elevation myocardial infarction with post-myocardial infarction pericarditis, as detected by late gadolinium enhancement (panel A and B). Atrial and ventricular strain parameters, along with their corresponding curves, are presented in Panels E–J.

Table 1
Baseline characteristics of patients with and without post-myocardial infarction pericarditis.

Variables	PMIP	No PMIP	p- values
Sex (male), n (%)	26 (79 %)	66 (74 %)	0.60
Age (years)	63.54 ± 9.06	65.40 ± 9.77	0.41
Hypertension, n (%)	14 (42 %)	47 (53 %)	0.46
Dyslipidemia, n (%)	11 (33 %)	32 (36 %)	0.96
Smoke, n (%)	19 (57 %)	41 (46 %)	0.14
Obesity, n (%)	6 (18 %)	17 (19 %)	0.97
Diabetes, n (%)	6 (18 %)	21 (24 %)	0.63
Familiary for CAD, n (%)	7 (21 %)	12 (13 %)	0.23
Infarct location			
Anterior, n (%)	16 (48 %)	51 (57 %)	0.38
Non-anterior, n (%)	17 (51 %)	38 (43 %)	0.39

Abbreviations: CAD coronary artery disease; PMIP post-myocardial infarction pericarditis.

Table 2
CMR findings of reperfused STEMI patients with and without post-myocardial infarction pericarditis.

Variables	PMIP	No PMIP	p-values
LVEF, %	33.90 ± 8.93	39.75 ± 12.51	**0.017**
LVEDV/BSA, mL/m2	121.38 ± 21.39	117.92 ± 29.33	0.23
LVESV/BSA, mL/m2	79.13 ± 36.84	67.24 ± 17.54	0.10
RVEF, %	50.63 ± 13.92	54.51 ± 11.41	0.14
RVEDV/BSA, mL/m2	71.87 ± 25.19	62.42 ± 17.54	0.10
RVESV/BSA, mL/m2	37.15 ± 18.63	28.96 ± 13.79	**0.025**
GLS, %	−6.32 ± 3.64	−9.42 ± 3.82	**0.002**
GCS, %	−8.90 ± 3.82	−10.22 ± 4.77	0.156
GRS, %	11.43 ± 4.87	15.86 ± 9.43	**0.012**
Reservoir, %	14.82 ± 4.9	22.13 ± 10.62	**0.001**
Conduit, %	7.30 ± 4.51	9.90 ± 8.37	0.083
Booster, %	9.30 ± 7.33	11.60 ± 6.13	0.087
LGE extent, number of segments	7.44 ± 2.47	5.36 ± 2.26	**0.001**
Papillary Muscle involvement, n (%)	12 (36 %)	9 (10 %)	**0.001**

Abbreviations: BSA, body surface area; CAD coronary artery disease; EDV, end-diastolic volume; ESV, end-systolic volume; GCS, global circumferential strain; GLS global longitudinal strain; GRS, global radial strain; LGE, late gadolinium enhancement; LV, left ventricle; PMIP post-myocardial infarction pericarditis; SV, stroke volume; RV, right ventricle.

= 0.012).

Moreover, a more extensive myocardial LGE was observed in STEMI patients with PMIP, with a mean myocardial segment involvement of 7.44 ± 2.47 segments compared to 5.36 ± 2.26 segments in patients without PMIP (p = 0.001). Furthermore, STEMI patients with PMIP demonstrated a higher prevalence of papillary muscle involvement by LGE (36 % vs. 10 %, p = 0.001).

3.3. Determinants of post-myocardial infarction pericarditis in STEMI Patients

The univariable and multivariable determinants of PMIP are presented in Table 3 **and** Table 4.

Univariable analysis revealed that left ventricular ejection fraction, end-diastolic indexed right ventricular volume, end-systolic indexed right ventricular volume, GLS, GRS, reservoir strain, number of segments involved by LGE, and papillary muscle involvement by LGE were associated with PMIP (β coefficient = -2.314, p = 0.019; β = 2.203, p = 0.028; β = 2.394, p = 0.017; β = 3.370, p = 0.001; β = -2.369, p = 0.018; β = -3.438, p = 0.001; β = 3.370, p = 0.001; β = 3.321, p = 0.001, respectively).

Further multivariable analysis revealed that GLS and reservoir strain

Table 3
Univariables determinants of post-myocardial infarction pericarditis in STEMI patients.

Variables	Univariable *β coefficent*	*p values*
Sex	−0.527	0.598
Age	−0.951	0.341
Hypertension	−0.732	0.464
Dyslipidemia	−0.047	0.962
Smoke	1.450	0.147
Obesity	0.031	0.975
Diabetes	−0.486	0.627
Familiary for CAD	1.183	0.237
Infart location	0.868	0.386
LVEF, %	−2.314	**0.019**
LVEDV/BSA, mL/m2	0.512	0.602
LVESV/BSA, mL/m2	1.583	0.114
RVEF, %	−1.501	0.133
RVEDV/BSA, mL/m2	2.203	**0.028**
RVESV/BSA, mL/m2	2.394	**0.017**
GLS, %	3.370	**0.001**
GCS, %	1.396	0.163
GRS, %	−2.369	**0.018**
Reservoir, %	−3.438	**0.001**
Conduit, %	−1.731	0.083
Booster, %	−1.696	0.090
LGE extent, number of segments	3.370	**0.001**
Papillary Muscle involvement, n (%)	3.321	**0.001**

Abbreviations: BSA, body surface area; CAD coronary artery disease; EDV, end-diastolic volume; ESV, end-systolic volume; GCS, global circumferential strain; GLS global longitudinal strain; GRS, global radial strain; LGE, late gadolinium enhancement; LV, left ventricle; PMIP post-myocardial infarction pericarditis; SV, stroke volume; RV, right ventricle.

Table 4
Multivariable logistic regression analysis of atrial and ventricular strain parameters for discrimination between patients with and without post-myocardial infarction pericarditis. Multivariable model was adjusted for variables that reached statistical significance (p < 0.05) in the univariable analysis.

Variables	Multivariable *β coefficent*	*p values*
GLS	2.475	0.013
Reservoir	−2.803	0.009

Abbreviations: GLS global longitudinal strain; PMIP post-myocardial infarction pericarditis.

were the only independent determinants of PMIP (β = 2.475, p = 0.013; β = -2.803, p = 0.009, respectively).

4. Discussion

The main findings of this study were as follows: (1) Patients with PMIP exhibited more extensive myocardial damage and dysfunction, as evidenced by the extent of LGE, papillary muscle involvement, and LV ejection fraction impairment; (2) In patients with PMIP, both reservoir function, GLS, and GRS were impaired; (3) GLS and reservoir function were independently associated with PMIP; (4) The presence of PMIP is not associated with future cardiac adverse events.

Patients with PMIP exhibited a more impaired LV ejection fraction and greater LGE extent, consistent with previous literature. Doulaptsis et al. evaluated PMIP in a cohort of 189 consecutive patients who underwent CMR for acute STEMI [1]. They reported that patients with pericarditis had larger infarct sizes and lower ejection fractions compared to those without PMIP, suggesting that CMR evidence of post-infarction pericardial injury may reflect myocardial infarction severity [1]. Furthermore, the observed correlation between papillary muscle involvement on LGE and PMIP further supports the notion that PMIP is associated with more extensive myocardial injury.

Additionally, in patients with PMIP, GLS values were lower compared to STEMI patients without PMIP, and it was independently associated with the presence of PMIP. GLS, which represents subendocardial fibers and quantifies the longitudinal shortening of myocardial fibers, is a well-established marker of subclinical myocardial dysfunction. In the context of PMIP, the more impaired GLS likely reflects more extensive myocardial damage and increased pericardial constraint due to inflammation, leading to myocardial restriction. In this setting, the subendocardial layer is more susceptible to loading conditions and mechanical stress [20–21].

Similarly, PMIP patients exhibited more impaired GRS, reflecting myocardial thickening and thinning in a radial direction toward the center of the LV [10,20], involving both the subepicardial and subendocardial layers. This may reflect more extensive myocardial involvement in this subgroup, potentially affecting fibers from endocardium to epicardium rather than being confined to the subendocardial layer.

Another observation in our study is the association between pericarditis and impaired left atrial strain parameters. Specifically, patients with PMIP exhibited significantly lower reservoir strain values compared to those without PMIP.

The left atrium plays a crucial role in cardiac function by facilitating left ventricular filling through its reservoir, conduit, and booster mechanism [11,22–25]. In STEMI patients with PMIP, we observed a more pronounced impairment in reservoir function, which may reflect decreased atrial relaxation and compliance. This change could be related to altered diastolic properties of the left ventricle and possible effects of pericardial involvement. [4,22].

Under normal conditions, the pericardium helps maintain the Frank-Starling mechanism by modulating ventricular filling [26]. Pericardial inflammation can alters its viscoelastic properties, potentially reducing distensibility and increasing pericardial restraint [27],which may in turn influence atrial strain.

Overall, this findings suggest that PMIP in STEMI may be associated with subtle alterations in atrial mechanics [27–30], possibly reflecting changes in myocardial stiffness or ventricular-atrial interactions in this context.

Despite its association with more extensive myocardial damage and functional atrial and ventricular impairment, our study did not identify a higher incidence of adverse cardiac events in PMIP patients during a median follow-up of 18 months. To the best of our knowledge, our study is the first to comprehensively evaluate both left atrial and ventricular strain parameters using CMR in the context of PMIP while also assessing long-term prognosis. This combined assessment of atrial and ventricular mechanics provides novel insights into the functional impact of PMIP, extending beyond conventional measures such as LV ejection fraction. Our results suggest that, although PMIP may reflect more extensive infarction-related injury and atrial and ventricular impairment, it does not necessarily translate into worse long-term clinical outcomes in terms of future cardiovascular events. By integrating functional strain analysis with infarct characterization, our findings help clarify the potential clinical significance of PMIP. Furthermore, these observations are in line with previous studies reporting variable prognostic significance of pericarditis after myocardial infarction [1,13], contributing to a better understanding of its potential long-term implications.

This study has several limitations that should be acknowledged. First, the relatively small sample size may limit the generalizability of our findings and reduce statistical power, and results should therefore be interpreted with caution. A larger, multicenter cohort would provide more robust conclusions and enhance the external validity of our results. Second, the retrospective design may introduce selection bias, which could influence observed associations. Lastly, although we did not observe an increased incidence of adverse events in patients with PMIP, the follow-up duration and cohort size may be insufficient to fully assess long-term prognostic significance. Future studies with larger populations and longer follow-up are needed to more definitively clarify the clinical impact of PMIP in STEMI patients.

5. Conclusion

Our study highlights that post-myocardial infarction pericarditis in STEMI patients is associated with more pronounced atrial and ventricular dysfunction, as well as greater myocardial injury. Importantly, however, its presence did not translate into an increased risk of adverse cardiovascular events during follow-up. These findings suggest that, despite its association with more severe acute myocardial involvement, post-myocardial infarction pericarditis may not confer additional long-term prognostic burden. Further studies with larger cohorts and extended follow-up are warranted to confirm these observations.

CRediT authorship contribution statement

Riccardo Cau: Writing – original draft, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Luigi Natale:** Writing – original draft, Visualization, Methodology, Investigation, Formal analysis. **Filippo Cademartiri:** Writing – review & editing, Methodology, Investigation. **Giuseppe Falconi:** Writing – review & editing, Resources, Investigation. **Jasjit S Suri:** Writing – review & editing, Validation. **Antonio Esposito:** Writing – review & editing, Supervision, Project administration. **Luca Saba:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] D. Costantinos, G. Kaatje, M.P. Giorgio, F. Anca, J. Stefan, B. Jan, Assessment of Early Post-Infarction Pericardial Injury by CMR, JACC Cardiovasc. Imaging 6 (3) (2013) 411–413, https://doi.org/10.1016/j.jcmg.2012.09.014.
[2] Dr. Tom Sasse Eriksson, Prof. Urs Eriksson FF. https://www.escardio.org/Journals/E-Journal-of-Cardiology-Practice/Volume-15/Post-cardiac-injury-syndrome-aetiology-diagnosis-and-treatment.
[3] E. Correale, A.P. Maggioni, S. Romano, et al., Comparison of frequency, diagnostic and prognostic significance of pericardial involvement in acute myocardial infarction treated with and without thrombolytics, Am. J. Cardiol. 71 (16) (1993) 1377–1381, https://doi.org/10.1016/0002-9149(93)90596-5.
[4] R. Cau, F. Pisu, G. Muscogiuri, et al., Atrial and ventricular strain using cardiovascular magnetic resonance in the prediction of outcomes of pericarditis patients: a pilot study, Eur Radiol. Published Online (2024), https://doi.org/10.1007/s00330-024-10677-9.
[5] G. Muscogiuri, M. Guglielmo, A. Serra, et al., Multimodality Imaging in Ischemic Chronic Cardiomyopathy, J Imaging. 8 (2) (2022), https://doi.org/10.3390/jimaging8020035.
[6] J. Bogaert, M. Francone, Cardiovascular magnetic resonance in pericardial diseases, J. Cardiovasc. Magn. Reson. 11 (1) (2009) 14, https://doi.org/10.1186/1532-429X-11-14.
[7] E. Vidalakis, M. Kolentinis, M. Gawor, M. Vasquez, E. Nagel, CMR in Pericardial Diseases - an Update, Curr. Cardiovasc. Imaging Rep. 13 (4) (2020) 14, https://doi.org/10.1007/s12410-020-9535-z.
[8] N. Aldweib, V. Farah, R.W.W. Biederman, Clinical Utility of Cardiac magnetic Resonance Imaging in Pericardial Diseases, Curr. Cardiol. Rev. 14 (3) (2018) 200–212, https://doi.org/10.2174/1573403X14666180619104515.
[9] R. Cau, A. Palmisano, J.S. Suri, F. Pisu, A. Esposito, L. Saba, Prognostic role of cardiovascular magnetic resonance in Takotsubo syndrome: a systematic review, Eur. J. Radiol. 177 (2024), https://doi.org/10.1016/j.ejrad.2024.111576.
[10] R. Cau, P. Bassareo, F. Cademartiri, et al., Epicardial fat volume assessed with cardiac magnetic resonance imaging in patients with Takotsubo cardiomyopathy, Eur. J. Radiol. 160 (2023) 110706, https://doi.org/10.1016/j.ejrad.2023.110706.
[11] R. Cau, C. Loewe, V. Cherchi, et al., Atrial Impairment as a Marker in Discriminating between Takotsubo and Acute Myocarditis using Cardiac magnetic Resonance, J. Thorac. Imaging 37 (6) (2022) W78–W84, https://doi.org/10.1097/RTI.0000000000000650.
[12] G.H. Tofler, J.E. Muller, P.H. Stone, et al., Pericarditis in acute myocardial infarction: Characterization and clinical significance, Am. Heart J. 117 (1) (1989) 86–92, https://doi.org/10.1016/0002-8703(89)90660-1.

[13] M. Imazio, A. Negro, R. Belli, et al., Frequency and Prognostic significance of Pericarditis following Acute Myocardial Infarction Treated by Primary Percutaneous Coronary intervention, Am. J. Cardiol. 103 (11) (2009) 1525–1529, https://doi.org/10.1016/j.amjcard.2009.01.366.
[14] K. Thygesen, J.S. Alpert, A.S. Jaffe, et al., Fourth universal definition of myocardial infarction (2018), Eur. Heart J. 40 (3) (2019) 237–269, https://doi.org/10.1093/eurheartj/ehy462.
[15] R. Cau, A. Pinna, R. Montisci, et al., Impact of papillary muscle infarction on atrial and ventricular myocardial deformation in non-anterior STEMI patients, Int J Cardiovasc Imaging. Published Online (2025), https://doi.org/10.1007/s10554-024-03317-2.
[16] T. Unger, C. Borghi, F. Charchar, et al., 2020 International Society of Hypertension Global Hypertension Practice Guidelines, Hypertension 75 (6) (2020) 1334–1357, https://doi.org/10.1161/HYPERTENSIONAHA.120.15026.
[17] W.H. Organization, Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: report of a WHO/IDF consultation, Published Online (2006).
[18] K.M. Flegal, M.D. Carroll, R.J. Kuczmarski, C.L. Johnson, Overweight and obesity in the United States: prevalence and trends, 1960–1994, Int J Obes. 22 (1) (1998) 39–47.
[19] T.A. McDonagh, M. Metra, M. Adamo, et al., 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure, Eur. Heart J. 42 (36) (2021) 3599–3726, https://doi.org/10.1093/eurheartj/ehab368.
[20] A. Scatteia, A. Baritussio, C. Bucciarelli-Ducci, Strain imaging using cardiac magnetic resonance, Heart Fail. Rev. 22 (4) (2017) 465–476, https://doi.org/10.1007/s10741-017-9621-8.
[21] P.S. Rajiah, K. Kalisz, J. Broncano, et al., Myocardial Strain Evaluation with Cardiovascular MRI: Physics, Principles, and Clinical applications, Radiographics 42 (4) (2022) 968–990, https://doi.org/10.1148/rg.210174.
[22] R. Cau, P. Bassareo, J.S. Suri, G. Pontone, L. Saba, The emerging role of atrial strain assessed by cardiac MRI in different cardiovascular settings: an up-to-date review, Eur. Radiol. 32 (7) (2022) 4384–4394, https://doi.org/10.1007/s00330-022-08598-6.
[23] G.C.H. Gan, A. Ferkh, A. Boyd, L. Thomas, Left atrial function: Evaluation by strain analysis, Cardiovasc Diagn Ther. 8 (1) (2018) 29–46, https://doi.org/10.21037/cdt.2017.06.08.
[24] K. Inoue, F.H. Khan, E.W. Remme, et al., Determinants of left atrial reservoir and pump strain and use of atrial strain for evaluation of left ventricular filling pressure, Eur. Heart J. Cardiovasc. Imaging 23 (1) (2022) 61–70, https://doi.org/10.1093/ehjci/jeaa415.
[25] Meloni A, Saba L, Positano V, et al. Left atrial strain in patients with β-thalassemia major: a cross-sectional CMR study. Eur Radiol. Published online March 2024. doi: 10.1007/s00330-024-10667-x.
[26] D.H. Spodick, The normal and diseased pericardium: current concepts of pericardial physiology, diagnosis and treatment, J. Am. Coll. Cardiol. 1 (1) (1983) 240–251, https://doi.org/10.1016/s0735-1097(83)80025-4.
[27] B.A. Borlaug, Y.N.V. Reddy, The Role of the Pericardium in Heart Failure: Implications for Pathophysiology and Treatment, JACC Heart Fail. 7 (7) (2019) 574–585, https://doi.org/10.1016/j.jchf.2019.03.021.
[28] J. Buggey, B.D. Hoit, Left atrial strain: measurement and clinical application, Curr. Opin. Cardiol. 33 (5) (2018). https://journals.lww.com/co-cardiology/Fulltext/2018/09000/Left_atrial_strain__measurement_and_clinical.5.aspx.
[29] S.F. Nagueh, S.U. Khan, Left Atrial Strain for Assessment of Left Ventricular Diastolic Function: Focus on Populations with Normal LVEF, JACC Cardiovasc. Imaging 16 (5) (2023) 691–707, https://doi.org/10.1016/j.jcmg.2022.10.011.
[30] K. Jiwon, Y. Brian, C. pm,, et al., Left Atrial Strain Impairment Precedes Geometric Remodeling as a Marker of Post-Myocardial Infarction Diastolic Dysfunction, JACC Cardiovasc. Imaging 13 (10) (2020) 2099–2113, https://doi.org/10.1016/j.jcmg.2020.05.041.