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ST-segment elevation in acute pericarditis and myocardial involvement: electrocardiographic and clinical profiling

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ABSTRACT

Background Pericardium is considered electrically inert, but diffuse ST-elevation is an electrocardiographic marker of acute pericarditis. We hypothesised that ST-elevation in acute pericarditis may reflect underlying myocardial involvement. Accordingly, this study aimed to assess the association between ST-elevation and myocardial involvement in pericarditis patients and to further characterise the clinical features and long-term outcomes of myopericarditis compared with isolated pericarditis.

Methods This longitudinal multicentre study included 351 pericarditis patients (328 recurrent; 180 females), 70/351 with myopericarditis, defined by troponin elevation and/or suggestive cardiac MRI.

Results 121 patients had ST-elevation (34.5%); they were younger: 38 years (23–53) vs 47 (31–58) (median (IQR)) (p<0.001), more often male: 63.6% (77/121) vs 40.9% (94/230) (p<0.001) and had higher C reactive protein values: 92.0 (35–170) vs 58.4 mg/L (15.8–137.5) (median (IQR)) (p=0.002) and less frequent pericardial effusions: 71.1% (86/121) vs 83.5% (192/230) (p=0.004). Myocardial involvement was diagnosed in 70/351 (19.9%) patients, occurring more frequently among those with ST-elevation: 26.4% (32/121), compared with those without: 16.5% (38/230) (p=0.035). ST-elevation predicted myocardial involvement with an OR of 1.82 (95% CI 1.07 to 3.10). Compared with isolated pericarditis, patients with myopericarditis were more frequently male: 61.4% (43/70) vs 45.6% (128/281) (p=0.023) and had a higher prevalence of transient systolic dysfunction: 13.5% (7/52) vs 2.1% (3/141) (p=0.004). During follow-up, myopericarditis patients had a lower remission rate: 18.5% (12/65) vs 31.2% (82/263) (p=0.047) and a higher annual hospitalisation rate (median 0.5 vs 0.4/year, p=0.010), while recurrence rates and disease duration were similar. Treatment strategies, including use of corticosteroids and interleukin 1 blockers, were also comparable.

Conclusions ST-segment elevation in acute pericarditis was associated with myocardial involvement, supporting the concept that the pericardium is electrically inert. Myopericarditis was associated with lower remission rates and slightly higher hospitalisation needs compared to isolated pericarditis, despite otherwise comparable recurrence rates and treatment strategies.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Pericardium is considered electrically inert, but diffuse ST-elevation is an electrocardiographic marker of acute pericarditis.

WHAT THIS STUDY ADDS

⇒ ST-segment elevation was present in about one-third of patients with pericarditis and was associated with a higher likelihood of myocardial involvement, detected by troponin elevation and/or cardiac MRI.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study confirms that the pericardium is electrically inert and further characterises patients with pericarditis and ST-segment elevation. Compared with patients without ST-elevation, these patients are younger, more often male, more frequently hospitalised at the first presentation and have a longer disease course. They have higher C reactive protein levels and less frequent pericardial effusion. Myocardial involvement should be considered in these patients.

INTRODUCTION

Pericarditis and myocarditis often present with overlapping forms, and the term inflammatory myopericardial syndrome (IMPS) has been recently proposed.[1] It is estimated that myocardial involvement occurs in approximately 30% of cases of acute idiopathic pericarditis.[2 3]

The pericardium is considered electrically inert, and consequently, isolated pericardial inflammation should not result in ST-segment changes. Therefore, the presence of such alterations should reflect concomitant involvement of the adjacent myocardial layers.[4] Few studies have systematically investigated whether ST-segment elevation in patients diagnosed with pericarditis identifies a clinically meaningful subgroup with a higher likelihood of myocardial involvement and different clinical outcomes. In a cohort of 166 patients with pericarditis and/or myocarditis confirmed by cardiac MRI and/or endomyocardial biopsy, Imazio *et al* reported a higher prevalence of ECG abnormalities, particularly ST-segment elevation, in patients with myocarditis.[5]

Myopericarditis is diagnosed when pericarditis is associated with evidence of myocardial injury, inflammation or new-onset ventricular dysfunction.[1 6 7]

At a more basic diagnostic level, electrocardiography (ECG) remains essential: it is one of the required diagnostic criteria for acute pericarditis, with widespread ST-segment elevation representing a major diagnostic feature according to ESC guidelines.[1 8] However, in routine clinical practice, the diagnostic and prognostic implications of ST-segment elevation in patients with pericarditis remain unclear.

Building on these previous observations, our study aimed to describe the prevalence of ST-segment elevation in a large cohort of patients with recurrent pericarditis, exploring its association with myocardial involvement in routine clinical practice. Moreover, we aimed to better characterise clinical profile, management and long-term outcomes of myopericarditis compared with isolated pericarditis.

METHODS

This longitudinal (retrospective–prospective) multicentre study included patients followed for pericarditis at four Italian referral centres for pericardial diseases between January 2019 and May 2025. Patients were considered eligible if aged >18 years at enrolment, with a medical history of acute or recurrent pericarditis (idiopathic, viral, post-vaccine or post-cardiac injury), as defined by the 2015 and also the 2025 European Society of Cardiology Guidelines.[1 8] In addition, patients were required to have a minimum dataset available, including at least electrocardiographic data at the index episode.

Vaccine-related COVID-19 pericarditis was defined as an acute pericarditis occurring in recently vaccinated individuals, with no alternative identifiable cause and symptom onset within 4 weeks of vaccine administration.[9] COVID-19 infection-related pericarditis was defined as an acute pericarditis occurring within 6 weeks of SARS-CoV-2 infection.[10]

ST-segment elevation was defined as diffuse concave ST-segment elevation consistent with acute pericarditis, according to ESC guideline criteria and as documented in the original ECG reports. ECG was routinely obtained at the first clinical evaluation, either in the emergency department or in the outpatient setting. As this was a real-world cohort, the timing of ECG acquisition was not standardised with respect to symptom onset.

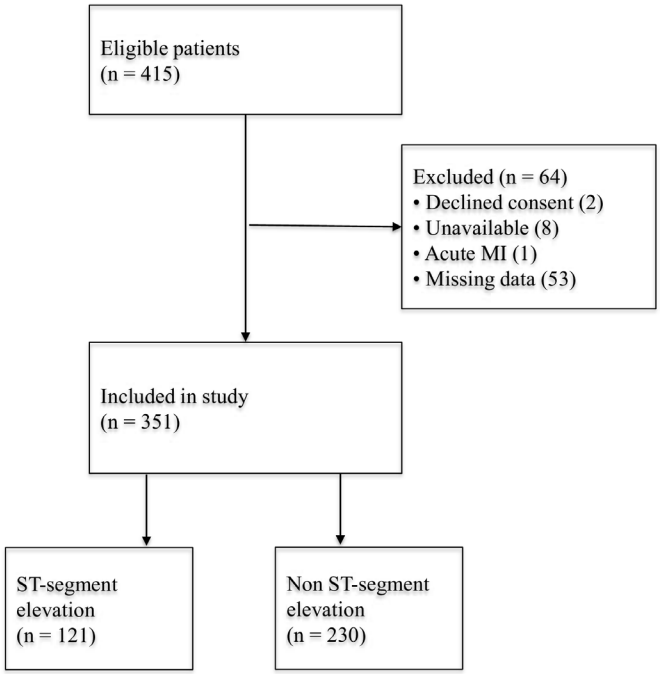

Figure 1 Study flow. Flow chart of patient selection and study population. MI, myocardial infarction.

Myocardial involvement was diagnosed in the presence of troponin levels exceeding the 99th percentile upper reference limit and/or MRI myocardial abnormalities. All subjects were systematically tested for troponin at least once. MRI findings considered suggestive of myocardial involvement for this study were restrictive: myocardial oedema/inflammation by T2-weighted short-tau inversion recovery imaging and/or myocardial late gadolinium enhancement (LGE). More subtle abnormalities such as T1 and T2 mapping and extracellular volume quantification were not considered, since MRI was not done according to a predefined standardised protocol but was carried out according to local medical judgement and availability, and at non-standardised time intervals.

Exclusion criteria included bacterial or neoplastic pericarditis, underlying connective tissue diseases or auto-inflammatory diseases.

Of the 415 eligible patients, 351 were included; reasons for exclusion (n=64) were: 2 patients declined consent, 8 patients were unavailable, 1 patient was excluded from the study due to an acute myocardial infarction close to the onset of pericarditis, and for 53 patients, a minimum data set was unavailable (figure 1).

This study followed the Strengthening the Reporting of Observational Studies in Epidemiology reporting guidelines[11] and all data have been processed in full compliance with privacy regulations and according to the principles of the 2008 Declaration of the Helsinki Committee for

Human Rights. All enrolled patients provided written informed consent to study participation.

Data collection was primarily based on medical records, in particular for ECG, troponin and MRI results, as well as patient interviews conducted during outpatient visits or by telephone follow-up. The following data were collected: age, sex, pericarditis aetiology, comorbidities and first-attack characteristics (ECG findings, C reactive protein (CRP) value, white blood cell count, neutrophil-to-lymphocyte ratio, presence of troponin elevation, haemoglobin value, pericardial effusion, pleural effusion, cardiac tamponade, hospitalisation during the acute episode and type and dosage of administered treatments). MRI results, when performed, were also recorded. The decision to perform MRI depended on the treating physicians and on local hospital protocols and availability. It should be noted that the first pericarditis episode could have occurred prior to the enrolment period. Follow-up data included the total number of recurrences, hospitalisations related to pericardial disease, use of interleukin (IL-1) inhibitors at any point during the disease course (in Italy only anakinra is available for pericarditis), achievement of disease remission (defined as the ability to discontinue all pericarditis-related treatments for at least 6 months due to a stable clinical course) and last available follow-up information (date and ongoing treatment, if any). For recurrent pericarditis patients, disease duration was calculated from the first attack to therapy cessation for those who could stop all therapy without recurrence for at least 6 months or to follow-up date for those still on treatment. The follow-up duration was calculated from the time of the first documented episode to the date of the last available clinical evaluation.

Laboratory parameters and ECG were relative to the first attack or, if not available, relative to the subsequent episode in which they were recorded.

End points

The primary aim of this study was to investigate whether the prevalence of myopericarditis differs according to the presence or absence of ST-segment elevation and to determine the diagnostic performance of ST-segment elevation in identifying myocardial involvement.

The secondary outcomes were:

1. To assess differences in ECG abnormalities, clinical presentation, epidemiological and laboratory characteristics between patients with myopericarditis and those with isolated pericarditis, focusing on demographic features, ST-segment elevation, CRP levels, presence of pericardial effusion, pleural effusion, cardiac tamponade and systolic dysfunction (defined as a left ventricular ejection fraction<55%) at first pericarditis attack.
2. To evaluate potential prognostic differences between the two groups, including remission rate, disease duration, annual rate of recurrences and hospitalisations. For this analysis, only patients with at least one documented recurrence were considered.
3. To compare the treatment received at first presentation between the two groups.
4. To compare the number of patients treated with IL-1 inhibitors (anakinra) at any time during the disease course in the two groups, as well as the number of those still on anti-IL-1 therapy at the last available follow-up.

Taken together, these objectives were designed to offer an integrated assessment of the diagnostic value of ST-segment elevation, as well as the clinical characteristics, treatment patterns and long-term outcomes of patients with myopericarditis compared with those with isolated pericarditis.

STATISTICAL ANALYSIS

Categorical variables were reported as absolute frequencies and percentages. Normality of distributions for quantitative variables was assessed using the Kolmogorov-Smirnov test; as all tested variables were non-normally distributed, data were expressed as medians and IQRs.

Comparisons between groups were performed using two-tailed tests, considering a p value<0.05 as statistically significant. For univariate analysis of categorical variables between myopericarditis and isolated pericarditis groups, the χ^2 test or Fisher's exact test was applied, as appropriate. The Mann-Whitney U test was employed for comparing non-normally distributed continuous variables between independent groups.

Sample size was calculated with a two-sided α=0.05, based on the prevalence of myocardial involvement among patients with and without ST-segment elevation reported in three previous cohorts (ranging from 9.8% vs 2.8% to 73.2% vs 20.8%).[5 12 13] According to these estimates, the required sample size to achieve 80% and 90% power ranged, respectively, from approximately 32/85 to 42/113 patients in the ST-elevation/non-ST-elevation groups.

Data were collected using Research Electronic Data Capture. Statistical analyses were performed using SPSS Statistics V.29.

RESULTS

The study population consisted of 351 patients, with a median follow-up period of 4.2 years (IQR 2.1–7.4). 23 patients were evaluated for an isolated pericarditis attack during the enrolment period, while 328 were followed for recurrent disease. The demographic characteristics of the study population are summarised in table 1. By the end of the follow-up period, only one patient had died, from causes unrelated to pericarditis.

Among the 351 patients, ST-segment elevation was observed in 121/351 cases (34.5%), while 230/351 (65.5%) had no ST-elevation. Representative ECGs of patients with pericarditis, with and without ST-segment elevation, are shown in figure 2. Myopericarditis, defined by troponin elevation and/or myocardial oedema and/or myocardial LGE by MRI, occurred more frequently in

Table 1 Characteristics of study population (351 patients)

Females, n (%)	180 (51.3%)
Age at first attack (years), median (IQR)) (range)	43 (29–57) (1–87)
Caucasian, n (%)	341 (97.2%)
Recurrent pericarditis, n (%)	328 (93.4%)
Aetiology, n (%)	
Idiopathic	271 (77.2%)
Post-pericardiectomy	23 (6.6%)
PCI related	10 (2.8%)
Other (viral, post-vaccines)	47 (13.4%)
► Post-COVID-19	► 13 (3.7%)
► Post-SARS-CoV-2 vaccine	► 14 (4.0%)
► Other viral infections	► 13 (3.7%)
► Others	► 7 (2.0%)
Main symptom at presentation	
Chest pain	299/313 (95.5%)
Dyspnoea	14/313 (4.5%)
Fever at first attack	146/249 (58.6%)
Myocardial involvement	70 (19.9%)
ST-elevation	121 (34.5%)

PCI, percutaneous cardiac intervention.

patients with ST-elevation (26.4%) compared with those without (16.5%) (table 2). The presence of ST-segment elevation was associated with a nearly twofold increased odds of myocardial involvement (OR 1.82, 95% CI 1.07 to 3.10, p=0.035). As a diagnostic marker, ST-elevation showed a sensitivity of 45.7% and a specificity of 68.3% in detecting myocardial involvement, with a positive predictive value of 26.4% (95% CI 19.4% to 34.9%) and a negative predictive value of 83.5% (95% CI 78.1% to 87.7%). The corresponding likelihood ratios (LR) were LR +1.44 and LR −0.79.

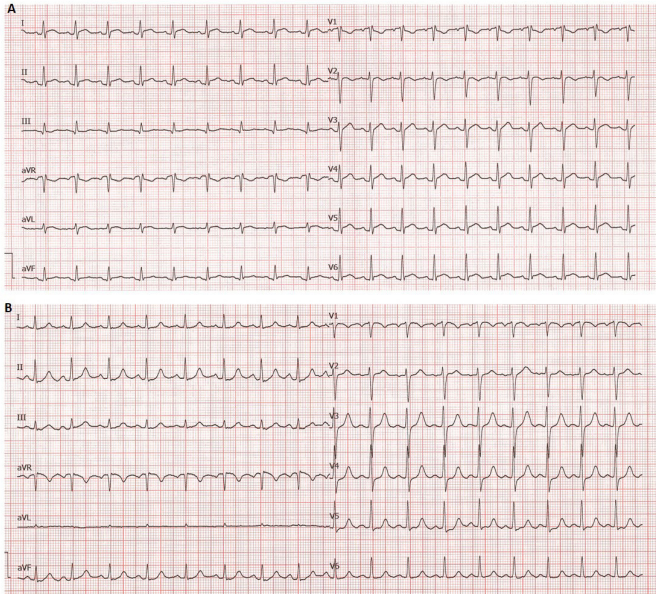

Figure 2 Electrocardiographic findings in acute pericarditis. (A) ECG with mild, nearly diffuse concave ST-segment elevation; mild ST depression in aVR and V1 and inferior leads; PR-segment depression are also evident. (B) ECG from a patient without ST-segment elevation, presenting non-specific repolarisation abnormalities. Both recordings demonstrate sinus tachycardia, commonly observed in inflammatory conditions. LEGEND: aVR, aVL and aVF indicate augmented voltage right arm, left arm and foot leads.

In table 2, the characteristics of patients with and without ST-segment elevation are summarised. Patients with ST-segment elevation were younger, more often male and more frequently presented with elevated CRP levels compared with those without ST-elevation, while pericardial effusion was less frequent. From a management and prognostic perspective, patients with ST-segment elevation were more often hospitalised at the first attack and, among those with recurrent pericarditis, experienced a longer disease duration.

Regarding the secondary endpoints, myopericarditis was diagnosed in 70 of the 351 (19.9%) patients included in the study (table 3). The diagnosis was established as follows: exclusively through troponin elevation in 32 out of the 70 (45.7%) patients, exclusively through MRI imaging in 15 out of 70 (21.4%) patients or through the combination of imaging and troponin in 23 out of 70 (32.9%) patients. It is worth noting that only 134/351 (38.2%) patients underwent MRI imaging (48 in the myopericarditis group and 86 in the isolated pericarditis group), performed at variable intervals after the first episode or subsequent recurrences, based on clinical judgement and local availability.

In table 3, the characteristics of patients with myopericarditis and isolated pericarditis are presented. Compared with patients with isolated pericarditis, those with myopericarditis were more frequently male, presented more often with abnormal ECG findings including ST-segment elevation and had a higher prevalence of atrial fibrillation during the disease course. Transient left ventricular systolic dysfunction (EF<55%) was also more common in the myopericarditis group. From a management standpoint, patients with myopericarditis were more frequently hospitalised at the first episode. Regarding long-term outcomes, they showed a lower remission rate and a higher annual rate of hospitalisations compared with those with isolated pericarditis, with similar recurrence rate and disease duration.

DISCUSSION

This large multicentre study is, to our knowledge, the first to specifically evaluate whether an electrocardiographic hallmark of acute pericarditis (diffuse ST-segment elevation) is associated with myocardial involvement. In our cohort, myocardial involvement was identified in approximately 20% of patients.

Although the pericardium is considered electrically inert, the spread of inflammation to the epicardial

Table 2 Univariate analysis comparing clinical characteristics, treatment and prognosis of patients with and without ST-elevation

	ST-elevation (n=121)	No ST-elevation (n=230)	P value
Epidemiology			
Age at first attack (years), median (IQR) (range)	38 (23–53) (10–87)	47 (31–58) (1–85)	**<0.001**
Male sex, n (%)	77/121 (63.6%)	94/230 (40.9%)	**<0.001**
Myopericarditis	32/121 (26.4%)	38/230 (16.5%)	**0.035**
► Troponin elevation, n (%)	25/121 (20.7%)	30/230 (13.0%)	0.066
► Myocardial oedema and/or LGE at MRI, n (%)	19/121 (15.7%)	19/230 (8.3%)	**0.046**
ECG			
AF—at first attack only, n (%)	8/121 (6.6%)	22/230 (9.6%)	0.424
AF—at any time, n (%)	13/121 (10.7%)	27/230 (11.7%)	0.861
Inflammatory markers			
CRP>10 mg/L at first attack, n (%)	106/121 (87.6%)	175/230 (76.1%)	**0.007**
CRP at first attack (mg/L), median (IQR) (range)	92.0 (35.0–170.0) (0.0–454.0)	58. 4 (15.8–137.5) (0.0–657.4)	**0.002**
WBC ($\times 10^9$/L) at first attack, median (IQR) (range)	10.4 (8.1–13.0) (1.7–28.5)	9.6 (7.6–12.0) (3.5–28.0)	0.083
N/L ratio at first attack, median (IQR) (range)	4.6 (3.2–7.1) (0.4–26.5)	4.0 (2.3–5.9) (0.0–39.0)	0.069
Echocardiogram			
EF<55%, n (%)	4/65 (6.2%)	6/128 (4.7%)	0.735
Pericardial effusion, n (%)	86/121 (71.1%)	192/230 (83.5%)	**0.004**
Pleural effusion, n (%)	43/121 (35.5%)	71/230 (30.9%)	0.336
Cardiac tamponade, n (%)	11/121 (9.1%)	18/230 (7.8%)	0.687
Hospitalisation during the first attack	83/121 (68.6%)	127/230 (55.2%)	**0.022**
Treatments (at any time of disease course)			
Colchicine, n (%)	118/121 (97.5%)	224/230 (97.4%)	0.345
NSAIDs, n (%)	118/121 (97.5%)	228/230 (99.1%)	0.720
Steroids, n (%)	93/121 (76.9%)	173/230 (75.2%)	0.794
Steroids maximum dose (mg), median (IQR) (range)	25.0 (25.0–50.0) (4.0–100)	25.0 (15.0–50.0) (2.5–100)	0.247
Patients who started anakinra, n (%)	34/121 (28.1%)	56/230 (24.3%)	0.444
Patients still in anakinra, n (%)	32/34 (94.1%)	40/56 (71.4%)	0.052
Recurrent forms of disease	112 (92.6%)	216 (93.9%)	0.653
Prognosis (evaluated only in the 328 patients with recurrent pericarditis)	ST-elevation (n=112)	No ST-elevation (n=216)	
Remission rate (≥6 months), n (%)	35/112 (31.3%)	59/216 (27.3%)	0.520
Disease duration (months), median (IQR) (range)	53.9 (25.0–101.6) (3.5–776.2)	40.0 (21.2–70.8) (2.8–478.6)	**0.031**
Annual rate of recurrences (n/year), median (IQR) (range)	1.1 (0.6–1.9) (0.1–6.9)	1.1 (0.7–1.9) (0.1–7.3)	0.573
Annual rate of hospitalisations (n/year), median (IQR) (range)	0.4 (0.2–0.9) (0.0–6.9)	0.4 (0.0–1.0) (0.0–8.6)	0.307

*Values in bold indicate statistical significance (P < 0.05).
AF, atrial fibrillation; CRP, C reactive protein; EF, ejection fraction; LGE, late gadolinium enhancement; N/L, neutrophils/lymphocytes; NSAIDs, non-steroidal anti-inflammatory drugs; WBC, white blood cells.

myocardium can induce electrical abnormalities, particularly ST-segment elevation, due to the creation of voltage gradients between inflamed and non-inflamed myocardial areas.[14] Our study confirms that ST-segment elevation in acute pericarditis, and particularly in recurrent pericarditis, is associated with a significantly higher prevalence of troponin elevation and/or myocardial involvement on MRI, suggesting an association between this characteristic ECG finding of pericarditis and mild myocardial involvement, usually without clinical manifestations.

However, since a substantial proportion of patients with isolated pericarditis also exhibited ST-segment elevation, this feature alone does not reliably distinguish between isolated pericardial and myocardial involvement in classical acute pericarditis.

Subclinical myocardial involvement should therefore be interpreted in light of the available diagnostic

Table 3 Univariate analysis comparing clinical characteristics, treatment and prognosis of patients with myopericarditis versus isolated pericarditis

	Myopericarditis (n=70)	Isolated pericarditis (n=281)	P value
Epidemiology			
Age at first attack (years), median (IQR) (range)	44 (27–62) (6–84)	43 (30–55) (1–87)	0.463
Male sex, n (%)	43/70 (61.4%)	128/281 (45.6%)	**0.023**
ECG			
ST-elevation, n (%)	32/70 (45.7%)	89/281 (31.7%)	**0.035**
No ST-elevation	38/70 (54.3%)	192/281 (68.3%)	**0.035**
Other ECG alterations			
PR depression, n (%)	11/70 (15.7%)	49/281 (17.4%)	0.860
Non-specific ST/T changes, n (%)	29/70 (41.4%)	106/281 (37.7%)	0.585
AF—at first attack only, n (%)	13/67 (19.4%)	17/245 (6.9%)	**0.003**
AF—at any time, n (%)	14/67 (20.9%)	26/245 (10.6%)	**0.019**
Inflammatory markers			
CRP>10 mg/L at first attack, n (%)	62/70 (88.6%)	219/281 (77.9%)	0.160
CRP at first attack (mg/L), median (IQR) (range)	92.0 (32.8–146.0) (0.2–657.4)	67.0 (19.0–150.0) (0.0–454.0)	0.225
WBC (×10⁹/L) at first attack, median (IQR) (range)	10.1 (8.1–13.4) (3.5–28.5)	9.7 (7.8–12.0) (1.7–28.0)	0.139
N/L ratio at first attack, median (IQR) (range)	4.4 (2.7–6.5) (0.4–39.0)	4.1 (2.9–6.1) (0.0–36.8)	0.906
Echocardiogram			
EF<55%, n (%)	7/52 (13.5 %)	3/141 (2.1 %)	**0.004**
Pericardial effusion, n (%)	52/69 (75.4 %)	226/277 (81.6 %)	0.241
Pleural effusion, n (%)	19/68 (27.9 %)	95/271 (35.1 %)	0.316
Cardiac tamponade, n (%)	7/70 (10.0 %)	22/281 (7.8 %)	0.627
Hospitalisation during the first attack	58/70 (82,9%)	152/281 (54,1%)	**<0.001**
Treatments (at any time of disease course)			
Colchicine, n (%)	67/70 (95.7%)	279/281 (99.3%)	0.056
NSAIDs, n (%)	67/70 (95.7%)	275/281 (97.9%)	0.659
Steroids, n (%)	52/70 (74.3%)	214/281 (76.2%)	0.756
Steroids maximum dose (mg), median (IQR) (range)	25.0 (14.4–50.0) (5.0–75.0)	25.0 (25.0–50.0) (2.5–100)	0.854
Patients who started anakinra, n (%)	20/70 (28.6%)	70/281 (24.9%)	0.543
Patients still in anakinra, n (%)	16/20 (80.0%)	56/70 (80.0%)	0.620
Recurrent forms of disease	65 (92,9%)	263 (93,6%)	0.790
Prognosis (evaluated only in the 328 patients with recurrent pericarditis)	Myopericarditis (n=65)	Isolated Pericarditis (n=263)	
Remission rate (≥6 months), n (%)	12/65 (18.5%)	82/263 (31.2%)	**0.047**
Disease duration (months), median (IQR) (range)	39.6 (20.0–85.7) (3.2–269.6)	43.2 (22.5–82.0) (2.8–776.2)	0.717
Annual rate of recurrences (n/year), median (IQR) (range)	1.0 (0.6–1.9) (0.1–6.7)	1.1 (0.7–1.9) (0.1–7.3)	0.626
Annual rate of hospitalisations (n/year), median (IQR) (range)	0.5 (0.2–1.2) (0.0–5.9)	0.4 (0.1–0.9) (0.0–8.6)	**0.010**

*Values in bold indicate statistical significance (P < 0.05).
AF, atrial fibrillation; CRP, C reactive protein; EF, ejection fraction; N/L, neutrophils/lymphocytes; NSAIDs, non-steroidal anti-inflammatory drugs; WBC, white blood cells.

tools, with different implications in clinical practice and research settings. High-sensitivity troponin assays may improve detection, and cardiac MRI likely plays a key role in defining myocardial involvement in IMPS.[15] Consistently, recent data using global longitudinal strain demonstrated reversible subclinical myocardial dysfunction during acute pericarditis despite preserved left ventricle ejection fraction and normal troponin.[16] Nonetheless, our findings remain clinically relevant, as they reflect contemporary real-world practice.

Of note, in our study, ST-segment elevation was associated with higher CRP levels, whereas no significant differences in inflammatory markers were observed between patients with myopericarditis and those with isolated

pericarditis. These findings suggest that the presence of myocardial involvement is not necessarily associated with a greater systemic inflammatory burden, in line with previous observations reporting no correlation between troponin elevation and CRP levels in acute pericarditis.[13] Interestingly, patients with ST-segment elevation, despite higher CRP levels compared with those without ST-segment elevation, less frequently presented with pericardial effusion. This apparent dissociation between systemic inflammation and local fluid accumulation may reflect a different inflammatory pattern, preferentially affecting the epicardial or subepicardial myocardium, leading to ECG changes and myocardial injury without necessarily resulting in significant pericardial fluid accumulation. In this context, pericardial effusion may be more closely related to pericardial permeability and fluid dynamics than to the overall systemic inflammatory burden.

As expected, systolic dysfunction was significantly more frequent in myopericarditis, although all patients fully recovered ventricular function after the acute phase. This confirms that even in the presence of transient reduced ejection fraction, IMPS typically follows a benign course, in contrast to isolated myocarditis, where persistent dysfunction or arrhythmic complications are common.[17]

From an epidemiological perspective, male sex was significantly more frequent among patients with myocardial involvement and/or ST-segment elevation, confirming a known sex-related predisposition.[13 18]

Regarding long-term outcomes, the myopericarditis group presented a lower remission rate than patients with isolated pericarditis, despite similar disease duration and recurrence rate. Treatment strategies were comparable across groups, including the use of corticosteroids and IL-1 inhibitors. Notably, patients with myopericarditis were not more frequently treated with second-line agents or long-term anti-IL-1 therapy. Overall, these findings suggest that myopericarditis generally follows a clinical course similar to isolated pericarditis, although with a somewhat less favourable remission profile and a greater tendency to hospitalisation. Careful long-term follow-up is therefore warranted, while more aggressive treatment or intensive monitoring may not be necessary in the absence of additional risk factors.

From a clinical perspective, this study adds relevant insights for patient management. ST-segment elevation in acute pericarditis identifies a subgroup of patients with a higher likelihood of myocardial involvement. Its presence may therefore prompt a more careful diagnostic assessment and eventually closer follow-up. These findings support improved risk stratification, without implying the need for routine treatment escalation in otherwise low-risk patients.

This study has several limitations that should be acknowledged. First, although the overall sample size was substantial, some statistical analyses may have been underpowered due to the relatively small number of patients with ST-segment elevation and/or myopericarditis compared with the isolated pericarditis group. This could have limited the ability to detect small but clinically relevant differences between the two populations.

Second, data regarding the first pericarditis episode were not available for all patients, particularly those with a long-standing disease history. This lack of completeness may have introduced recall or documentation bias, particularly for variables such as inflammatory markers.

Third, the observational nature of the study limited standardisation of diagnostic testing. In particular, MRI was performed in a minority of patients according to local practice, which may have led to an underestimation of the prevalence of myocardial involvement.[6 19]

An additional limitation relates to the setting of the study. The majority of patients were enrolled at tertiary referral centres specialised in pericardial diseases, which commonly manage patients with recurrent, refractory or complicated pericarditis referred from other institutions. This referral pattern likely explains the high prevalence of recurrent forms in our cohort and may partly account for the lower remission rates observed in patients with myocardial involvement, compared with previously published cohorts predominantly including first-episode pericarditis. In particular, this difference may explain the apparent discrepancy with earlier studies reporting a more benign long-term course of pericarditis with myocardial involvement, such as the cohort described by Imazio et al. in 2013.[2] Therefore, our findings should be interpreted within the context of a selected population representative of real-world tertiary care rather than unselected acute pericarditis. At the same time, this setting provides complementary information to previous studies, by offering insights into the clinical course of patients with recurrent or more complex pericardial disease, who are under-represented in earlier cohorts.

CONCLUSIONS

This study highlights that ST-segment elevation in acute pericarditis is present in one-third of patients and associated with myocardial involvement detected as troponin elevation and/or with MRI. Myocardial involvement was observed in about one-fifth of patients, and these patients showed a lower remission rate, despite similar inflammatory burden, recurrence rate and disease duration. Likewise, treatment strategies, including the use of corticosteroids and IL-1 inhibitors, did not differ significantly.

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Ethics approval This study involves human participants. The study was approved by the ethics committee 'Comitato Etico Milano Area 1' protocol number: 35480/2023, approval date: 31 July 2023. Please note that at the time of the beginning of this study, this committee was fully operational and competent for ethical oversight. Following the national reorganisation of ethics committees in Italy, the original committee has been discontinued. The responsibilities have since been transferred to a newly designated territorial ethics committee, 'Comitato etico territoriale Lombardia 1', whose contact details are provided below. Participants gave informed consent to participate in the study before taking part.

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