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Outcomes of acute pericarditis with an inflammatory phenotype

Massimo Imazio [a,b,*], Antonio Brucato [c], George Lazaros [d], Alessandro Andreis [d], Ruggiero Mascolo [c], Silvia Berra [c], Emilia Lazarou [d], Costas Tsioufis [d], Andrea Solano [d], Valentino Collini [b]

[a] Department of Medicine (DMED), University of Udine, Italy
[b] Cardiology and Cardiothoracic Department, University Hospital "Santa Maria della Misericordia" (ASUFC) Udine, Italy
[c] Department of Medicine, Ospedale Sacco, Milano, and University of Milano Statale (UNIMI), Milan, Italy
[d] University Cardiology, Azienda Ospedaliero-Universitaria Città della Salute, Torino, Italy

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ABSTRACT

Background: Patients with pericarditis may show elevation of C-reactive protein (CRP) and pericardial effusion at presentation. There are limited data on the prognostic implications of this inflammatory phenotype.
Objectives: Aim of the present study is to evaluate the outcome of the inflammatory phenotype in a cohort of patients with acute pericarditis.
Methods: Observational cohort study of consecutive adult patients with acute pericarditis in 4 referral centers for pericarditis (Athens, Milan, Turin, Udine).
Results: Our cohort included 918 patients with acute pericarditis (median age of 56, IQR 28 years, 55.6 % females). The etiology of pericarditis was respectively idiopathic in 82.1 %, post-cardiac injury syndrome in 9.3 %, and systemic inflammatory disease in 4.9 % of cases. CRP elevation was detected at presentation in 778 cases (84.7 %), an inflammatory phenotype (CRP elevation and pericardial effusion) was found in 557 patients (60.7 %). Baseline medical therapy included a NSAID in 74.9 %, colchicine 70.9 %, and corticosteroids 25.1 % of cases. After a mean follow-up of 22.5 months, patients with an inflammatory phenotype had a higher recurrence rate at 18 months (respectively 46.0 % vs. 31.0 %; $p < 0.0001$), and a shorter recurrence-free survival (Log-rank $p = 0.0001$). In multivariable analysis the inflammatory phenotype presentation was independently associated with an increased risk of recurrences (OR 2.005, 95 % CI 1.454 to 2.765; $p < 0.0001$).
Conclusions: The inflammatory phenotype of presentation of acute pericarditis is associated with an increased risk of recurrences, highlighting the importance of timely individualized therapy and close follow-up for these patients.

1. Introduction

Patients with acute pericarditis may present with or without C-reactive protein (CRP) elevation [1] and with or without associated inflammatory features (e.g. fever, pericardial and pleural effusion) [2,3]. Such presentation with one or more additional inflammatory features represents an "inflammatory phenotype". The inflammatory phenotype of acute pericarditis (AP) can be described as CRP elevation accompanied by the presence of pericardial effusion at presentation. Patients with an inflammatory phenotype of pericarditis may share common immunopathogenic pathways that could be targeted by novel biologic anti-inflammatory therapies [4–8]. Currently it is unknown if this phenotype of presentation could be associated with a worse prognosis [3]. Previous findings have shown that CRP elevation at presentation could be associated with more recurrences and a shorter recurrence-free survival [1].

Aim of the present paper is to assess the clinical features and outcomes of patients with an inflammatory phenotype at presentation, defined by at least CRP elevation and pericardial effusion.

* Corresponding author at: Department of Medicine (DMED), University of Udine and Cardiothoracic Department, University Hospital "Santa Maria della Misericordia" (ASUFC), Piazzale Santa Maria della Misericordia 15, 33100 Udine, Italy.
E-mail address: massimo.imazio@uniud.it (M. Imazio).

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2. Material and methods

2.1. Population and study design

This is an observational, retrospective, cohort study including consecutive adult patients (>18 years) with acute pericarditis from 4 referral centres for pericarditis (Hyppokration Cardiology, University of Athens, Internal Medicine Department, Ospedale Fatebenefratelli, University of Milan, University Cardiology, Città della Salute e della Scienza di Torino, and Cardiothoracic Department, University Hospital Santa Maria della Misericordia, Udine) from January 2013 to June 2022. The University Hospital Santa Maria della Misericordia was the coordinating center. Acute and recurrent pericarditis was diagnosed according to the 2015 European Society of Cardiology (ESC) guidelines for the diagnosis and treatment of pericardial diseases [8]. According to these guidelines criteria, the clinical diagnosis of acute pericarditis is based on 2 out of 4 criteria (pericarditic chest pain, pericardial rubs, ECG changes, and new or worsening pericardial effusion). For the diagnosis of recurrence, the same criteria were considered with a documented first episode of acute pericarditis and a symptom-free interval of 4–6 weeks or longer. We considered as definition of the inflammatory phenotype the presence of CRP elevation (defined as >1.0 mg/dl) accompanied by pericardial effusion at presentation. The study was approved by the Institutional Review Board of the Department of Medicine at the University of Udine. Informed consent was provided by study participants.

2.2. Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) or median and interquartile range (IQR), according to the data distribution. Categorical variables were presented as absolute numbers and percentages. The Student *t*-test or the Mann–Whitney *U* test was used to compare continuous variables between groups, as appropriate. Comparison of categorical variables was performed by Chi-squared analysis or the Fisher exact test, as appropriate. A multivariable analysis was performed to assess baseline clinical features associated with an inflammatory phenotype of presentation. Event-free survival was defined as freedom from recurrence. Event-free survival was determined using the Kaplan–Meier approach for matched subgroups with or without an inflammatory phenotype. Comparisons between survival distributions were performed using the log-rank test. Analyses were performed using MedCalc Software (MedCalc Software Ltd., Ostend, Belgium; https://www.medcalc.org; 2024)

3. Results

3.1. Baseline characteristics of the studied population

Overall, 918 patients were included in this study. The baseline clinical features of the whole studied population, and subgroups according to the presence or absence of an inflammatory phenotype of presentation are reported in Table 1. Patients had a median age of 56 (IQR 28) years, 510 were females (55.6 %), and all were Caucasian. The reported etiology of pericarditis was idiopathic in 754 patients (82.1 %), post-cardiac injury syndrome in 85 cases (9.3 %), and autoimmune or systemic inflammatory disease in 45 cases (4.9 %). CRP elevation was detected at presentation in 778 cases (84.7 %), an inflammatory phenotype with CRP elevation and presence of pericardial effusion was detected in 557 patients (60.7 %). Baseline medical therapy include a non-steroidal anti-inflammatory drug (NSAID) and colchicine 651 cases (70.9 %), a NSAID alone in 37 cases (4.0 %). Moreover, 230 patients (25.1 %) received corticosteroid therapy and colchicine because of intolerance, contraindication or partial response to NSAID/colchicine.

Table 1
Comparison of baseline clinical features and recurrences of the overall studied population and according to the inflammatory phenotype of presentation.

Feature	All (n = 918)	Inflammatory Phenotype (n = 557)	Non-Inflammatory Phenotype (n = 361)	p*
Age (mean ± SD)	54.6 ± 19.2	50.3 ± 18.7	57.4 ± 19.0	<0.001
Female gender	510 (55.6 %)	282 (50.6 %)	228 (63.1 %)	<0.001
Idiopathic etiology	754 (82.1 %)	457 (82.0 %)	608 (82.2 %)	0.931
Post-cardiac injury etiology	85 (9.3 %)	59 (10.6 %)	26 (7.2 %)	0.084
Autoimmune etiology	45 (4.9 %)	29 (5.2 %)	16 (4.4 %)	0.596
Chest pain	790 (86.1 %)	464 (83.3 %)	326 (90.3 %)	0.003
Dispnoea	326 (35.5 %)	252 (45.2 %)	74 (20.5 %)	<0.001
Fever	387 (42.2 %)	277 (49.7 %)	110 (30.5 %)	<0.001
Pericardial rubs	149 (16.2 %)	87 (15.6 %)	62 (17.2 %)	0.533
ST segment elevation	312 (34.0 %)	155 (27.8 %)	157 (43.5 %)	<0.001
CRP elevation	778 (84.7 %)	557 (100.0 %)	221 (19.1 %)	<0.001
Pericardial effusion	626 (68.2 %)	557 (100.0 %)	69 (61.0 %)	<0.001
Pleural effusion	387 (42.2 %)	326 (58.5 %)	61 (16.9 %)	<0.001
Recurrences	368 (40.1 %)	256 (46.0 %)	112 (31.0 %)	<0.001

SD = standard deviation; CRP = C-reactive protein.
* = comparison between patients with or without the inflammatory phenotype.

3.2. Follow-up data and outcomes

After a mean follow-up of 22.5 months, 368 patients (40.0 %) had recurrences. The mean number of recurrences was 1.5 per patient (range 1–5). Patients with an inflammatory phenotype had a higher recurrence rate at 18 months (respectively 256/557 cases (46.0 %) vs. 112/361 cases (31.0 %; $p < 0.0001$), and a shorter recurrence-free survival (Fig. 1, Log rank $p = 0.0001$). No cases of cardiac tamponade and constrictive pericarditis were recorded. A comparison of clinical features of patients with or without recurrences during follow-up is reported in Table 2.

In multivariable analysis (Table 3), including potential significant variables derived from univariate analysis (Table 2), such as age, etiology, fever, pleural effusion, inflammatory phenotype, and use of corticosteroids, increasing age was associated with a reduced risk of recurrences (OR 0.976, 95 % CI 0.968 to 0.983; $p < 0.0001$), while the inflammatory phenotype presentation was associated with an increased risk of recurrences (OR 2.005, 95 % CI 1.454 to 2.765; $p < 0.0001$).

4. Discussion

Recurrent pericarditis is the most challenging complication of pericarditis in clinical practice, affecting up to 50 % of those with multiple

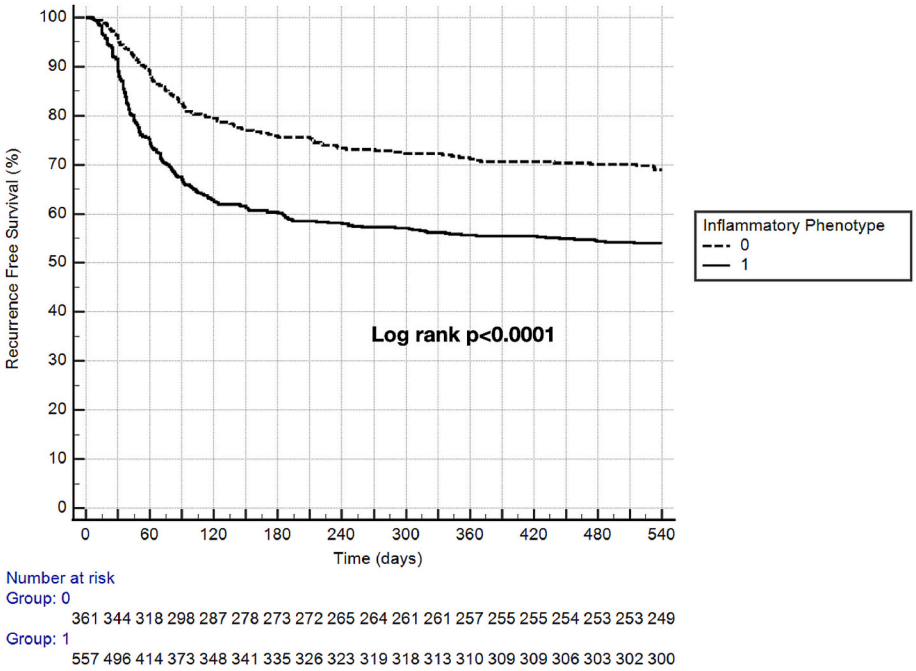

Fig. 1. Recurrent-free survival in patients with or without an inflammatory phenotype.

Table 2
Comparison of clinical features in patients with or without recurrences during follow-up.

Feature	Recurrent course $n = 368$	Not Recurrent course $n = 550$	p
Age (mean ± SD)	50.5 ± 18.6	57.4 ± 19.2	<0.0001
Female	193 (52.4 %)	318 (57.8 %)	0.2046
Idiopathic etiology	292 (79.3 %)	462 (84.0 %)	0.072
Post-pericardiotomy	35 (9.5 %)	50 (9.1 %)	0.830
Autoimmune	22 (6.0 %)	23 (4.2 %)	0.217
Fever	176 (47.8 %)	211 (38.4 %)	0.005
CRP elevation	319 (86.7 %)	459 (83.5 %)	0.182
Pleural Effusion	175 (47.6 %)	212 (38.6 %)	0.007
Inflammatory Phenotype	256 (69.6 %)	301(54.7 %)	<0.001
Colchicine use	266 (72.3 %)	385 (70.0 %)	0.456
Corticosteroids use	104 (28.3 %)	126 (22.9 %)	0.067

Table 3
Risk factors for recurrences in the studied population according to multivariable analysis.

Variable	Odds ratio	95 % CI	P
Age	0.9755	0.9681 to 0.9831	<0.0001
Idiopathic etiology	0.7242	0.50523 to 1.0380	0.0790
Fever	1.1458	0.8598 to 1.5269	0.3530
Pleural effusion	1.2623	0.9243 to 1.7238	0.1430
Inflammatory phenotype	2.0049	1.4539 to 2.7649	<0.0001
Baseline corticosteroids	1.2527	0.9003 to 1.7430	0.1813

recurrences [9–12]. In this observational study we investigated the outcomes of patients with acute pericarditis and an inflammatory phenotype of presentation showing a higher risk of recurrences in such patients.

The pathogenesis of pericarditis is complex with the interaction of genetic factors, currently mainly associated with autoinflammatory conditions [13–16], an infectious or non-infectious trigger and the individual inflammatory and immune response [2,17]. Most patients with acute pericarditis have elevation of CRP, a marker of activation of systemic inflammation. The inflammatory response can then trigger immune mediated responses perpetuating the disease.

In this study all consecutive patients with acute pericarditis were enrolled including patients with post-pericardiotomy syndromes and autoimmune diseases. In patients with an underlying disease, effective control of this disease contributes to the reduction of the recurrence rate.

A timely and more individualized treatment can allow to target medical therapies and improve outcomes. Patients with evidence of activation of systemic inflammation can show different clinical features, including signs (fever), and laboratory and instrumental findings (elevation of CRP, pericardial and pleural effusion). In order to make simple the clinical evaluation, in this paper we defined the inflammatory phenotype as the combination of CRP elevation and pericardial effusion [18,19].

In the largest published series on this topic, the inflammatory phenotype was studied in patients with recurrent pericarditis without a specific evaluation of the outcomes [3]. A timely recognition of the presence of an inflammatory phenotype should allow a timely individualized therapy, and a close follow-up, since these patients may have more recurrences during follow-up. Patients with an inflammatory phenotype could receive more targeted therapies at the inflammatory response (e.g. colchicine and anti-IL-1 agents) [2,4,5,7,20]. Anti-IL-1 agents (anakinra and rilonacept) are novel anti-inflammatory therapies and biologic agents for pericarditis targeting IL-1 [21]. They have been shown to be particularly efficacious in reducing recurrence rates and improving symptomatic outcomes in patients with corticosteroid-dependent and colchicine resistant recurrent pericarditis with evidence of CRP elevation at presentations and seems particularly suited for patients with recurrent pericarditis unable to control pericarditis with conventional medical therapies, including NSAIDs, colchicine, and corticosteroids [22–27]. In this setting combining these agents with colchicine could improve outcomes providing a sequential block of the inflammasome pathway leading to IL-1 [28].

This study has potential limitations. It is a retrospective study that included patients from tertiary referral centers for pericarditis, thus we cannot exclude the possible preferential referral of more complicated cases. Nevertheless, it is currently the largest study on patients with acute pericarditis with and without an inflammatory phenotype.

In conclusion, the inflammatory phenotype of presentation of acute pericarditis is associated with a increased risk of recurrences, highlighting the importance of timely individualized therapy, and close

follow-up for these patients.

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None.

CRediT authorship contribution statement

Massimo Imazio: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Investigation, Formal analysis, Data curation, Conceptualization. **Antonio Brucato:** Writing – review & editing, Visualization, Validation, Supervision, Data curation. **George Lazaros:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Investigation, Data curation. **Alessandro Andreis:** Writing – review & editing, Visualization, Validation, Supervision, Investigation, Data curation. **Ruggiero Mascolo:** Writing – review & editing, Visualization, Validation, Supervision, Investigation, Data curation. **Silvia Berra:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Data curation. **Emilia Lazarou:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Data curation. **Costas Tsioufis:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Data curation. **Andrea Solano:** Writing – review & editing, Visualization, Validation, Supervision, Investigation, Data curation. **Valentino Collini:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

None.

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