Cardiac magnetic resonance in the assessment of pericardial abnormalities: a case series

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Background
Cardiac magnetic resonance (CMR) has a unique role in evaluating pericardial disease, permitting non-invasive tissue analysis, and haemodynamic assessment.

Case summary
In Case 1 of recurrent pericarditis, CMR confirmed reactivation of inflammation with late gadolinium enhancement and native T1/T2 mapping techniques, prompting therapeutic changes. In constrictive pericarditis, CMR is the only modality capable of differentiating a subacute potentially reversible form (Case 2), from a chronic, burnt out irreversible phase characterized by constrictive physiology (Case 3).

Discussion
Cardiac magnetic resonance is an effective tool to tailor individual therapy, particularly in cases of recurrent and constrictive pericarditis. Late gadolinium enhancement provides diagnostic and prognostic information, and multi-parametric mapping has emerged as a promising tool with incremental diagnostic value.

Keywords
Cardiac magnetic resonance • Pericardial disease • Pericarditis • Constrictive pericarditis • Case series

ESC Curriculum
2.3 Cardiac magnetic resonance • 6.6 Pericardial disease

Learning points
• Cardiac magnetic resonance with late gadolinium enhancement and native T1/T2 mapping has the unique ability to provide a differential diagnosis in the whole spectrum of pericardial diseases;
• Cardiac magnetic resonance provides incremental diagnostic and prognostic information and the possibility of individualizing therapy;
• Cardiac magnetic resonance is particularly useful in cases of suspected recurrent pericarditis and constrictive pericarditis.

Introduction
Inflammatory diseases of the pericardium constitute a pathological spectrum ranging from acute pericarditis to chronic constrictive pericarditis (CP). 1

The interaction between inflammation and haemodynamics is optimally characterized with multimodality non-invasive imaging [echocardiography, cardiac computed tomography (CCT), and cardiac magnetic resonance (CMR)]. 1

Cardiac magnetic resonance represents the most versatile modality due to high spatial resolution and image contrast devoid of ionizing radiation. Cardiac magnetic resonance allows tissue characterization, haemodynamic assessment, and provides prognostic information. 1–3
We describe three clinical cases that illustrate the indispensable role of CMR in the evaluation of the broad spectrum of pericardial diseases.

### Timeline

| Time       | Progress                                                                 |
|------------|--------------------------------------------------------------------------|
| **Case 1** |                                                                          |
| Day 1      | Acute chest pain                                                        |
|            | Admission to the emergency department                                    |
| Day 2      | Diffuse concave-upward ST-segment elevation in electrocardiography       |
|            | Blood analysis with elevated inflammatory and cardiac biomarkers         |
|            | Transthoracic echocardiography showed moderate pericardial effusion     |
|            | Thoracic radiography with mild pleural effusion                         |
| Days 2–7   | Admission to the cardiology department                                   |
|            | Started colchicine 0.5 mg/day and ibuprofen                              |
| Day 9      | Fever and recurrence of chest pain                                       |
| Day 10–30  | Hospital readmission                                                     |
|            | Thoracic radiography with moderate pleural effusion (increased)         |
|            | Transthoracic echocardiography showed mild pericardial effusion         |
|            | Investigation of auto-immune, auto-inflammatory, and bacterial infectious causes: negative |
| Day 37     | Started prednisone 0.5 mg/kg/day                                         |
| Month 3    | Discharge with prednisone and colchicine                                |
| Month 3    | Tapering 5 mg/day of prednisone each week                                |
| Month 4    | Start prednisone                                                         |
|            | Recurrence of chest pain                                                |
| Month 4    | Admission to the emergency department                                    |
|            | Transthoracic echocardiography without pericardial pleural effusion     |
|            | No alterations on electrocardiography                                    |
|            | Blood analysis with elevated inflammatory markers (Polymerase chain reaction 14.3 mg/dL, 12,700 leukocytes) and negative cardiac biomarkers |
| Month 25   | Cardiac magnetic resonance (CMR) study showed myopericarditis: Late gadolinium enhancement (LGE)+ T1 and T2 mapping + Restarted prednisolone |

### Continued

| Time       | Progress                                                                 |
|------------|--------------------------------------------------------------------------|
| Case 2     | 10 years prior to the pericardial disease diagnosis                       |
| Day 1      | Progressive worsening dyspnoea, peripheral oedema, and increased abdominal perimeter |
| Month 4    | Cardiology outpatient clinic first observation                           |
|            | Transthoracic echocardiography with pericardial thickening and respiratory septal shift |
| Month 4    | CMR study: LGE + T1 mapping + Pericardial thickness of 8 mm              |
|            | Constrictive physiology                                                  |
|            | Computed tomography study: no pericardial calcification and severe pleural effusion |
|            | Blood analysis: mild increased in C-reactive protein (4.3 mg/dL) with normal white blood cell count, haemoglobin 13.6 g/dL, hyponatraemia (129 mmol/L), and normal serum immuno-electrophoresis |
| Month 5    | Submitted to thoracentesis—pleural effusion analysis compatible with neoplastic origin |
| Case 3     | First hospitalization for heart failure and atrial flutter               |
| Day 1      | Transthoracic echocardiography with preserved left ventricle ejection fraction, diastolic dysfunction, and bialtral enlargement |
| Month 25   | Worsening dyspnoea (NYHA II–III), peripheral oedema, and ascites         |
|            | Cardiology outpatient clinic observation and started aetiology investigation |
| Month 26   | Transthoracic echocardiography and cardiac catheterization with signs of pericardial constriction |
**Case reports**

**Case 1**

A 23-year-old female, with no relevant past medical history except Polycystic Ovary Syndrome, previously hospitalized with polyserositis (pericardial and pleural effusions) and treated with colchicine and prednisolone had recurrent chest pain after tapering and discontinuing corticosteroid therapy. While the physical examination, trans-thoracic echocardiography and electrocardiography showed no abnormalities, a CMR study (Figure 1) confirmed recurrent pericarditis and concomitant myocardial involvement (myopericarditis) with preserved left ventricle ejection fraction (69%). Cardiac magnetic resonance showed increased pericardial thickness (maximum 2.5 mm) with marked pericardial late gadolinium enhancement (LGE). As it may be hard to differentiate between pericardial LGE and fat in this sequence, native T1 mapping distinguishes pericardial inflammation (high signal—bright image) from fat (low signal—dark image)—Figure 1D,E. Myocardial involvement was demonstrated by the increased T2 signal [52 ms—normal value 47.69 (4.28) ms, Figure 1G] and LGE with a subepicardial pattern in the basal segment of left ventricular inferolateral wall. Given these results which were compatible with acute inflammation, the patient was commenced on immunosuppressive therapy (prednisolone 15 mg/day). A comprehensive aetiological study was carried out, which excluded bacterial infections, namely tuberculosis and zoonotic pathogens. A viral aetiology was suspected, although viral serology and molecular assays did not show any acute viral infection. While spontaneous ovarian hyperstimulation can be associated with polyserositis, thyroid function and hormonal investigations were in the normal range. The initial autoimmunity panels were negative and the patient had no other concomitant symptoms (such as arthralgias). The patient is being followed up in the Rheumatology department and autoimmunity panels remain unchanged and progressive weaning from corticosteroid therapy was initiated without recurrence of symptoms.

**Case 2**

A 75-year-old-man with a diagnosis of heart failure with preserved ejection fraction, with medical history of multiple cardiovascular risk factors (type 2 diabetes mellitus, arterial hypertension, smoking, and dyslipidaemia), chronic obstructive pulmonary disease, and prostate cancer (treated with local surgery and hormone therapy), was referred for a CMR study due to worsening dyspnoea, peripheral oedema, pleural effusion, ascites, and evidence of pericardial thickening on echocardiography. Cardiac magnetic resonance confirmed a marked increase in pericardial thickness (8 mm) (Figure 2), signs of constrictive physiology (respiratory pleural shift in real-time cine imaging—Video 1, dilatation of inferior vena cava with late reverse diastolic flow in with phase-contrast study) with ongoing inflammation (severe pericardial LGE and elevated native T1 signal in the pericardium). The study was complemented with a CT scan that excluded pericardial calcification. The analysis of the pleural fluid was compatible with neoplastic aetiology. The patient died for a non-cardiac cause before the conclusion of all investigations.

**Case 3**

A 59-year-old-female with a previous history of acute decompen-sated heart failure in the context of atrial tachycardia, 2 years ago, was admitted due to progressive exertional dyspnoea [New York Heart Association (NYHA) Class III]. At physical examination, the patient had elevated jugular venous pressure and peripheral oedema and a transthoracic echocardiography and subsequent cardiac catheterization was diagnostic of constrictive pericarditis (CP). A CMR study (Figure 3) confirmed a chronic form of CP without myocardial involvement: thickened pericardium (8 mm-anterior; 6 mm-lateral), biastral enlargement (left atrium> right atrium), constrictive physiology (‘septal bounce’, respiratory septal shift—Video 2) and pericardial adherence (lack of slippage between the parietal and visceral pericardium in tissue-tagging sequences—Video 3). After partial pericardectomy, the patient’s functional class improved to NYHA Class I and CMR demonstrated reduction in pericardial thickness and, by real time cine imaging, significant improvement in constrictive physiology (Figure 3J–M). The aetiology remains idiopathic, since autoimmune and infectious causes (including analysis of bronchoalveolar lavage fluid acquired by bronchoscopy) were excluded. The histopathological analysis of the pericardium reported characteristics of a chronic, non-inflammatory, constrictive phase (dense fibrosis and calcification).

**Discussion**

Although transthoracic echocardiography is the first-line imaging modality in pericardial diseases its inability to characterize tissue is a limitation.1

In Case 1, CMR confirmed reactivation of pericarditis with techniques of LGE and mapping. LGE is highly sensitive for identifying inflammation and neovascularization of the pericardium.1,3

In patients with recurrent pericarditis, quantification of LGE provides incremental diagnostic value to conventional clinical criteria, predicting recurrence at 6 months.6 Furthermore, treatment tailored by CMR findings results in quicker remission and less steroid therapy.7

Inflammation may also be depicted by multiparametric mapping. High values of native T1 and T2 mapping (bright image) suggest pericardial oedema/inflammation. The comparison of mapping signal through consecutive CMR during patient’s follow-up allows the

| Time          | Progress                                      |
|---------------|-----------------------------------------------|
| Month 29      | Bronchoscopy: bronchoalveolar lavage fluid    |
|               | analysis excluded infectious pulmonary disease |
| Month 30      | CMR study:                                   |
|               | Pericardial thickness of 8 mm                 |
|               | Constrictive physiology                       |
| Month 36      | Partial pericardectomy                        |
| Month 37      | 2nd CMR study:                                |
|               | No constrictive physiology                   |
|               | Regression of symptoms                        |
evaluation of treatment response and relapse, without the use of gadolinium-containing contrasts. Native T1 has also the potential of detailed tissue characterization, allowing the distinction between pericardial enhancement and pericardial fat (both positive in LGE sequences). The diagnosis of CP is often challenging, and CMR may constitute the modality of choice, since transthoracic echocardiography cannot reliably visualize the pericardium unless there is marked thickness (>5 mm). Cardiac magnetic resonance allows the measurement of the thickness of the entire pericardium and the evaluation of the extent of pericardial involvement. Although pericardial thickness >5–6 mm is highly specific (~100%) for constriction, up to 18% of patients with histologically proven CP have pericardial thickness <2 mm.

Thickening is usually most pronounced over the right heart (right ventricle and anterior atrioventricular groove), as seen in Case 3. Cardiac magnetic resonance also contributes to the physiological assessment by documenting ventricular interdependence, using real time free-breathing sequences in short axis views where a septal bounce can be detected on deep inspiration. Cardiac magnetic resonance better differentiates small effusions from pericardial thickening and has higher temporal resolution than CT, enabling detection of rapid haemodynamic processes that characterize constrictive physiology. In addition, phase-contrast acquisition in caval veins demonstrates constrictive physiology with diminished or absent forward, or even reversed systolic flow, and increased early diastolic forward flow and late backward flow.

**Figure 1** Steady-state free precession (SSFP) sequence in long-axis (A) and short-axis (B). (C) Black blood T1 weighted (double inversion recovery) showing maximum pericardium thickness of 2.5 mm (arrow). Native T1 mapping [980 ms—normal value 990.66 (17.5) ms] in short-axis (D) and long-axis (E), contributing to differentiate between fat (red star) from fluid/inflammation (bright/arrow). (F) Native T2 mapping sequence showing pericardial inflammation (arrow). (G) Increased myocardium native T2 mapping [52 ms—normal value 47.69 (4.28) ms], suggestive of concomitant myocardial oedema. (H–K) Late gadolinium enhancement (LGE) sequence in short-axis (median and apical slices) and long-axis views showing hyperenhancement in the pericardium (orange arrows) and in myocardium (subepicardial pattern in the basal infero-lateral wall—blue arrow).
Tissue tagging facilitates the identification of patients with CP by defining visceral–parietal adherence patterns as the failure for orthogonally aligned tagged stripes to ‘slide’ past each other.9

The versatility of CMR permits staging the pericardial disease. The demonstration of oedema and/or inflammation on T2 images and LGE sequences points towards an acute inflammatory process. The absence of high signal in T2 images suggests a healing process, without active inflammation. If pericardial enhancement remains in LGE acquisition, without increases T2 signal, a subacute process should be taken into consideration. In the late stage, when fibrotic component predominates, neither T2 nor LGE show increased signal (Table 1).

A case series with CMR and histopathologic correlation in CP showed pericardial LGE correlates with greater fibroblastic proliferation, chronic inflammation, and neovascularization, whereas patients without pericardial LGE had more pericardial fibrosis and calcification, and lesser degrees of pericardial thickening.2

Video 1 Cardiac magnetic resonance study of Case 2: cine sequences showing respiratory septal shift in inspiration.

Figure 2 Cardiac magnetic resonance study of Case 2 constrictive pericarditis. (A) Steady-state free precession sequence, noticing the large left pleural effusion (*). (B) Black blood T1 weighted Turbo Spin Echo sequences showing circumferential thickening of the pericardium (arrow). (C) Computed tomography scan excluded calcification of the pericardium. (D, E) Real-time cine imaging functional assessment of ventricular interdependence (D) normal septal position during expiration and (E) marked leftward septal shift seen immediately after deep inspiration (white arrows demonstrate the relative change in cavity size due to the septal shift). (F) Phase contrast study showing reverse diastolic flow in inferior vena cava (star). (G and H) Native T1 mapping with high native T1 values in the pericardium. (I and J) Late gadolinium enhancement sequence in short-axis view and four-chamber view with severe hyperenhancement in the pericardium (orange arrow) and in the myocardium (subendocardial pattern in the basal inferolateral wall—blue arrow).
Reversible CP was associated with pericardial and systemic inflammation, with reduction in pericardial LGE with anti-inflammatory therapy, concomitant with resolution of CP physiology and symptoms. Finally, CMR can contribute to pre-operative planning, as seen in Case 3.4

**Conclusion**

The unique capability of CMR to combine the evaluation of anatomy, haemodynamic and inflammatory status, provides superior diagnostic information. These inflammatory syndromes are not mutually exclusive, but rather a continuum in the phenotypes in a wide spectrum of
pericardial diseases, sometimes overlapping or transitioning from one phase to the other. Cardiac magnetic resonance is an effective tool to tailor individual therapy, particularly in cases of recurrent and constrictive pericarditis, given that the presence of ongoing inflammation has been found to be the best predictor of reversibility.

***Lead author biography***

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***Supplementary material***

Supplementary material is available at European Heart Journal—Case Reports online.

**Slide sets:** A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

**Consent:** The authors confirm that written consent for submission and publication of these case reports including images and associated text has been obtained from the patient in line with COPE guidance.

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**Table 1** Contribution of cardiac magnetic resonance in the classification of constrictive pericarditis in acute, subacute, or chronic

|          | Acute | Subacute | Chronic |
|----------|-------|----------|---------|
| T2       | +     | -        | -       |
| LGE      | +     | +        | -       |

LGE, late gadolinium enhancement.
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