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Management of pericarditis: recent advances

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Abstract

Pericarditis is the most frequent pericardial syndrome and a common cause of chest pain in emergency and cardiology practice. During the past two decades, management has moved from largely empirical anti-inflammatory treatment towards a mechanism-based, risk-stratified, and imaging-supported approach. Diagnosis remains clinical, based on typical chest pain, pericardial rub, electrocardiographic changes and pericardial effusion, but inflammatory biomarkers and multimodality imaging, especially cardiac magnetic resonance, increasingly support diagnostic confidence, differential diagnosis, and therapeutic decisions. For most patients with acute idiopathic or presumed viral pericarditis, aspirin or a non-steroidal anti-inflammatory drug combined with colchicine is first-line therapy, with exercise restriction and follow-up guided by symptoms and C-reactive protein. Corticosteroids should be avoided when possible or used at low-to-moderate doses only for selected indications, because they promote chronicity and recurrence in unselected cases. The major recent advance is the recognition of recurrent pericarditis as an autoinflammatory, interleukin (IL)-1-mediated disease in a

substantial proportion of patients with an inflammatory phenotype. Randomized and registry data now support IL-1 inhibition (eg, anakinra or rilonacept) for corticosteroid-dependent and colchicine-resistant recurrent pericarditis, allowing rapid symptom control, withdrawal of corticosteroids, and marked reduction in recurrences. Remaining challenges include optimal treatment duration, cost and access to biologics, management of patients without overt systemic inflammation, and personalized strategies for tapering and return to activity.

Key words

cardiac magnetic resonance, colchicine, interleukin-1, pericarditis, recurrent pericarditis

Introduction

Pericarditis is an inflammatory syndrome of the pericardial sac that may occur as an isolated disease or as part of a broader inflammatory myocardial and pericardial syndrome, that could be expression of a systemic disorder. Contemporary guidelines have emphasized a continuum between pericardial and myocardial inflammation, while still recognizing acute pericarditis, recurrent pericarditis, pericardial effusion, tamponade and constrictive pericarditis as clinically useful syndromes [1,2]. Most cases in high-income countries are idiopathic or presumed viral, whereas tuberculosis, purulent infection, malignancy, systemic autoimmune disease, renal failure and post-cardiac injury syndromes become more relevant in selected populations [2-5]. The practical challenge is not usually the initial recognition of a typical case, but the identification of patients who need hospitalization, etiological work-up, advanced imaging or second-line treatment. Another major challenge is recurrence. Recurrent pericarditis represents the most frequent clinically relevant complication after an acute episode. The risk of recurrence after a first episode is approximately 15–30% but may rise to about 50% after a first recurrence, particularly in patients not treated with colchicine. Several factors influence this risk.

Treatment-related factors include inadequate initial anti-inflammatory dosing, premature tapering or discontinuation of therapy before symptom resolution and C-reactive protein (CRP) normalization, and corticosteroid exposure, especially when corticosteroids are used at high doses or as first-line therapy without a specific indication [6-10]. Clinical factors associated with a more complicated course include fever, subacute presentation, large pericardial effusion, cardiac tamponade, and lack of response to aspirin or non-steroidal anti-inflammatory drugs after at least one week of therapy. Persistent or recurrent elevation of CRP identifies ongoing inflammatory activity and should prompt caution before tapering treatment.

Recurrent pericarditis can be disabling, with repeated emergency visits, hospitalizations, impaired sleep, anxiety, reduced work productivity and long-term dependence on corticosteroids or other therapies.

Recent advances can be grouped into five domains: improved triage and risk stratification; more rational use of biomarkers and multimodality imaging; consolidation of colchicine as standard treatment; a more cautious role for corticosteroids; and the emergence of targeted interleukin (IL)-1 inhibition for difficult recurrent disease.

Diagnosis and initial risk stratification

Acute pericarditis should be diagnosed within the broader framework of inflammatory myopericardial syndromes. According to the 2025 European Society of Cardiology (ESC) Guidelines, a definite diagnosis of pericarditis requires an appropriate clinical presentation together with more than one additional diagnostic criterion, whereas possible pericarditis is defined by a compatible clinical presentation with only one additional criterion. The diagnosis is considered unlikely / rejected when only the clinical presentation is present without additional supporting criteria (Table 1). Additional diagnostic criteria include pericardial rubs, typical electrocardiographic changes such as PR depression or widespread ST-segment elevation, CRP elevation [11], new or worsening pericardial effusion, and imaging evidence of

pericardial inflammation, including pericardial edema and / or late gadolinium enhancement on cardiovascular magnetic resonance. These criteria integrate the traditional clinical approach with biomarkers and multimodality imaging, allowing a more graded and clinically oriented diagnostic classification [1]. Troponin should be measured to detect concomitant myocardial involvement, which changes follow-up and exercise advice [1,2].

Initial evaluation should include history, examination, electrocardiography (ECG), echocardiography, chest radiography when appropriate, full blood count, renal function, liver tests, inflammatory markers and troponin. Echocardiography is central because it identifies effusion, tamponade physiology and associated structural heart disease. Most patients with a typical idiopathic presentation, no high-risk features and good response to treatment can be managed as outpatients with early follow-up [12,13].

According to the 2025 ESC Guidelines, patients with acute pericarditis should undergo early risk stratification to identify high-risk features that indicate a greater likelihood of non-viral etiology, complications, or need for in-hospital management. Hospital admission is recommended for patients with high-risk pericarditis for monitoring, etiological investigation, and treatment. In the acute setting, high-risk features include fever >38 °C, subacute onset, large pericardial effusion, cardiac tamponade, and failure to respond to aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) after at least 1 week of therapy. Additional relevant features include associated myocarditis, immunodepression, trauma, oral anticoagulant therapy, effusive–constrictive or incessant pericarditis, signs of right heart failure, and extensive pericardial late gadolinium enhancement on cardiovascular magnetic resonance (CMR; Table 2) [1].

Biomarkers and imaging: from diagnosis to treatment guidance

CRP has become more than a diagnostic adjunct. In acute pericarditis, high-sensitivity CRP is elevated in most patients and can be used to monitor disease activity and guide treatment

duration [11]. Quantitative data are available from observational cohorts. In a prospective study of 200 consecutive patients with viral or idiopathic acute pericarditis, high-sensitivity CRP was elevated at presentation in 156 patients (78%). Among patients with elevated values, normalization occurred in 60% by week 1, 85% by week 2, 95% by week 3, and 100% by week 4. Early assessment after symptom onset and previous anti-inflammatory treatment were the main recognized explanations for a normal value at presentation [11].

The expected magnitude of elevation is variable and depends on timing, phenotype, and associated inflammatory features. In a cohort of 225 patients with acute idiopathic pericarditis and available serial high-sensitivity CRP measurements, the initial value ranged from 0.1 to 34.0 mg/dL, with a median of 5.4 mg/dL, and the maximal value ranged from 0.1 to 39.0 mg/dL, with a median of approximately 9–10 mg/dL. Values were higher in patients with fever, pericardial effusion, pleural effusion, older age, and major cardiac complications; fever, pleural effusion and age were independent correlates of maximal CRP levels. The frequency of elevation also increased with time from symptom onset: elevated values were found in 76.0% of patients tested within 6 hours, 92.3% at 7–12 hours, 96.0% after 12 hours, and 98.1% after 24 hours [12]. Thus, a normal early CRP does not exclude pericarditis, particularly when blood sampling is performed very soon after symptom onset or after anti-inflammatory treatment has already been started.

A common practical mistake is to stop anti-inflammatory therapy because pain has improved while inflammation is still active. Treatment should usually be continued until both symptoms and CRP have normalized, followed by gradual tapering, especially in recurrent disease.

Cardiac magnetic resonance (CMR) has changed the management of difficult cases. Pericardial edema on T2-weighted or short tau inversion recovery sequences and late gadolinium enhancement identify active inflammation, whereas thickening, calcification and impaired ventricular filling may suggest chronic constriction [14–18]. CMR is particularly useful when

recurrent chest pain is atypical, inflammatory markers are equivocal, echocardiography is unrevealing, or escalation to corticosteroids, immunosuppression or IL-1 blockade is being considered. Serial CMR may also help guide tapering in selected patients treated with biologics, although cost, availability and standardization remain limitations [18].

Representative echocardiographic and cardiovascular magnetic resonance findings in acute and chronic pericarditis are shown in Figure 3, highlighting the complementary value of echocardiography for effusion and hemodynamic assessment and cardiovascular magnetic resonance for identifying active inflammation, pericardial enhancement, and chronic constrictive features.

Computed tomography is complementary. It is especially useful for calcification, complex or loculated effusions, suspected malignancy, trauma, pulmonary disease and pre-surgical planning. Echocardiography remains first-line for effusion and hemodynamics, while CMR is superior for tissue characterization and active inflammation.

First-line treatment of acute pericarditis

The first-line regimen for uncomplicated acute idiopathic or presumed viral pericarditis is aspirin or a NSAID plus colchicine, with gastroprotection [1]. Choice of anti-inflammatory drug should consider comorbidities: aspirin is preferred after myocardial infarction or when antiplatelet therapy is required; ibuprofen is widely used because of its safety profile and short half-life; indomethacin is effective but less well tolerated, especially in older patients. Full anti-inflammatory doses should be used initially, rather than analgesic doses, and then tapered after symptom resolution and CRP normalization (Tables 3 and 4).

Colchicine is one of the major evidence-based advances in pericarditis. In the COPE and ICAP trials, colchicine added to conventional anti-inflammatory therapy reduced incessant or recurrent pericarditis after a first acute episode [6,7]. A common adult regimen is 0.5 mg twice daily for patients weighing at least 70 kg and 0.5 mg once daily for those below 70 kg, usually

for three months after a first episode and at least six months in recurrent pericarditis. Loading doses are avoided to reduce gastrointestinal intolerance. Dose reduction or avoidance is required in severe renal or hepatic impairment, relevant drug interactions, frailty and pregnancy-specific scenarios.

Patients should avoid strenuous physical activity until symptoms, inflammatory markers, electrocardiographic changes and effusion have resolved. Athletes require more conservative restriction and careful reassessment before return to competition, particularly if myocardial involvement is suspected [1,2]. Education is important: patients should understand the expected time course, the need for adherence to colchicine, the danger of abrupt treatment withdrawal and the warning symptoms of tamponade or complicated disease.

Recurrent pericarditis: mechanisms and conventional treatment

Recurrent pericarditis is diagnosed after a documented first episode, a symptom-free interval of at least four to six weeks, and a subsequent episode meeting criteria for pericarditis or supported by biomarkers or imaging [1,2]. The disease may follow repeated viral or autoimmune triggers, but many idiopathic cases show features of autoinflammation: abrupt flares, fever or systemic symptoms, elevated CRP, serositis and response to IL-1 inhibition [19-24].

The first recurrence is usually treated with the same principles as acute pericarditis: full-dose aspirin or non-steroidal anti-inflammatory drug plus colchicine, with longer duration and slower tapering [8-10]. The CORE, CORP and CORP-2 trials established colchicine as effective for first and multiple recurrences [8-10]. Each drug should be tapered separately, starting with the anti-inflammatory agent, while maintaining colchicine, the last drug to withdraw [25]. Recurrences often follow rapid tapering at vulnerable dose thresholds; therefore, reductions should be small and guided by symptoms and CRP.

The 2025 ESC Guidelines recommend against corticosteroids as first-line therapy for a first episode of pericarditis in the absence of a specific indication (Class III). However, low- to

moderate-dose corticosteroids may be considered in selected cases (Class IIa), particularly when aspirin/NSAIDs and colchicine are contraindicated, not tolerated, or ineffective, or when there is a specific indication such as systemic inflammatory disease requiring corticosteroids, post-pericardiotomy syndrome, post-vaccine pericarditis, severe renal failure, or concomitant therapies interacting with NSAIDs, such as oral anticoagulation (Figure 1). When corticosteroids are used, prednisone 0.2–0.5 mg/kg/day, or an equivalent dose, is preferred, followed by slow tapering only after clinical remission and normalization of inflammatory markers; high-dose corticosteroids should be avoided because they may promote recurrence, corticosteroid dependence, and drug toxicity [1].

Azathioprine and intravenous immunoglobulin have been used as steroid-sparing options in refractory recurrent pericarditis, especially when there is an autoimmune phenotype or when biologics are unavailable [26-28]. Their evidence base is weaker than that for IL-1 inhibitors, and response may be slower. Pericardiectomy is now a last-resort option for highly selected patients with refractory recurrent pericarditis despite optimal medical therapy, or for chronic irreversible constriction, and should be performed in experienced centers, such practice is usually adopted in the US but not in Europe in the absence of concomitant constriction [29-31]. The new proposed algorithm for the treatment of acute and recurrent pericarditis according to the 2025 ESC guidelines [1] is illustrated in Figure 2.

Interleukin-1 inhibition: the main therapeutic advance

The clearest recent shift is the move from non-specific immunosuppression to targeted cytokine inhibition in corticosteroid-dependent and colchicine-resistant recurrent pericarditis. IL-1 is a central mediator of innate immunity and inflammasome activation [32]. This mechanism explains why some patients relapse repeatedly despite conventional therapy and why corticosteroids may suppress symptoms without resetting the inflammatory process.

Anakinra is a recombinant IL-1 receptor antagonist that blocks IL-1α and IL-1β. In the AIRTRIP randomized trial, anakinra markedly reduced recurrences in patients with colchicine-resistant, corticosteroid-dependent recurrent pericarditis [19]. The International Registry of Anakinra for Pericarditis (IRAP) registry subsequently supported its effectiveness and safety in real-world practice, showing reductions in recurrences, emergency department admissions, hospitalizations and corticosteroid use [20]. Typical treatment is daily subcutaneous administration, with local injection-site reactions being the most frequent adverse event. Tapering should be slow because abrupt discontinuation can lead to relapse.

Rilonacept is a weekly IL-1α and IL-1β cytokine trap. In the RHAPSODY phase 3 trial, rilonacept produced rapid symptom and CRP improvement and substantially reduced recurrence risk compared with placebo [21]. Long-term extension data suggest sustained benefit during continued treatment, although recurrence after discontinuation remains an important issue [22]. Patient-reported outcomes from RHAPSODY also showed improvements in pain, quality of life and sleep [23]. Rilonacept's weekly dosing is convenient, and it has regulatory approval for recurrent pericarditis in the US; access and cost remain major determinants of use.

A major limitation of anti-IL-1 agents is that treatment discontinuation can be challenging, as some patients appear to remain clinically quiescent only while therapy is continued. The mechanisms underlying this treatment dependence are not fully understood. It remains uncertain whether, in these cases, IL-1 blockade truly modifies the underlying disease process or primarily suppresses inflammatory activity and symptoms, with recurrence emerging once pharmacological control is withdrawn.

IL-1 inhibition is most compelling in patients with an inflammatory phenotype: recurrent attacks with elevated CRP, objective imaging inflammation, corticosteroid dependence, colchicine resistance or intolerance, and repeated hospitalization [25]. Before starting biologics, infection, tuberculosis and malignant disease should be excluded according to risk. Vaccination

status, lipid profile, blood counts and liver tests should be reviewed [32]. Shared decision-making is essential because treatment may be prolonged and the best discontinuation strategy is still uncertain.

Special clinical settings

Post-cardiac injury syndromes, including post-pericardiotomy syndrome and post-myocardial infarction pericarditis, are immune-mediated inflammatory syndromes following injury to the pericardium or myocardium. Colchicine has shown benefit in preventing post-pericardiotomy syndrome in COPPS and COPPS-2, although perioperative tolerability, gastrointestinal adverse effects and patient selection are important [33,34]. Treatment of established post-cardiac injury pericarditis generally follows the same anti-inflammatory principles as idiopathic disease, with attention to bleeding risk, renal function and concomitant antithrombotic therapy.

Myopericarditis requires caution. Mild troponin elevation with preserved ventricular function usually has a good prognosis, but exercise restriction should be longer and follow myocarditis recommendations. CMR is valuable to define the extent of myocardial involvement, detect oedema or fibrosis, and guide follow-up [1,17,18].

Anticancer therapy should also be considered in the differential diagnosis of pericarditis. Immune checkpoint inhibitors may cause immune-related pericardial disease, either as isolated pericarditis/pericardial effusion or in association with myocarditis or myopericarditis. Although uncommon, immune checkpoint inhibitor (ICI)-associated pericarditis is increasingly recognized and may be clinically significant, with reported presentations ranging from mild pericarditis to large effusion and cardiac tamponade. Diagnosis is challenging in patients with malignancy because malignant pericardial involvement, infection, radiation-related disease may mimic immune-mediated pericarditis. Therefore, new pericarditis or pericardial effusion during ICI therapy should prompt cardio-oncology assessment, evaluation for concomitant myocardial involvement with troponin, ECG and imaging, and individualized management

including anti-inflammatory therapy, corticosteroids when an immune-related adverse event is suspected, pericardial drainage when clinically indicated, and temporary or permanent ICI interruption according to severity and oncological context [35,36].

Systemic autoimmune and autoinflammatory diseases are important causes of pericarditis and should be considered particularly in patients with recurrent disease, systemic symptoms, extra-cardiac manifestations, or abnormal immunological tests. Systemic lupus erythematosus is the most representative systemic autoimmune disease associated with pericarditis. Recent data from the University of Toronto Lupus Clinic showed that pericarditis affects approximately one in five patients with systemic lupus erythematosus, usually early in the disease course; most cases resolved within three months, although chronic or relapsing disease occurred in a clinically relevant minority [37]. Other systemic diseases associated with pericardial involvement include rheumatoid arthritis, Sjögren syndrome, systemic sclerosis, mixed connective tissue disease, vasculitis, Behçet disease, familial Mediterranean fever, and Still disease. Still disease is particularly relevant because it may present with systemic inflammation, serositis, pericarditis, and, in severe cases, associated myocardial involvement. Recent case-based evidence has also reported successful use of IL-1 blockade with anakinra in severe relapsed Still disease complicated by perimyocarditis, supporting the concept that selected systemic inflammatory phenotypes may benefit from targeted cytokine inhibition in addition to disease-specific rheumatological management [38].

Pregnancy requires individualized management. Aspirin and non-steroidal anti-inflammatory drugs may be used in selected phases but should generally be avoided late in pregnancy; colchicine is increasingly considered acceptable when clinically indicated, based on broader safety experience in Familial Mediterranean Fever, but decisions should involve obstetric and cardiology expertise. Corticosteroids may be used when necessary, at the lowest effective dose. IL-1 inhibitors in pregnancy require specialist discussion because data remain limited [39].

Practical treatment algorithm

A practical approach begins with confirming pericarditis and excluding red flags. Low-risk acute pericarditis can be treated as an outpatient with aspirin or a non-steroidal anti-inflammatory drug, colchicine, gastroprotection and reassessment within one week. Therapy continues until symptoms and CRP normalize, then the anti-inflammatory drug is tapered. Colchicine is continued for the planned course.

For the first recurrence, clinicians should confirm objective inflammation, check adherence and dosing, look for triggers or specific etiologies, restart full-dose anti-inflammatory therapy and prescribe colchicine for at least six months. For multiple recurrences, corticosteroid dependence or failure of colchicine, the key step is phenotyping. If there is active inflammation, IL-1 inhibition should be considered early to avoid prolonged corticosteroid exposure [25]. If inflammation is absent, alternative diagnoses such as musculoskeletal pain, neuropathic pain, coronary disease, gastro-esophageal disease or anxiety-related pain should be reconsidered before escalating immunomodulation.

Tapering is often where management succeeds or fails. Patients should be asymptomatic, CRP should be normal, and, in difficult cases, imaging should show improvement before tapering. Only one treatment should be reduced at a time. Corticosteroids, when present, should be tapered first and most slowly. Biologic tapering remains less standardized; options include dose-spacing or stepwise withdrawal after a prolonged remission, supported by biomarkers and, in selected cases, CMR.

Future directions

The future of pericarditis management is likely to be personalized. Important unanswered questions include how to identify patients who should receive IL-1 inhibitors earlier, how long biologic therapy should continue, whether CMR can safely guide withdrawal, and how to manage patients with recurrent pain but minimal measurable inflammation. Biomarkers beyond

CRP, genetic susceptibility to autoinflammatory disease, and better patient-reported outcome measures may refine treatment selection.

Another priority is implementation. Many patients still receive inadequate colchicine duration, excessive corticosteroids, or delayed referral after multiple relapses. Wider education, multidisciplinary pericardial disease clinics, standardized tapering protocols and access to imaging and biologics could improve outcomes. The recent guideline emphasis on inflammatory myocardial and pericardial syndromes should also encourage clinicians to assess myocardial involvement systematically and tailor return-to-exercise advice [1].

Conclusions

Management of pericarditis has advanced substantially. The foundation remains accurate diagnosis, risk stratification, aspirin or non-steroidal anti-inflammatory drugs, colchicine and careful follow-up with CRP. CMR has become a key tool for difficult or recurrent cases, allowing objective assessment of pericardial inflammation. Corticosteroids should no longer be viewed as routine second-line treatment; rather, they should be reserved for selected indications and used at the lowest effective dose with slow tapering. For patients with corticosteroid-dependent and colchicine-resistant recurrent pericarditis, IL-1 inhibition has transformed care, offering rapid control of inflammation and major reductions in recurrence. The next step is to integrate these advances into practical, affordable and personalized pathways.

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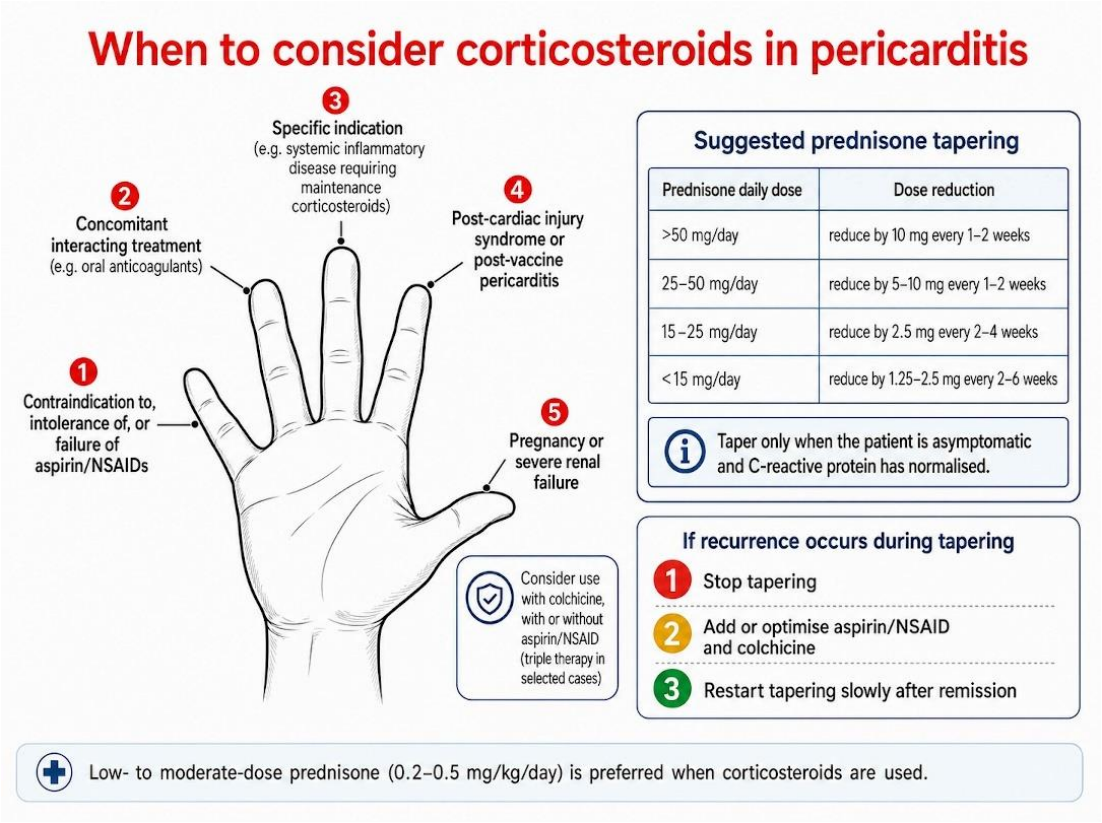

Figure 1 Selected indication for corticosteroid in patients with pericarditis (see text for explanation). Abbreviations: see Table 2

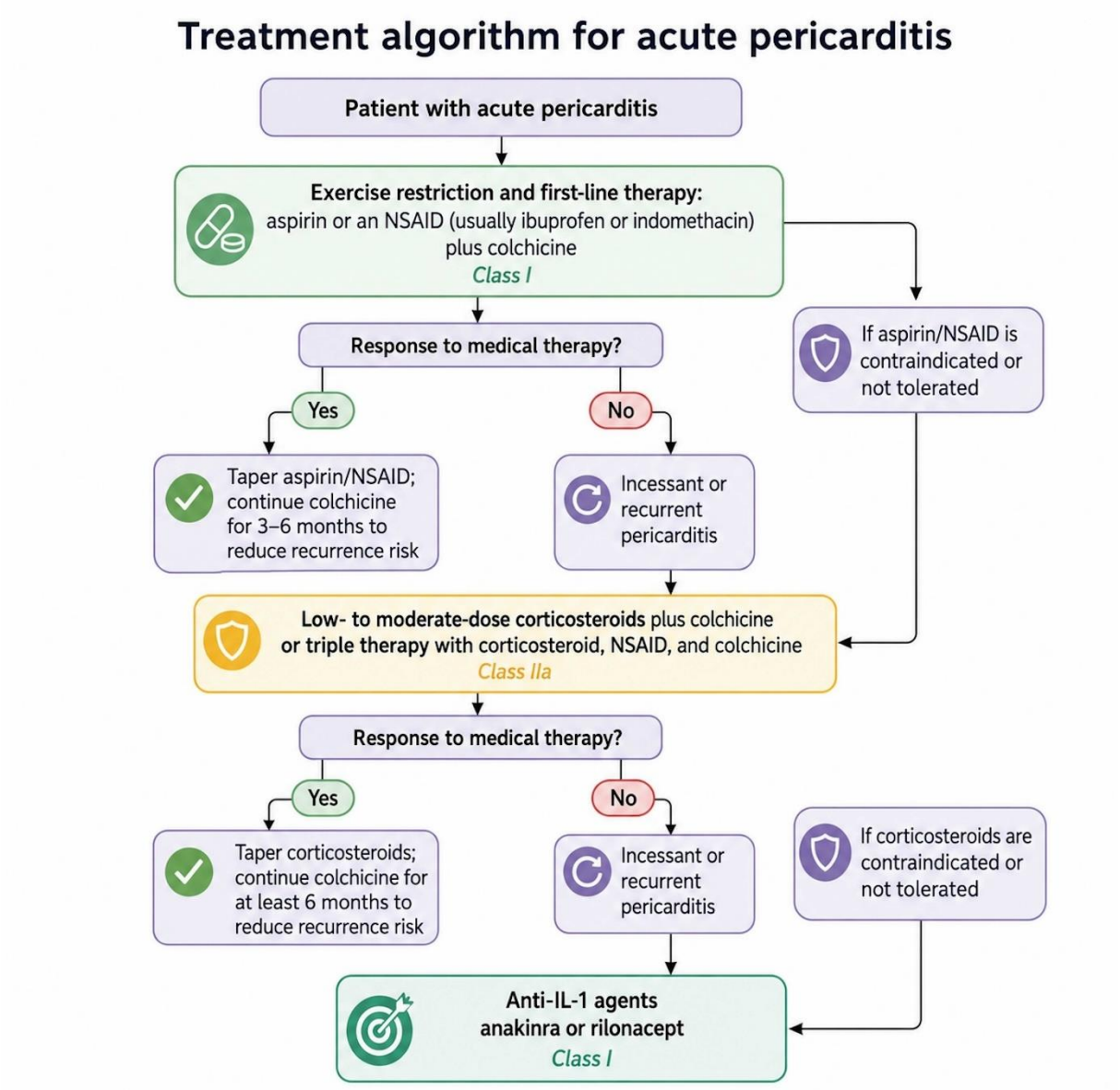

Figure 2 New therapeutic algorithm for pericarditis according to the 2025 European Society of Cardiology guidelines [1]. Abbreviations: see Table 2

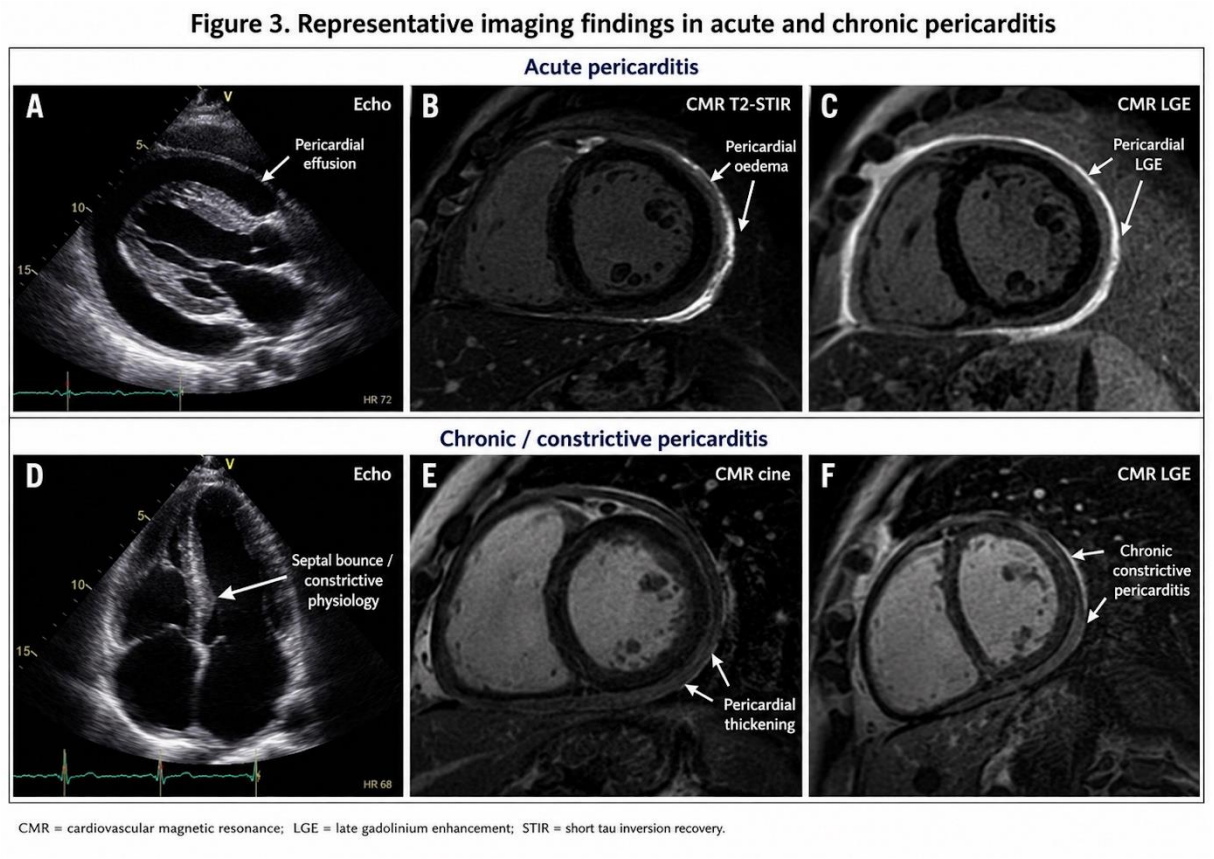

Figure 3 Representative imaging findings in acute and chronic pericarditis. Echocardiography may demonstrate pericardial effusion and hemodynamic features suggesting tamponade or constrictive physiology, whereas cardiovascular magnetic resonance allows tissue characterization, including pericardial oedema on T2-weighted or short tau inversion recovery sequences and pericardial late gadolinium enhancement as markers of active inflammation. In chronic disease, imaging may show pericardial thickening, calcification, septal bounce, ventricular interdependence, and other features of constrictive pericarditis.

Table 1 New diagnostic categories and diagnostic criteria for pericarditis according to the 2025 European Society of Cardiology guidelines		
Diagnostic category	**ESC 2025 definition**	**Practical interpretation**
Definite pericarditis	Compatible clinical presentation plus >1 additional criterion	Diagnosis can be established clinically when symptoms are supported by at least two objective findings
Possible pericarditis	Compatible clinical presentation plus 1 additional criterion	Diagnosis is plausible but less secure; further assessment, follow-up, biomarkers or imaging may be needed
Unlikely / rejected pericarditis	Compatible clinical presentation without additional criteria	Pericarditis should not be retained unless new objective evidence emerges
Abbreviations: ESC, European Society of Cardiology		

Table 2 High-risk features supporting hospital admission in acute pericarditis according to the 2025 ESC Guidelines [1]		
Source / category	**High-risk feature**	**Comment**
Major features	Fever >38 °C, subacute onset	Validated prognostic predictors
	Large pericardial effusion	>20 mm on echocardiography
	Cardiac tamponade	Requires urgent assessment and often drainage
	Lack of response to aspirin or NSAID	After at least 1 week of therapy
Minor features	Pericarditis associated with myocarditis	Suggests myopericardial involvement
	Immunodepression	Raises concern for specific infectious or neoplastic causes
	Trauma	Including iatrogenic or chest trauma-related disease
	Oral anticoagulant therapy	Concern for haemorrhagic effusion/complications
New	Effusive–constrictive pericarditis	Listed as high risk in clinical risk stratification
	Incessant pericarditis	Listed as high risk in clinical risk stratification
	Signs and symptoms of right heart failure	Listed as high risk in clinical risk stratification

Table 2 High-risk features supporting hospital admission in acute pericarditis according to the 2025 ESC Guidelines [1]

Source / category	**High-risk feature**	**Comment**
	Extensive pericardial LGE on CMR	Listed as high-risk imaging criterion

Abbreviations: CMR, cardiovascular magnetic resonance; LGE, late gadolinium enhancement; NSAID, nonsteroidal anti-inflammatory drug

Table 3 Doses and tapering of commonly prescribed anti-inflammatory and immunomodulatory drugs for pericarditis

Drug	**Initial dose**	**Usual duration at full dose**	**Tapering**
Aspirin	750–1000 mg three times daily	1–2 w	Decrease by 250 mg every 1–2 w
Ibuprofen	600–800 mg three times daily	1–2 w	Decrease by 200 mg every 1–2 w
Indomethacin	25–50 mg three times daily	1–2 w	Decrease by 25 mg every 1–2 w
Colchicine	0.5 mg once daily if <70 kg or severe renal impairment; otherwise, 0.5 mg twice daily	3–6 mo	Not usually required; withdraw as the last drug after stable remission
Prednisone	0.2–0.5 mg/kg/d	2–4 w	Taper slowly over several mo
Azathioprine	Start at 1 mg/kg/d, then increase gradually to 2–3 mg/kg/day	Several mo	Taper over several mo
IVIG	400–500 mg/kg i.v. daily for 5 days	5 d	Not required
Anakinra	1–2 mg/kg/day, up to 100 mg/d in adults	At least 6 mo; often >12 mo	Required; at least 3–6 mo
Rilonacept	320 mg once, followed by 160 mg weekly	Not clearly established in ESC table	Not clearly established

Table 4 Corticosteroid tapering schedule in 2025 ESC guidelines	
Prednisone daily dose	**Suggested tapering**
>50 mg	Reduce by 10 mg/d every 1–2 w
50–25 mg	Reduce by 5–10 mg/d every 1–2 w
25–15 mg	Reduce by 2.5 mg/d every 2–4 w
<15 mg	Reduce by 1.25–2.5 mg/d every 2–6 w

Every prednisone reduction should be made only when the patient is asymptomatic and CRP is normal, particularly below 25 mg/day. Higher corticosteroid doses should generally be avoided, except in special cases and only for a few days, with rapid tapering to 25 mg/day.

Short title: Management of pericarditis: recent advances