Trends in Cardiovascular Medicine 36 (2026) 317–326

Contents lists available at ScienceDirect

Trends in Cardiovascular Medicine

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

Myocarditis and pericarditis in focus: A critical appraisal of the 2025 ESC vs ACC position statements from the Italian society of cardiology working group on cardiomyopathies and pericardial diseases

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ARTICLE INFO

Keywords:
Myocarditis
Pericarditis
Guidelines
Consensus document

ABSTRACT

The 2025 European Society of Cardiology (ESC) guidelines and the 2024–2025 American College of Cardiology (ACC) consensus documents redefine the management of myocarditis and pericarditis, with notable convergence, yet key differences. Both emphasize early, accurate diagnosis, particularly through cardiac magnetic resonance (CMR), which now often supersedes immediate biopsy in stable, uncomplicated cases of acute myocarditis. The ESC introduces a unified "inflammatory myopericardial syndrome" (IMPS) framework encompassing myocarditis, pericarditis, and overlap syndromes, while the ACC provides separate pathways, including a novel four-stage clinical classification of myocarditis. Therapeutically, both endorse non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine for pericarditis and myopericarditis, and heart failure–directed therapy for myocarditis, while reserving immunosuppression for select cases. Importantly, interleukin-1 (IL-1) blockade has emerged as a pivotal therapy in recurrent pericarditis, receiving a Class I recommendation in ESC guidelines and strong endorsement in ACC guidance. Prognostic assessment focuses on identifying high-risk features and structured follow-up with imaging and biomarkers. Divergences in terminology, staging, and diagnostic thresholds underscore opportunities for further harmonization. The ESC and ACC documents align in a patient-tailored, evidence-informed approach to management.

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https://doi.org/10.1016/j.tcm.2026.01.002

Introduction

Myocarditis and pericarditis are inflammatory cardiac syndromes whose management has changed substantially with new evidence. Until recently, clinicians relied on older ESC documents (the 2013 position statement on myocarditis and the 2015 pericardial disease guidelines) [1,2].

In 2024–2025, the European Society of Cardiology (ESC) and American College of Cardiology (ACC) issued updated guidance, marking a "renaissance" in this field. The ESC 2025 Guidelines provide a unified framework for both conditions [3], while the ACC released separate documents: a 2024 Expert Consensus Decision Pathway on myocarditis[4] and a 2025 concise clinical guidance on pericarditis [5], supported by a previous 2024 multimodality imaging position paper [6]. These incorporate advances in diagnostic imaging (especially CMR), immunologic mechanisms (including autoinflammatory pathways), and novel treatments such as IL-1 inhibitors.

This review compares ESC and ACC recommendations across diagnostic definitions, clinical classification, imaging strategies, therapy, prognosis, and follow-up. It highlights areas of convergence and divergence, the main innovations in each document, and their practical application, to help clinicians integrate the strengths of both approaches in managing myocarditis and pericarditis.

Diagnostic definitions and criteria

Diagnostic criteria for myocarditis and pericarditis have been revised and updated.

Myocarditis – ESC vs ACC

Both ESC and ACC emphasize diagnosing myocarditis by combining clinical presentation with objective tests, shifting away from routine endomyocardial biopsy (EMB). The ESC 2025 guidelines classify myocarditis as Definite, Possible, or Unlikely:

- *Definite*: clinical syndrome plus CMR or EMB confirmation.
- *Possible*: clinical syndrome plus ≥1 supportive finding (ECG changes, troponin rise, or abnormal echo) when CMR or EMB are uncertain or not available.
- *Unlikely*: symptoms without objective findings.

The ACC 2024 document does not use the exact labels but follows the same principle: a compatible clinical syndrome plus noninvasive evidence (especially CMR) is sufficient for diagnosis in uncomplicated patients. Both guidelines prioritize CMR—according to revised Lake Louise criteria—[7] as the main diagnostic tool, with EMB reserved for intermediate/high-risk cases. They also agree that routine viral serology is not recommended unless results would alter management. A notable ACC addition is a four-stage myocarditis framework (A–D) ranging from at-risk to advanced disease.

Pericarditis – ESC vs ACC

Both sets of guidelines modernize acute pericarditis criteria to incorporate biomarkers and imaging. Traditionally, ≥2 of 4 classic features were required [2]. The ACC 2025 makes pleuritic chest pain mandatory, classifying cases as:

- *Definite*: chest pain + ≥2 objective features (rub, ECG changes, CRP/ESR elevation, effusion, or CMR inflammation).
- *Possible*: chest pain + 1 feature.
- *Unlikely/rejected*: only chest pain.

The ESC 2025 uses a nearly identical framework but allows chest-pain–negative presentations (e.g., dyspnea with strong objective evidence), that are usually encountered in the elderly [8,9]. Both highlight that most patients present with pleuritic pain and that 10–20 % may have a "non-inflammatory" phenotype with normal CRP. Both 2025 ESC guidelines[3] and ACC paper[5] elevate CMR and CRP to equal importance with classic signs, representing an evolution from 2015 criteria [2].

Overlap syndromes (Myopericardial involvement)

Both ESC and ACC recognize a continuum of combined myocardial and pericardial inflammation. ESC introduces the umbrella term inflammatory myopericardial syndrome (IMPS) and retains the traditional labels myopericarditis (pericarditis-dominant) and perimyocarditis (myocarditis-dominant) [3]. ACC paper uses similar terminology [4]. About 15–30 % of pericarditis patients show myocardial involvement (elevated troponin or wall-motion abnormalities).

Both guidelines recommend managing overlap cases using principles of *both* conditions—treating pericardial inflammation while monitoring myocardial function and arrhythmias. Significant myocardial involvement (e.g., troponin elevation plus reduced EF) should be managed primarily as myocarditis with correspondingly intensive follow-up.

Clinical classification and disease staging

Time-Course classification – acute, subacute, chronic

ESC and ACC largely agree on time-based categories for myocarditis and pericarditis. ESC defines acute myocarditis as ≤4 weeks, subacute as >4 weeks to ≤3 months, and chronic as >3 months [3]. Within acute disease, ESC identifies:

- Fulminant myocarditis: sudden onset with hemodynamic instability requiring inotropes or mechanical circulatory support (MCS).
- Complicated myocarditis: acute disease with LVEF <50 %, sustained arrhythmias, high-grade AV block, heart failure, or shock.

For pericarditis, ESC uses similar cutoffs: acute ≤4 weeks, subacute/incessant >4 weeks to ≤3 months, chronic >3 months [3]. ACC definitions are nearly identical, using "acute," "incessant," "recurrent," and "chronic," with only minor wording differences [5]. Both note that incessant pericarditis often indicates a more aggressive course than resolving acute disease.

ACC's four-stage myocarditis classification

A key difference is the ACC's four-stage system (A–D) for myocarditis [4]:

- Stage A: at-risk individuals (e.g., viral exposure, immune checkpoint inhibitors, genetic predisposition).
- Stage B: asymptomatic patients with objective injury or inflammation (e.g., troponin rise, CMR findings).
- Stage C: symptomatic active myocarditis.
- Stage D: severe disease such as fulminant myocarditis or end-stage inflammatory cardiomyopathy.

ESC does not use this staging, focusing instead on acute/subacute/chronic phases plus severity descriptors (fulminant, complicated) [3]. However, ESC acknowledges subclinical myocarditis and recommends close monitoring in high-risk groups, conceptually similar to ACC Stages A and B. Overall, both classify disease by duration and severity, but ACC adds a risk-based layer not present in ESC terminology.

Table 1
Myocarditis: comparative diagnostic criteria and classification.

Feature	2025 ESC Guidelines	2024 ACC Consensus
Diagnostic definition	Clinical syndrome plus objective evidence required. 1. Definite: typical presentation + CMR or EMB confirmation. 2. Possible: presentation + ≥1 supportive finding* 3. Unlikely if only symptoms without findings.	Emphasizes clinically suspected myocarditis confirmed by noninvasive tests. CMR and biomarkers allow diagnosis without biopsy in many cases. EMB not required unless needed for etiology. No formal "definite/possible" labels.
Clinical classification	Time-based: 1. Acute ≤4 weeks (includes Fulminant); 2. Subacute 1–3 months; 3. Chronic >3 months. By severity:"Complicated" if LVEF <50 %, arrhythmias, heart block, HF, or shock present .	Time-based definitions align (acute <1 month, chronic >3 months).Additionally introduces 4 Stages (A–D): 1. Stage A – at-risk (exposures, genetic predisposition); 2. Stage B – subclinical (inflammation/injury without symptoms); 3. Stage C – active symptomatic myocarditis; 4. Stage D – advanced (fulminant or end-stage). Provides a framework for risk stratification beyond time alone.
Overlap	ESC criteria classify such a patient according to dominant syndrome (myocarditis vs pericarditis) and manage accordingly: 1. "Myopericarditis" (pericardial involvement predominant). 2. "Perimyocarditis" (myocardial involvement predominant).	"Peri-myocarditis" in ACC wording (same meaning). ACC notes ~15 % of pericarditis patients have myocardial involvement (troponin leak or wall motion abnormality). These are managed with a hybrid approach: standard pericarditis therapy plus vigilance for ventricular dysfunction/arrhythmia as in myocarditis . If myocardial injury is substantial (e.g. LV dysfunction), patients should be treated as myocarditis.

*= ECG changes, troponin rise, echo abnormality; both ACC and ESC stress that concomitant myocarditis is a risk factor for worse outcome in pericarditis (warrants hospitalization and specialty care).

Table 2
Myocarditis: Comparative use of Cardiac Magnetic Resonance (CMR) and endomyocardial biopsy (EMB).

Diagnostic test	2025 ESC Guidelines	2024 ACC Consensus
CMR	CMR considered a noninvasive diagnostic gold standard. Adopts updated Lake Louise Criteria (LLC): CMR evidence of non-ischemic inflammation requires ≥1 T2-based sign (edema) plus ideally ≥1 T1-based sign (LGE or mapping abnormality). Having both T2 and T1 criteria increases specificity; if only one is positive in a fitting clinical scenario, myocarditis can still be diagnosed (with lesser certainty).	Strongly emphasizes CMR as a pivotal diagnostic tool: if patient is stable, CMR (with T1/T2 mapping) should be obtained to confirm inflammation. Recognizes that CMR can "noninvasively" diagnose myocarditis previously confirmed only by biopsy. If CMR is contraindicated or nondiagnostic, other imaging (e.g. FDG-PET) may provide incremental information.
EMB	Still considered the reference standard for etiologic diagnosis. ESC advises selective use of EMB: indicated when results would change management (e.g., suspected giant cell myocarditis, unclear diagnosis, or failure to improve). A "paradigm shift" is noted: routine biopsy is not required in all cases now that CMR can confirm inflammation in uncomplicated cases.	Provides detailed guidance on when to perform EMB. EMB is recommended if the diagnostic/prognostic benefit outweighs risk – particularly if specific diagnoses requiring targeted therapy are suspected (giant cell, eosinophilic, sarcoidosis, etc.). A decision algorithm stratifies which suspected myocarditis patients need early EMB . ACC notes biopsy is under-utilized but can be lifesaving (e.g., early EMB in fulminant cases improves 1-year transplant-free survival).

Recurrent disease

Both societies define recurrent pericarditis as a new episode after a symptom-free interval, with recurrence rates of ~20–30 % after the first episode and up to ~50 % after the second [3,5]. Recurrent myocarditis is less common (up to ~10 %), but recognized by ESC as new symptoms or inflammatory activity after remission. ESC places special emphasis on genetic testing in recurrent myocarditis/pericarditis, or when features suggest an underlying cardiomyopathy (e.g., arrhythmias, extensive LGE) [3].

Both guidelines note that persistent or recurrent inflammation may evolve into inflammatory cardiomyopathy, characterized by chronic myocardial inflammation with ventricular dysfunction and remodeling. ESC recommends considering EMB in such cases to identify treatable causes [3]. ACC similarly acknowledges that some myocarditis patients progress to dilated cardiomyopathy and require long-term heart failure therapy [4].

Tables 1–4 provide comparative summaries of key diagnostic criteria, classification schemes, and imaging recommendations in the ESC and ACC documents, highlighting their concordance as well as terminology differences.

Imaging recommendations and the role of multimodality imaging

Modern imaging is central to both ESC guidelines and ACC papers guidance for myocarditis and pericarditis [3–7].

Transthoracic echocardiography (TTE)

TTE remains the first-line test due to its availability and ability to identify pericardial effusion, ventricular dysfunction, and regional wall-motion abnormalities. ESC guidelines and ACC papers both emphasize its role in the initial evaluation. In myocarditis, TTE may be normal, if inflammation is mild or focal, but reduced systolic function or non-coronary wall-motion abnormalities raise suspicion. In pericarditis, TTE helps confirm effusion and detect tamponade or constriction. Both guidelines stress that a normal TTE

Table 3
Pericarditis: comparative diagnostic criteria and classification.

Feature	2025 ESC Guidelines	2025 ACC Consensus
Diagnostic criteria	Definite: typical presentation (usually pleuritic chest pain) with >1 supportive criterion (pericardial rub, ECG changes, elevated CRP, new/worsening effusion, or CMR signs) . Possible: chest pain + 1 criterion; Unlikely: chest pain alone without objective findings. This effectively requires ≥2 traditional criteria for a confident diagnosis, similar to classic teaching. Allows that clinical presentation could rarely be atypical (e.g., primarily dyspnea) if other criteria strongly positive.	Novel criteria: Mandatory – pleuritic chest pain or a characteristic clinical presentation. Plus ≥1 of: pericardial friction rub; diffuse ST-elevation and/or PR-depression on ECG; elevated inflammatory markers (CRP/ESR); pericardial effusion (new or increasing); or CMR evidence of pericardial inflammation (LGE and/or edema). Definite if chest pain + ≥2 findings; Possible if chest pain + 1 finding; Unlikely if no objective findings. Emphasizes chest pain as a required symptom (recognizing that truly "painless" acute pericarditis is rare).
By duration	Acute pericarditis: <4–6 weeks. Subacute/Incessant: 4–6 weeks up to 3 months (persistent symptoms without remission). Chronic: >3 months. Recurrent: new episode after symptom-free interval ≥4–6 weeks. These definitions match prior ESC (2015) usage. Incessant: pericarditis with failure to achieve remission by ∼6 weeks or relapse during tapering of therapy.	Acute: <4–6 weeks; Incessant: >6 weeks, <3 months (no remission); Recurrent: recurrence after ≥4–6 weeks without symptoms; Chronic: >3 months. ACC explicitly notes that incessant pericarditis can behave more aggressively. ACC also highlights classification by inflammatory phenotype: each episode (acute or recurrent) can be inflammatory (high CRP, fever, etc.) or non-inflammatory (normal CRP, often autoimmune-associated), which influences therapy choices.
High-risk features	Identifies "red flags" that portend poor prognosis or need hospitalization, largely adopted from 2015 criteria . These include: high fever, subacute onset, large effusion, cardiac tamponade physiology, and lack of response to NSAIDs, as well as elevated troponin (myocardial involvement) . Presence of any warrants more aggressive evaluation (hospital admission, invasive procedures).	Lists same risk factors for poor outcome / admission: temp >38 °C, insidious onset, large effusion or tamponade, failure of NSAID therapy, and concomitant myocarditis . ACC also notes that a non-response to NSAIDs and a high CRP or severe pericardial LGE on CMR signal a more refractory course . These patients often require escalation to second-line therapy (e.g. IL-1 blockers or immunosuppressives) and should be managed in specialized centers. Both guidelines are concordant in recommending hospitalization for any pericarditis with hemodynamic compromise or high-risk features .

Table 4
Pericarditis: high risk features and multimodality imaging.

Feature	2025 ESC Guidelines	2025 ACC Consensus
High-risk features	Identifies "red flags" that portend poor prognosis or need hospitalization, largely adopted from 2015 criteria. These include: high fever, subacute onset, large effusion, cardiac tamponade physiology, and lack of response to NSAIDs, as well as elevated troponin (myocardial involvement). Presence of any warrants more aggressive evaluation (hospital admission, invasive procedures).	Lists same risk factors for poor outcome /admission: temp >38 °C, insidious onset, large effusion or tamponade, failure of NSAID therapy, and concomitant myocarditis. ACC also notes that a non-response to NSAIDs and a high CRP or severe pericardial LGE on CMR signal a more severe course. These patients often require escalation to second-line therapy (e.g. IL-1 blockers or immunosuppressives) and should be managed in specialized centers.
Multimodality Imaging	Echocardiography: first-line in all pericarditis to detect pericardial effusion, assess hemodynamics, and screen for myocardial involvement. CMR: recommended in cases that are diagnostically unclear, or in severe/recurrent cases to confirm inflammation and guide therapy. CT: considered for suspected pericardial calcification or when CMR is contraindicated, and in effusive cases to characterize fluid (e.g. tuberculous vs transudate).	Echocardiography: indispensable initial test for all suspected pericarditis . ACC notes echo can be normal in many cases, but if effusion is present it should be quantified and followed serially. Echo also evaluates constrictive physiology. CMR: advocated as a second-line imaging modality for diagnosis, risk stratification, and follow-up of pericarditis (especially for complicated cases, incessant or recurrent pericarditis, or prior to therapy escalation).* CT: highlighted for its ability to detect pericardial calcifications and delineate pericardial thickness when CMR is unavailable. CT with contrast can also show pericardial enhancement (inflammation).
Overlap	ESC criteria classify such a patient according to dominant syndrome (myocarditis vs pericarditis) and manage accordingly: 1. "Myopericarditis" (pericardial involvement predominant). 2. "Perimyocarditis" (myocardial involvement predominant).	Myo-pericarditis: similar definition. ACC notes these patients should be monitored for arrhythmias and ventricular function. Troponin-positive pericarditis is essentially treated as pericarditis plus beta-blockers and ACE-I if any LV dysfunction.

*= CMR findings (pericardial LGE and edema) confirm active inflammation and can be semi-quantified; ACC even provides grading criteria for pericardial LGE (none, mild, severe). Overall, ACC strongly promotes a multimodality imaging approach (echo + CMR ± CT) to fully characterize pericardial disease.
Both ACC and ESC stress that concurrent myocardial involvement ups the ante on monitoring and potentially justifies cardiac MRI in all such cases to quantify myocardial inflammation. The presence of even mild myocarditis (peri–myocarditis) is a poor prognostic marker in pericarditis, mandating closer follow-up.

does not exclude myocarditis or pericarditis—if suspicion persists, further imaging (usually CMR) is required.

Cardiac magnetic resonance (CMR)

CMR is considered the non-invasive gold standard for myocarditis. The Lake Louise Criteria (2018) rely on detecting edema (T2) and fibrosis or inflammation (LGE/T1) [7]. Having both criteria greatly increases specificity. ESC and ACC agree that CMR has transformed diagnosis and reduced the need for biopsy [4,5]. Both integrates CMR into its diagnostic algorithm once ischemia is excluded. In pericarditis, CMR is recommended mainly for complicated, recurrent, or chronic cases, helping visualize active inflammation through pericardial LGE or edema. ESC also gives CMR a

Class I recommendation for follow-up in myocarditis to document healing [5]. CMR findings offer prognostic value—myocardial LGE predicts arrhythmias, and pericardial LGE predicts recurrence.

Computed tomography (CT)

CT is useful when CMR is not feasible or useful, especially to show pericardial thickening, calcification, or enhancement, and to help evaluate constriction or large effusions. CT attenuation can differentiate exudates from transudates. CT is not routine for myocarditis, but cardiac CT performed for chest pain may be helpful to rule out coronary artery diseases and additional abnormalities.

Nuclear imaging

Nuclear imaging is optional and used selectively. FDG-PET may help when CMR is not possible, especially for suspected cardiac sarcoidosis. ESC notes PET may assist in complex diagnostic cases (e.g. cardiac sarcoidosis) but is not part of standard evaluation.

In summary, both ESC and ACC strongly support an imaging-first strategy: start with TTE, then use CMR for most suspected myocarditis and selected pericarditis cases [4,5]. Early imaging—ideally within 1–2 weeks—improves diagnostic yield. Their aligned recommendations highlight major advances in imaging that now allow precise diagnosis, risk stratification, and follow-up of myocardial and pericardial inflammation.

Therapeutic strategies: pharmacological and interventional approaches

Supportive and anti-inflammatory therapy in myocarditis

For acute myocarditis without fulminant features, both ESC and ACC stress supportive management and standard heart failure/arrhythmia therapy [3,4]. About half of patients recover fully, while others may develop persistent dysfunction or progress to dilated cardiomyopathy [3,10–12]. Initial treatment focuses on hemodynamic optimization with ACE inhibitors/ARBs, beta-blockers (when appropriate), and diuretics.

Routine immunosuppression is not recommended for typical viral lymphocytic myocarditis [10–14]. It becomes indicated only when biopsy identifies a specific immune-mediated cause, such as giant cell myocarditis (GCM), eosinophilic myocarditis, or cardiac sarcoidosis [3,15]. ESC provides graded recommendations; ACC offers decision algorithms. Both note that most immunomodulatory therapies (e.g., corticosteroids, interferon) show mixed or limited evidence, and IVIG has not demonstrated consistent benefit, except possibly in select fulminant cases [16]. Immunosuppression (often corticosteroids + azathioprine) may be considered for virus-negative inflammatory cardiomyopathy [10–13].

For fulminant myocarditis, both guidelines call for rapid escalation, including high-dose immunosuppression when indicated and mechanical circulatory support (MCS) such as VA-ECMO in cardiogenic shock [3,4,17–19]. Survival improves when MCS is initiated early. Both societies advise urgent transfer to advanced heart failure centers for possible LVAD or transplant if recovery does not occur. ESC specifically recommends immediate biopsy in fulminant cases to identify diagnoses like GCM that require urgent immunotherapy [3].

4.2 Activity Restrictions and Adjunctive Measures: Both guidelines state that patients with active myocarditis must avoid exercise to reduce the risk of arrhythmias and sudden death. The ACC advises no competitive sports or vigorous activity for 3–6 months, with reassessment before returning to play [4]. The ESC recommends at least 1 month of rest from anything beyond daily sedentary activity, followed by an individualized plan based on illness severity and recovery. The same indication is also provided for pericarditis in 2025 ESC guidelines [3]. This highlights a paradigm change from previous 2020 ESC guidelines on sport cardiology, allowing a tailored approach and not fixed bans of 3 to 6 months [20].

The ESC also distinguishes between complete remission (normal symptoms, ECG, biomarkers, and imaging) and remission with residual findings (e.g., persistent LGE or wall-motion abnormalities). Patients with residual abnormalities require a more cautious, personalized return-to-exercise plan and follow-up [3].

Both ESC and ACC emphasize identifying and removing possible triggers of myocarditis, such as cardiotoxic medications or immune checkpoint inhibitors [3,4].

A comparison table of ESC vs. ACC recommendations for therapies for myocarditis in different scenarios is reported in Table 5.

Therapy for pericarditis

ESC and ACC largely agree on treatment [3,4]. First-line therapy for acute pericarditis is high-dose NSAIDs or aspirin plus colchicine, unless contraindicated [21–23]. NSAIDs control pain and inflammation, while colchicine lowers recurrence risk; ESC gives it a Class I, Level A recommendation [3]. Both ESC and ACC recommend colchicine for ≥3 months in acute and ≥6 months in recurrent cases, tapering it last after remission [3,4]. Corticosteroids relieve symptoms but should be avoided initially because they increase recurrence risk. The main difference between ESC and ACC papers is that the 2025 ESC guidelines provide a possible indication for corticosteroids in specific cases (contraindications or failure of NSAIDs, systemic inflammatory disease on maintenance therapy with corticosteroids, post-cardiac injury syndromes, renal failure, pregnancy, triple therapy with NSAID and colchicine in recurrent cases) [3].

Second-line and adjunct therapies

In both ESC guideline and ACC position paper, the major therapeutic advance is IL-1 inhibition (anakinra, rilonacept) [24,25]. Interleukin-1 (IL-1) blockade is used in pericarditis because IL-1 is a central driver of the autoinflammatory cascade underlying pericardial inflammation; among the three available IL-1–targeting agents, we highlight anakinra and rilonacept due to their strongest clinical evidence and regulatory approval for recurrent pericarditis, while canakinumab and the newer IL-1 trap goflikicept have more limited but emerging data in this setting.

ESC gives a Class I A recommendation for IL-1 blockers in recurrent, steroid-dependent pericarditis, and suggests they may help even when CRP is normal but CMR shows inflammation [3,4]. ACC also endorses IL-1 blockers for inflammatory phenotypes, but prefers steroids in non-inflammatory presentations (normal CRP) [4]. Both acknowledge that stopping IL-1 therapy often leads to recurrence, so prolonged treatment (1–2 years or more) may be needed. Tapering should be individualized based on disease activity, tolerability, and patient preference.

Overall, both societies strongly support IL-1 inhibitors for refractory pericarditis, with the ACC favoring earlier use in clearly inflammatory cases [4], and ESC recommending them after failure of standard therapy, including triple therapy with NSAID, colchicine, and corticosteroids, giving priority to cost-effectiveness [3].

Myocardial and pericardial interventions

For myocarditis, invasive therapy focuses on advanced heart failure support. Temporary pacing or defibrillators may be used for conduction disease or arrhythmias. If patients deteriorate despite optimal care, mechanical circulatory support (MCS) such as

Table 5
Comparative table of suggested therapies for myocarditis (2025 ESC guidelines vs ACC position paper).

Condition / Scenario	2025 ESC Guidelines – Suggested therapy	ACC Position Paper – Suggested therapy
Uncomplicated acute (no fulminant features)	Supportive care + GDMT for HF/arrhythmias: hemodynamic optimization with ACEi/ARB, beta-blocker (when appropriate), diuretics, monitoring; many recover spontaneously. NSAID for chest pain.	Similar foundation: supportive care + HF/arrhythmia management within staged pathway; noninvasive diagnosis often sufficient in stable patients, therapy aligned to stage and risk.
Routine immunosuppression in "typical" viral/lymphocytic myocarditis	Not recommended routinely. Immunomodulatory strategies overall have mixed/limited evidence; IVIG not consistently beneficial (possible select fulminant scenarios).	Emphasizes uncertainty; immunosuppression reserved for select patients, especially when immune-mediated disease is identified; highlights limited evidence base for broad lymphocytic myocarditis.
Biopsy-proven immune-mediated myocarditis (e.g., GCM, eosinophilic, sarcoid)	Immunosuppression indicated once EMB identifies immune-mediated histotype (particularly if complicated/fulminant course).	Etiology-directed therapy when identified: e.g., high-dose corticosteroids (often + calcineurin inhibitor for GCM), regimens for eosinophilic or sarcoid myocarditis.
Fulminant myocarditis / cardiogenic shock	Rapid escalation; early temporary MCS (often VA-ECMO) in refractory shock; urgent transfer to advanced HF center; EMB ASAP in fulminant cases (even on MCS) to detect entities requiring urgent immunotherapy (e.g., GCM).	Similar: low threshold for advanced HF center; MCS (including VA-ECMO) as lifesaving bridge; emphasizes advanced-center capabilities and staged management (Stage D).
Empiric immunosuppression in fulminant forms (before biopsy/virology results)	ESC highlights immediate/urgent EMB in fulminant cases to guide immunotherapy; empiric immunosuppression is generally not positioned as routine without diagnostic direction, but urgent diagnosis is prioritized to avoid delays when immune histotypes suspected.	ACC stresses that immunosuppression is indicated particularly for histologically confirmed immune-mediated myocarditis; acknowledges practice variation and need for multidisciplinary input; provides practical steroid regimens used in case series.
Virus-negative lymphocytic myocarditis / inflammatory cardiomyopathy	Immunosuppression may be considered for virus-negative inflammatory cardiomyopathy (commonly corticosteroids + azathioprine), reflecting selective use.	Notes some support (e.g., TIMIC and observational data) for select patients with lymphocytic myocarditis when EMB viral PCR is negative; not unanimous consensus; recommends multidisciplinary team involvement.
IVIG / interferon / other immunomodulators	IVIG: no consistent benefit overall; possibly in select fulminant cases; broader immunomodulatory therapies have mixed evidence.	IVIG may be considered in certain inflammatory/autoimmune contexts; antivirals evolving—consult ID specialists; overall immunomodulator evidence limited.
Arrhythmia / conduction support	Temporary pacing/defibrillator strategies for complications; advanced HF interventions if deterioration.	Similar emphasis within staged management and advanced-center care pathways.

Abbreviations: ACEi/ARB = angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; GDMT = guideline-directed medical therapy; EMB = endomyocardial biopsy; GCM = giant cell myocarditis; MCS = mechanical circulatory support; VA-ECMO = veno-arterial extracorporeal membrane oxygenation.

ECMO or LVAD, or heart transplantation, may be required [3,26]. Some fulminant cases recover fully with temporary MCS plus immunotherapy; others require transplant if no improvement occurs after several weeks [3,4].

Interest in IL-1 and IL-6 inhibitors for myocarditis exists, but evidence is limited. The ARAMIS trial did not show clear benefit for anakinra in virus-negative acute myocarditis. Thus, unlike pericarditis, IL-1 blockade is not standard myocarditis therapy. ACC mentions IL-1 inhibitors only when myocarditis coexists with systemic inflammatory disease or pericarditis.

Both ESC and ACC reserve invasive procedures for complications or refractory pericarditis [3,4]. Pericardiocentesis is Class I for cardiac tamponade or large symptomatic effusions and also provides fluid for diagnostic testing. For constrictive pericarditis unresponsive to medical therapy, pericardiectomy is the definitive treatment. ESC distinguishes active constrictive pericarditis (with inflammation—try anti-inflammatory therapy first) from pericardial constriction (fibrotic—surgery recommended early). According to 2025 ESC guidelines, early pericardiectomy is suggested to improve outcomes in pericardial constriction with a careful evaluation of pre-operative tricuspid regurgitation, that can worsen after removal of the pericardium [3]. ACC notes that radical pericardiectomy is a last resort for severe, treatment-resistant cases, ideally at specialized centers [4].

Overall, ESC and ACC remain closely aligned. Myocarditis is managed mainly with supportive care and targeted immunosuppression when biopsy is indicated; advanced interventions (MCS, pericardiectomy, transplant) are recommended in complicated cases. For pericarditis NSAIDs + colchicine is recommended as first-line therapy; cautious steroid use; IL-1 inhibitors for steroid-dependent recurrent pericarditis, but with earlier use in ACC document also before corticosteroid use [4]. A comparison table of ESC vs. ACC recommendations for therapies for pericarditis in different scenarios is reported in Table 6.

Prognosis and risk stratification

Myocarditis prognosis

Myocarditis has a wide range of outcomes—from full recovery to chronic heart failure or sudden cardiac death (SCD) [3,4,32]. Both ESC and ACC stress early risk assessment [3,4]. While many young male patients recover, up to 30 % of biopsy-proven cases may progress to dilated cardiomyopathy [3,4,10–12].

Fulminant myocarditis carries high short-term risk: about 25 % of severe cases result in death or transplant during hospitalization. Patients with reduced LV function, extensive LGE on CMR, or arrhythmias at presentation have worse prognosis. Persistent LGE on follow-up CMR increases the risk of ventricular arrhythmias and sudden death [3,4].

Both guidelines recommend close rhythm monitoring, including ECG during the acute phase and Holter monitoring after discharge. Ventricular arrhythmias can occur early during active inflammation; monomorphic VT typically appears later from scar and warrants careful risk stratification due to the high recurrence risk (up to 40 %) [27]. 2025 ESC guidelines suggest to wait for recovery in patients with arrhythmic presentations related to the acute inflammatory phase (e.g. those presenting with VF or polymorphic VT) with the possible use of a wearable cardiac defibrillator for 3 to 6 months and then reassess the need for primary prevention of SCD [3]. High-grade AV block usually resolves, so temporary pacing is preferred; permanent pacemakers are rarely needed.

ESC uses the category "inflammatory cardiomyopathy" for chronic cases with ongoing inflammation and remodeling, who

Table 6
Comparative table of suggested therapies for pericarditis (2025 ESC guidelines vs ACC position paper).

Condition / Scenario	2025 ESC Guidelines – Suggested therapy	ACC Position Paper – Suggested therapy
Acute first-line therapy	High-dose NSAID or aspirin + colchicine if no contraindications; colchicine has Class I, Level A recommendation; NSAIDs typically 1–2 weeks or until CRP normalizes, then taper.	Same first-line: NSAID/aspirin + colchicine, with colchicine for ≥3 months in acute disease.
Colchicine duration and "how to wean"	Continue colchicine ≥3 months (acute) and ≥6 months (recurrent); taper colchicine last, after stable remission.	Same principle: colchicine ≥3 months acute and ≥6 months recurrent; explicitly: taper last after remission.
Corticosteroids (role and indications)	Avoid as initial therapy due to recurrence risk, but 2025 ESC provides specific indications when needed; suggests prednisone 0.2–0.5 mg/kg (with colchicine) and very slow taper over months.	Generally, reserves steroids for NSAID contraindications or non-inflammatory phenotypes (often immune-mediated) where NSAIDs less effective; also recommends slow taper to prevent rebound.
IL-1 inhibitors (anakinra / rilonacept): when to use	Major advance but positioned after failure of conventional therapy (including combinations); Class I A for recurrent, steroid-dependent pericarditis; Class IIa even if CRP normal but CMR shows inflammation.	Endorses IL-1 inhibitors as pivotal second-line for clearly inflammatory phenotypes (elevated CRP), often earlier in algorithm than ESC in those cases; prefers steroids when non-inflammatory phenotype (normal CRP).
IL-1 inhibitor duration and taper	Recognizes frequent relapse when stopped; prolonged therapy (often 1–2+ years) may be required; taper individualized based on activity/tolerance/preference.	Similar: relapse common upon discontinuation; prolonged treatment often needed; taper individualized.
"After IL-1 failure" / adjunct immunosuppressants	Acknowledges use of IVIG and steroid-sparing immunosuppressants (e.g., azathioprine/methotrexate) mainly in difficult steroid-dependent cases (more limited evidence).	Lists azathioprine and IVIG as options that "may be considered" in refractory cases failing steroids and IL-1 agents.
Constrictive disease with inflammation on CMR: "immunosuppression protocol/guide"	New terminology: "constrictive pericarditis" = active constriction with inflammation → try anti-inflammatory therapy first; "pericardial constriction" = chronic non-inflammatory → early pericardiectomy.	Notes pericardiectomy is last resort for severe refractory disease; emphasizes referral to expert centers. (CMR-guided inflammatory vs non-inflammatory phenotype approach is consistent with escalation strategy.)
Interventions (effusion/tamponade; refractory constriction)	Pericardiocentesis for tamponade / large symptomatic effusions; pericardiectomy definitive for refractory constriction, with earlier surgery in non-inflammatory constriction.	Similar: pericardiocentesis for tamponade; radical pericardiectomy reserved for refractory cases at specialized centers.

Abbreviations; CMR = cardiac magnetic resonance; CRP = C-reactive protein.

should receive standard heart failure therapy [3]. ACC includes these patients in Stage D, who may require durable LV assist devices or transplant [4].

Both societies note that genetics may influence prognosis. Recurrent myocarditis or progression to unexplained cardiomyopathy should prompt genetic testing, as some cases reflect underlying familial cardiomyopathies (e.g. desmosomal or titin variants) [27]. Myocarditis may sometimes represent the first manifestation of a genetic arrhythmogenic cardiomyopathy (hot phases of an arrhythmogenic CMP) [27]. A recurrent pericarditis with a family history and/or an inflammatory phenotype[28] may be associated with genetic variants related to autoinflammatory or immune diseases in 10–15 % of cases [29], warranting genetic testing since such cases have a worse prognosis with more recurrences and possible need for targeted and prolonged therapies [30].

According to the 2025 ESC Guidelines on Inflammatory Myocardial and Pericardial Syndromes, genetic testing should be considered in patients with definite myocarditis or pericarditis who have a family history of inherited cardiomyopathies, sudden cardiac death, or recurrent inflammatory disease to identify underlying pathogenic variants that may influence management and prognosis; testing may also be appropriate in cases suggesting specific genetic or systemic conditions, where genotype-guided diagnosis could alter therapeutic strategies.

Pericarditis prognosis

Acute pericarditis is usually self-limited, but recurrence is the main issue, occurring in 20–30 % of idiopathic cases—especially when colchicine is not used [21–23]. Risk factors include early corticosteroid use, inadequate anti-inflammatory treatment, autoimmune causes, poor response to NSAIDs, high CRP, and large initial effusions. Both ESC and ACC advise closer follow-up in these high-risk patients [3,4].

Constrictive pericarditis is rare (<1 % of idiopathic cases) [31]. When due to acute inflammation, it may be transient and resolve with medical therapy. Serial imaging (echo or MRI) is recommended to confirm resolution.

The introduction of IL-1 inhibitors (anakinra, rilonacept) has transformed outcomes for recurrent pericarditis, producing very low relapse rates. The main challenge is recurrence after stopping therapy; long-term use is often needed. Side effects are generally manageable. Mortality in idiopathic pericarditis is extremely low when treated properly, apart from reversible tamponade events.

Follow-Up recommendations

Both ESC and ACC emphasize that management continues well beyond hospital discharge [3,4]. The ESC 2025 guidelines recommend structured myocarditis follow-up at 6, 12, and 24 months, with more frequent visits for complicated cases [3]. Follow-up should include a clinical exam, ECG, Holter monitoring, echocardiography, and usually a CMR at 3–6 months. Uncomplicated patients typically need only the scheduled visits; those with persistent abnormalities may require lifelong monitoring. The ACC also supports ongoing reassessment, especially for Stage C/D patients, advising repeat troponin, echocardiography, and CMR at 3–6 months to guide return-to-exercise decisions [4].

For pericarditis, follow-up focuses on confirming resolution of inflammation—normal CRP, symptom improvement, and effusion clearance—followed by slow medication tapering. The ACC recommends specialized Pericardial Disease Centers for refractory cases, with frequent visits during active disease and semiannual visits once stable [5,6].

Both guidelines stress patient education, early recognition of recurrence, and consistent monitoring. Innovations such as CMR-based risk stratification and genetic testing are acknowledged as important tools for long-term prognosis [3,6].

Areas of alignment, divergence, and clinical application

Alignment

The 2025 ESC and contemporary ACC guidelines reflect a broad consensus on managing myocarditis and pericarditis. Both recommend a multidisciplinary, evidence-based approach that integrates advanced diagnostics and individualized therapy. Key areas of agreement include:

- Diagnostic paradigm shift: Both guidelines move from routine invasive biopsy toward reliance on CMR for diagnosis of myocarditis, with biopsy reserved for select intermediate/high-risk. Similarly, both incorporate CMR and CRP into the diagnostic criteria for pericarditis, recognizing the contemporary role of multimodality imaging.
- Classification consistency: The time-based definitions of acute, subacute (incessant), chronic, and recurrent are essentially the same in ESC and ACC [3,4], facilitating a common understanding. Both recognize fulminant myocarditis as a distinct emergency subset, and recurrent pericarditis as a common clinical challenge.
- First-line treatment: There is uniform recommendation for NSAIDs + colchicine in acute pericarditis, avoidance of early corticosteroids unless absolutely necessary, and standard heart failure therapy plus exercise abstinence in myocarditis. Both also agree on using beta-blockers judiciously in pericarditis patients who have inappropriate sinus tachycardia or persistent pain (the ESC gives this a Class IIa recommendation) – the ACC similarly mentions heart rate control as part of symptom management in pericarditis.
- IL-1 inhibitors in recurrent pericarditis: Both have embraced the evidence by endorsing anakinra and rilonacept for difficult recurrent cases. This represents a nearly paradigm-changing approach for patients with multiple recurrences, offering a high likelihood of remission where before frequent relapses were common.
- Follow-up and lifestyle: Both stress follow-up imaging (especially CMR in myocarditis) and exercise restriction for a defined period post-myocarditis (3–6 months in the ACC documents [4,5], vs at least 1 month and then individualized in ESC guidelines) [3]. The importance of specialist referral for complicated cases is highlighted by both (e.g., referral to advanced HF centers or dedicated pericardial centers for complex disease).
- Patient-centered care: Both documents provide detailed algorithms for clinical management and emphasize tailoring therapy duration and choices to patient response (e.g., tapering schedules based on clinical remission, or continuing IL-1 blockers until a stable remission is achieved). They also encourage addressing patient quality of life and psychological health. This holistic, patient-focused approach is a notable alignment.

Divergence

Differences between the ESC and ACC are relatively minor, but a few substantive points deserve attention:

- Myocarditis staging vs. no staging: The ACC's introduction of Stage A/B (at-risk and subclinical myocarditis) is a conceptual addition not explicitly used by the ESC (which does not formally stage myocarditis in this manner). This could influence how proactively one monitors certain patients: for example, the ACC might label a chemotherapy patient with an asymptomatic troponin rise as "Stage B myocarditis" and initiate closer follow-up or therapy, whereas the ESC might not formally call it myocarditis at that point. As research evolves, this staging approach might become more widely adopted if it proves useful in guiding management.
- Mandatory chest pain in pericarditis criteria: The ACC insists on chest pain for diagnosing acute pericarditis [5,6], whereas the ESC recognizes possible atypical presentations without chest pain (e.g., dyspnea as the primary symptom in an elderly patient with a large effusion) if other criteria are met [3,8,9].
- Terminology: The ESC uses a new acronym "IMPS" (Inflammatory Myopericardial Syndrome) to highlight the spectrum of disease from pure myocarditis to pure pericarditis through mixed forms [3]. This underlines the possible common etiology and anatomical continuum in these conditions. Both ESC and ACC still use the term "myopericarditis" to describe overlap cases, so in practice terminology is not confusing. Another minor difference is ESC's use of "inflammatory cardiomyopathy"[3] for chronic myocarditis with dysfunction, which the ACC does not specifically name (it addresses those cases under general heart failure management) [4].
- Level of evidence grading: The ESC provides formal class (I, IIa, IIb, III) and level (A, B, C) gradings for recommendations, whereas the ACC documents, as expert consensus statements, do not assign levels of evidence in the same way (though they sometimes indicate in text whether something is based on expert opinion or trial data). Fundamentally, however, they agree on the recommended actions even if the presentation differs.
- Pediatric and special populations: While not heavily detailed above, there are small differences in emphasis. The ESC has dedicated sections on myocarditis/pericarditis in children and on management in pregnancy/lactation. The ACC documents might mention these in passing but not as in-depth. This reflects the ESC guideline's broad scope vs. the ACC's concise focus. For instance, ESC notes that myocarditis in children often differs in presentation and outcomes, and that pregnancy requires special management considerations (avoiding certain drugs, timing of interventions).[3] It should be noted that in pregnancy, both guidelines advise avoiding NSAIDs in late gestation, while low-dose corticosteroids are considered safe when necessary; colchicine is regarded as compatible according to ESC 2025, whereas ACC guidance remains more cautious. Additionally, azathioprine and intravenous immunoglobulins (IVIG) are deemed safe for use during both pregnancy and breastfeeding [3]. The use of IL-1 inhibitors is still debated, as current evidence is limited and primarily based on retrospective studies, though it appears somewhat favorable. In particular, anakinra might be maintained throughout conception, pregnancy, and breastfeeding in women with rheumatic and musculoskeletal conditions when other treatment options are not feasible [3].

From a clinical applicability standpoint, these guidelines are largely complementary. The ESC guideline is comprehensive (spanning everything from epidemiology to patient advice) [3], whereas the ACC documents are targeted "decision pathways" or "consensus statements" [4–6]. Many clinicians may find it useful to consult both. In practice, a clinician following either will arrive at very similar management plans: obtain early CMR in uncomplicated myocarditis, start colchicine for pericarditis, use heart failure medications for myocarditis with heart failure, avoid unnecessary steroids, and consider advanced therapies for severe cases. The slight differences (like the ACC's stage classification concept) do not result in fundamentally different treatments but rather offer additional perspective. The key innovations introduced – (1) formally acknowledging non-invasive diagnostic pathways (reducing reliance on biopsies except when truly needed), (2) integrating novel immunotherapies (IL-1 blockers) into standard care, and (3) highlighting the value of specialized centers and multidisciplinary teams for complex cases – are common to both approaches.

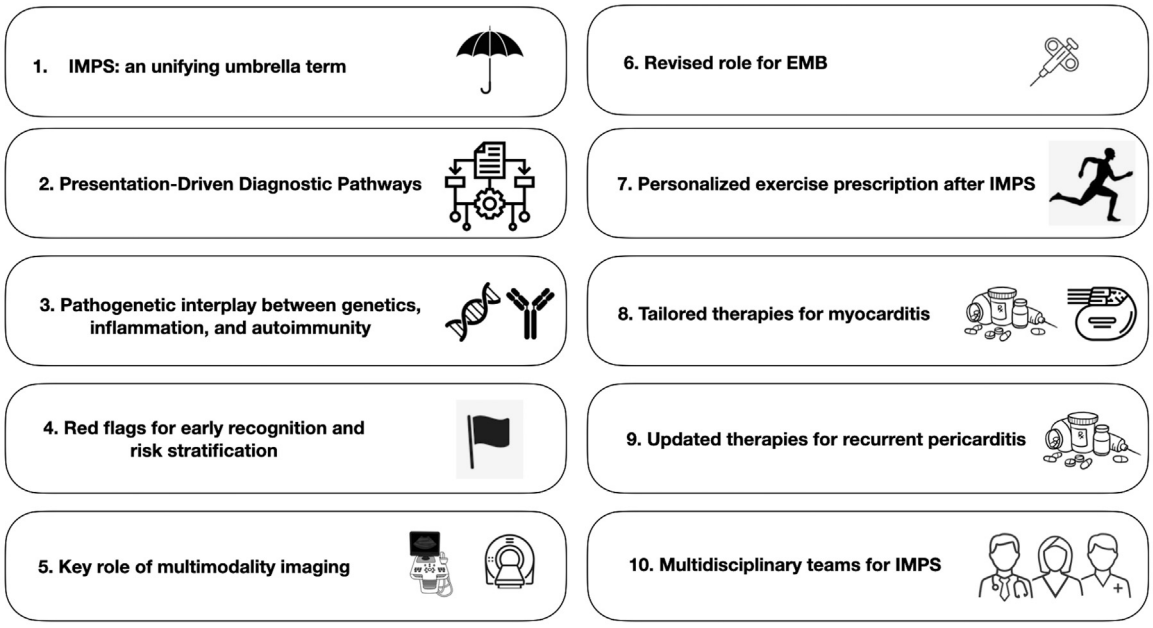

Fig. 1. The 10 key novelties in the clinical management of myocarditis and pericarditis according to the 2025 European guidelines and ACC papers.

Conclusions

The 2025 ESC guidelines[3] and the latest ACC consensus statements[4,5] represent a major step forward in standardizing care for myocarditis and pericarditis. Both emphasize diagnosing these conditions with greater accuracy through noninvasive imaging and biomarkers, stratifying risk to guide treatment intensity, using established therapies early (NSAIDs, colchicine, heart failure medications), and incorporating new options such as IL-1 inhibitors for difficult cases. They also highlight individualized care, multidisciplinary management for complex presentations, and close follow-up to detect persistent or recurrent disease.

Clinicians should stay current with both ESC and ACC recommendations, which are now highly aligned and usually lead to the same clinical decisions. Advances in imaging and new targeted immunotherapy are already improving outcomes and transforming conditions once marked by high morbidity into more manageable diseases [33]. Continued collaboration among international societies will likely refine these strategies further. By applying the shared principles of prompt imaging, selective biopsy, targeted anti-inflammatory therapy, tailored exercise restriction, and structured follow-up, cardiologists can deliver state-of-the-art care for patients with myocarditis and pericarditis (Fig. 1).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript. No conflicts of interest are relevant to this submission.

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