UPDATES IN THE 2025 ESC GUIDELINES FOR MYOCARDITIS AND PERICARDITIS: AN INTEGRATIVE APPROACH TO INFLAMMATORY MYOPERICARDIAL SYNDROMES AND IMPLICATIONS FOR CLINICAL PRACTICE

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Apstrakt: Introduction: Myocarditis is an inflammatory disease of the myocardium that can present with highly heterogeneous clinical manifestations, ranging from asymptomatic forms to fulminant heart failure and sudden cardiac death. The aim of this review paper is to present contemporary diagnostic and therapeutic approaches according to the latest ESC recommendations, integrating clinical experience and emphasizing the need for further research. LITERATURE REVIEW: In 2025, the European Society of Cardiology (ESC) published the first integrated guidelines addressing the diagnosis and treatment of myocarditis and pericarditis, introducing the new concept of inflammatory myopericardial syndrome (IMPS). This umbrella term recognizes the frequent clinical overlap between these two entities and their shared pathophysiological mechanisms. The paper analyzes key updates in classification, diagnostics, genetic evaluation, and therapeutic approaches, with particular emphasis on revised cardiac magnetic resonance (CMR) criteria (Lake Louise criteria), expanded indications for endomyocardial biopsy (EMB), and innovations in the treatment of pericarditis, including interleukin-1 inhibitors. The diagnostic paradigm for myocarditis has been changed. The discussion includes a comparison of ESC recommendations with American ACC/AHA guidelines, as well as contributions from domestic literature, particularly studies in the field of diastolic stress testing and inflammatory cardiomyopathies. The COVID-19 pandemic has further highlighted myocarditis as a potential complication of viral infections. CONCLUSION: The contemporary approach to myocarditis is shifting the paradigm by introducing the concept of inflammatory myopericardial syndrome and involves integrated diagnostics and therapy in accordance with the latest ESC guidelines, recognizing this syndrome as a clinically significant entity. Advances in the use of cardiac magnetic resonance imaging, broader indications for endomyocardial biopsy, and the introduction of targeted therapies, including interleukin-1 inhibitors, enable more precise diagnosis and individualized treatment strategies. Despite these advances, the heterogeneity of clinical presentation remains a challenge in everyday practice. Further research is necessary to improve understanding of the pathophysiology and to optimize the treatment of these patients..

Key words: Myocarditis, pericarditis, inflammatory myopericardial syndrome, ESC guidelines, cardiac magnetic resonance imaging, COVID-19, endomyocardial biopsy.

INTRODUCTION

The 2025 ESC guidelines represent a turning point in the approach to inflammatory heart diseases, unifying myocarditis and pericarditis within a single framework [1]. This decision stems from an increasingly clear understanding that these two entities are functionally, anatomically, and pathophysiologically closely related, and that treating them separately often leads to fragmentation in diagnosis and therapy. The new concept of inflammatory myopericardial syndrome (IMPS) serves as an umbrella term encompassing a clinical continuum ranging from isolated myocarditis, through combined myopericarditis and perimyocarditis, to isolated pericarditis, including complex mixed forms, up to chronic inflammatory cardiomyopathy and constrictive pericarditis [2–5, 6–8]. Such an integrative approach aims to improve collaboration among specialists and guide future research.

The term inflammatory cardiomyopathy has also been introduced, referring to chronic myocarditis associated with cardiac dysfunction and ventricular remodeling with a hypokinetic phenotype, with or without dilation. The role of cardiac magnetic resonance (CMR) is now central in diagnostics [9–11]. In the domestic literature, among others, contributions by the

authors of this review and their collaborators have advanced the understanding of diastolic dysfunction and echocardiographic parameters in inflammatory cardiomyopathies [12–14], while endomyocardial biopsy is recommended in high-risk cases [1, 15–21]..

EPIDEMIOLOGY AND CLASSIFICATION

The guidelines report an incidence of pericarditis ranging from 3 to 32 cases per 100,000 inhabitants per year, while the incidence of myocarditis is between 6 and 8 cases per 100,000 inhabitants [1,15]. Higher rates have been observed in men and younger adults. A particular challenge is the fact that a large number of subclinical and mild cases, including those diagnosed within the context of MINOCA (myocardial infarction with non-obstructive coronary arteries), remain undiagnosed [6,9], which may lead to underestimation of the true incidence and prevalence of chronic forms of the disease.

IMPS is classified into the following clinical entities: isolated pericarditis, isolated myocarditis, myopericarditis (predominant pericarditis with mild myocardial involvement), perimyocarditis (predominant myocarditis with pericardial features), and chronic inflammatory cardiomyopathy (chronic myocarditis with structural remodeling, dysfunction, and an arrhythmogenic substr [1].

ETIOLOGY AND PATHOPHYSIOLOGY

The etiology of myocarditis and pericarditis is heterogeneous. In developed countries, viral infections predominate (enteroviruses, adenoviruses, parvovirus B19, human herpesvirus 6, influenza virus, hepatitis C virus) [5,16,17], whereas in endemic regions tuberculosis remains an important cause of pericarditis, particularly in predisposed individuals with HIV infection. Bacterial causes (diphtheria, borreliosis, staphylococcal infections) are less common. Autoimmune mechanisms may lead to inflammation in the context of systemic diseases (lupus, sarcoidosis, vasculitis), while toxic agents (anthracyclines, alcohol, cocaine) and drugs (checkpoint inhibitors) can also induce myocarditis. A genetic basis plays an important role in susceptibility to viral infections and in determining the severity of the clinical presentation, with variants in sarcomeric and desmosomal genes being associated with myocarditis, and autoinflammatory diseases with recurrent pericarditis [6,8,15].

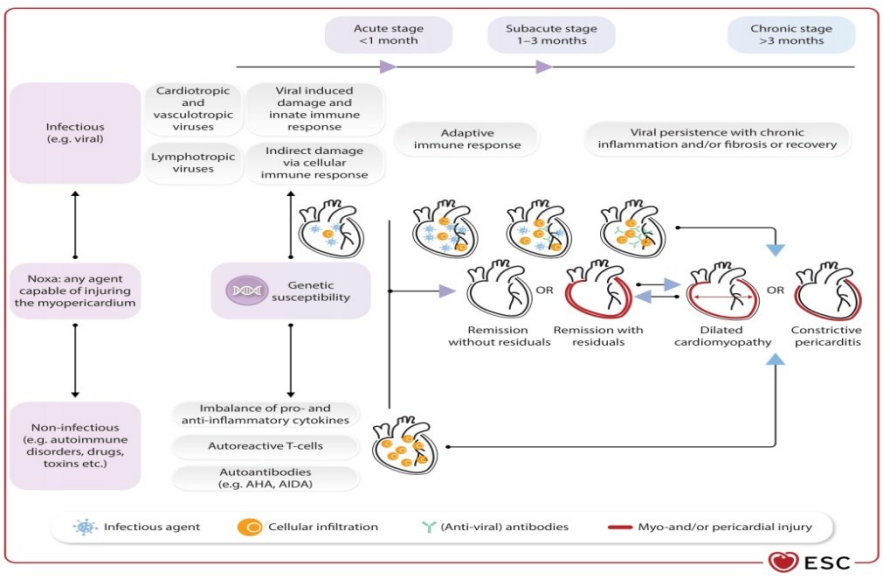
The pathophysiology of myocarditis is complex and involves acute, immune-mediated, and chronic phases of inflammation [5,6,17], progressing through several stages (Figure 1):

Acute phase: Direct myocardial injury caused by pathogen entry (e.g., viruses) into cardiomyocytes, leading to cell necrosis.

Immunological phase: Activation of the innate and adaptive immune response (macrophages, T-lymphocytes) with the release of cytokines and inflammatory mediators, resulting in further myocardial damage. Genetic variants in sarcomeric and desmosomal genes are increasingly being identified [1,18–20].

Chronic phase: In some patients, the inflammatory response persists, leading to progressive myocardial fibrosis, ventricular remodeling, and the development of dilated cardiomyopathy..

Figure 1. Stages of inflammatory myopericardial syndrome. AHA – anti-cardiac antibodies; AIDA – antibodies against the intercalated disc. Adapted from: *Eur Heart J*, Volume 46, Issue 40, 21 October 2025, Pages 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192



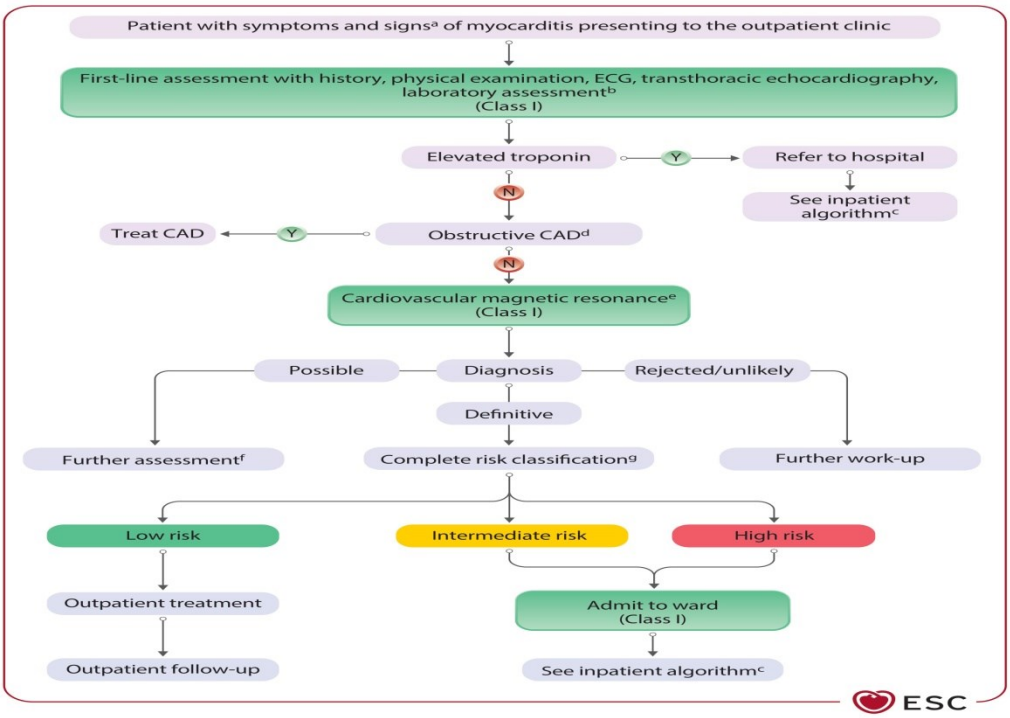
CLINICAL PRESENTATION

The clinical presentation of IMPS is highly variable. According to the time course, myocarditis is classified into: acute (≤4 weeks), subacute (4–12 weeks), and chronic (>3 months, with persistent inflammation and remodeling).

Myocarditis may present with a wide spectrum of clinical manifestations, ranging from mild or asymptomatic forms to heterogeneous clinical phenotypes, including asymptomatic mild myocarditis, chest pain, perimyocarditis, heart failure, arrhythmic presentation, sudden cardiac death, and fulminant myocarditis with fulminant heart failure and cardiogenic shock [6,18–20].

Myocarditis is considered complicated when there is LVEF <50%, acute heart failure, ventricular arrhythmias, or high-degree atrioventricular (AV) block. Complicated forms may be associated with ventricular arrhythmias, AV blocks, and significantly reduced ejection fraction [7–9,18–20] (Figure 2).

Figure 2. Diagnostic algorithm and triage for outpatients with myocarditis. Adapted from: *Eur Heart J*, Volume 46, Issue 40, 21 October 2025, Pages 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192



The term fulminant myocarditis [21] is reserved for patients presenting with cardiogenic shock and the most severe form of the disease, which often requires intensive treatment and mechanical circulatory support.

Pericarditis clinically manifests as dry (fibrinous), effusive, as an impending or acute cardiac tamponade, reversible partial constriction, or a chronic constrictive form [2,4]. Timely differentiation between inflammatory and non-inflammatory phenotypes is crucial.

DIAGNOSTIC APPROACH

The 2025 ESC recommendations significantly reshape the essence of the diagnostic pathway and disease staging, reflecting a paradigm shift in the diagnostic process. This is largely driven by the major role of cardiac magnetic resonance imaging (CMR), which has become the gold standard for diagnosing myocarditis. At the same time, the role of endomyocardial biopsy has been refined and is now mainly reserved for severe, unclear, or high-risk cases, as well as for guiding appropriate therapy based on pathological and histological characterization with immunohistochemistry and PCR detection of viral genomes in the myocardium.

Previously, diagnostic categories included: suspected, confirmed, and proven myocarditis. In the new framework, classification has been updated to: unlikely/excluded, possible, and definite myocarditis (Figure 3 and Tables 1 and 2)..

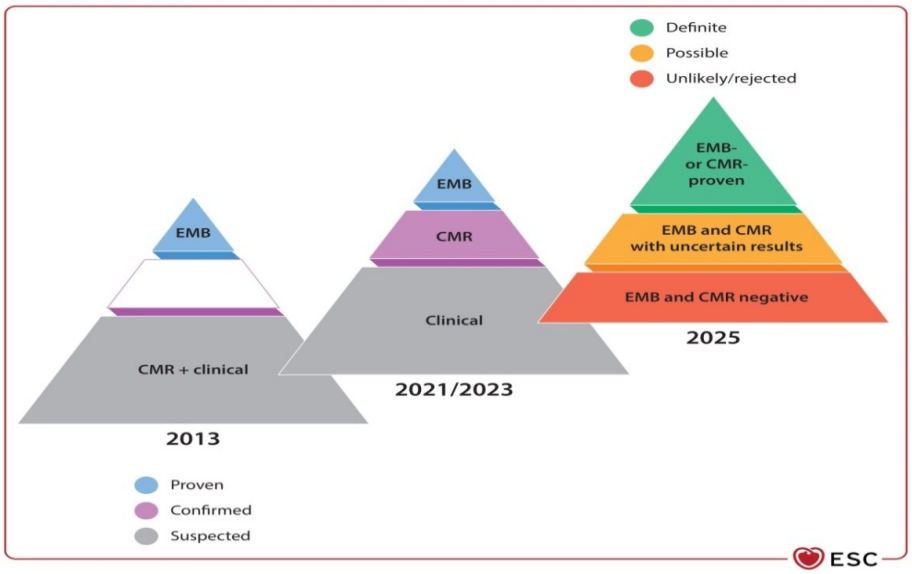
Table 1. Diagnostic criteria and classification for inflammatory myopericardial syndrome (IMPS)
If diagnostic criteria for myocarditis and/or pericarditis are fulfilled

Category	Myocarditis	Pericarditis
Definite	Clinical presentation + confirmation by CMR or EMB	Clinical presentation with >1 additional criterion
Possible	Clinical presentation with at least 1 additional criterion; CMR or EMB inconclusive or unavailable	Clinical presentation with 1 additional criterion
Unlikely / Excluded	Clinical presentation only, without additional criteria	Clinical presentation only, without additional criteria

Table 2. Additional criteria (in addition to clinical presentation)

Criterion	Myocarditis	Pericarditis
Clinical	Nonspecific findings	Pericardial friction rub
ECG	ST–T changes	PR depression, diffuse ST-segment elevation
Biomarkers	Elevated troponin	Elevated CRP
Imaging	Abnormal strain, wall motion abnormalities, reduced ejection fraction (EF)	New or worsening pericardial effusion
CMR findings	Myocardial edema and/or LGE (late gadolinium enhancement)	—

Figure 3. Paradigm shift in the clinical diagnosis of myocarditis



Basic diagnostic elements

Initial evaluation – biomarkers: Mandatory measurement of troponin (marker of myocardial injury/necrosis), high-sensitivity C-reactive protein (hsCRP, marker of inflammation), and natriuretic peptides BNP/NT-proBNP (markers of heart failure). Routine viral serologies are not recommended [1,3,5,19–20].

Electrocardiography (ECG): May show nonspecific ST-segment changes [5] (ST elevation/depression, T-wave changes, AV block, arrhythmias). There is no typical ECG pattern for myocarditis; however, ECG is rarely completely normal and is considered heterogeneous across different IMPS forms. Diffuse, concave upward ST-segment elevation is typical of pericarditis.

Echocardiography: A basic imaging modality for assessment of ventricular function (LVEF), presence of pericardial effusion, tamponade, and regional wall motion abnormalities. However, it often cannot definitively confirm inflammation [12–14].

If troponin is normal and LVEF is preserved, this indicates a favorable prognosis.

Non-invasive imaging – Cardiac Magnetic Resonance (CMR)

Cardiac magnetic resonance imaging is a key modality according to the revised Lake Louise criteria [1,10,11] and has become central in the diagnostic work-up of IMPS.

Diagnosis is established based on the revised Lake Louise criteria, which require at least one abnormality based on T1 mapping (indicating interstitial fibrosis or edema) and at least one abnormality based on T2 mapping (indicating myocardial edema) [11].

CMR enables detection of edema, necrosis, and fibrosis through late gadolinium enhancement (LGE), typically in an epicardial or mid-myocardial distribution, which helps differentiate inflammatory disease from myocardial ischemia and coronary artery disease.

The 2025 ESC recommendations emphasize an individualized approach based on clinical presentation and risk assessment (Table 3).1].

Table 3. Risk stratification algorithm and triage of patients with suspected myocarditis and pericarditis in outpatient settings. Hospitalization is recommended for all patients with myocarditis and high-risk pericarditis.

Risk	High risk	Intermediate risk	Low risk
Myocarditis	• Acute HF/cardiogenic shock • Dyspnoea NYHA III–IV refractory to medical therapy • Cardiac arrest/syncope[a] • Ventricular fibrillation/sustained ventricular tachycardia[a] • High-level AV block[a]	• New/progressive dyspnoea • Non-sustained ventricular arrhythmias • Persistent release or relapsing troponin	Stable symptoms or oligosymptomatic
	Imaging criteria:	**Imaging criteria:**	**Imaging criteria:**
	• Newly reduced LVEF (<40%)[a] • Extensive LGE on CMR[a]	• Newly mildly reduced LVEF (41%–49%) and/or WMA • Preserved LVEF (≥50%) and LGE ≥2 segments on CMR	• Preserved LVEF (≥50%) without LGE or limited LGE (<2 segments) on CMR
Pericarditis	• Signs and symptoms of cardiac tamponade • Fever (temperature >38°C) • Effusive–constrictive pericarditis • Failure of NSAID therapy • Incessant pericarditis	• Signs and symptoms of right HF	• Response to adequate therapy within 1–2 weeks
	Imaging criteria:	**Imaging criteria:**	**Imaging criteria:**
	• Large PEff (>20 mm end-diastole) • Cardiac tamponade • Extensive pericardial LGE on CMR	• Moderate–large PEff (10–20 mm end-diastole) • Constrictive physiology regardless of the size of the effusion	• Absence or mild PEff • Absence of pericardial LGE on CMR

Invasive diagnostics – Endomyocardial biopsy (EMB)

Endomyocardial biopsy (EMB) is considered the gold standard in severe forms of disease [17,21,22], providing definitive diagnosis and etiological classification. Indications have been expanded compared with previous guidelines and are now reserved for high-risk cases (Table 4). Molecular biological (PCR) analysis for viral genomes is mandatory prior to initiation of immunosuppressive therapy [21–23]].

TABLE 4. Indications for endomyocardial biopsy (EMB) according to ESC 2025 [21]

Clinical situation	Level of recommendation
Fulminant myocarditis with cardiogenic shock	Strong indication
Suspected giant-cell myocarditis	Strong indication
Suspected eosinophilic myocarditis	Strong indication
Acute heart failure of unknown etiology (<2 weeks duration)	Recommended
Heart failure (2 weeks–3 months) with left ventricular (LV) dilation and new ventricular arrhythmias	Recommended
Suspicion of cardiac sarcoidosis or autoimmune myocarditis	Recommended
Refractory ventricular arrhythmias of unknown etiology	Consider
Persistent left ventricular dysfunction without clear cause (LVEF <40%, extensive LGE on CMR)	

GENETIC TESTING

There is increasing evidence of an association between myocarditis and inherited cardiomyopathies. The ESC 2025 guidelines recommend genetic testing in selected patients with familial forms and recurrent pericarditis [1]. Studies highlight mutations in desmosomal genes as part of an inherited predisposition [1], particularly in patients with high diagnostic yield, such as those with:

Family history of cardiomyopathy or sudden cardiac death

Arrhythmic presentation of disease

Presence of septal or "ring-like" LGE on CMR

Persistent systolic dysfunction without recovery

Recurrent myocarditis or pericarditis (a genetic cause is identified in approximately 15% of patients with recurrent pericarditis) [1]

THERAPY OF MYOCARDITIS

Therapeutic management is individualized and depends on etiology, clinical presentation, and hemodynamic status [1,6] (Table 5). In stable patients, analgesics and NSAIDs are used, with the addition of colchicine when pericardial symptoms are present [1,31]. In heart failure, standard HF guideline-directed medical therapy is applied (ACE inhibitors/ARNI, beta-blockers, mineralocorticoid receptor antagonists, SGLT2 inhibitors) [6,29].

Immunosuppressive therapy is reserved for virus-negative forms [21] or specific entities such as giant-cell and eosinophilic myocarditis [22–24]. In fulminant myocarditis, mechanical circulatory support (VA-ECMO) is recommended [1,20].

The treatment of myocarditis is systematized into five key domains:

1. General measures and symptomatic therapy

Restriction of physical activity for 3–6 months. In the acute phase, in hemodynamically stable patients, analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), and colchicine are used if pericardial symptoms are present.

2. Heart failure therapy

In patients with heart failure, standard guideline-directed therapy is used: ACE inhibitors or ARNI, beta-blockers, mineralocorticoid receptor antagonists (MRA), sodium-glucose cotransporter-2 inhibitors (SGLT2i), and diuretics in cases of congestion.

3. Immunosuppressive therapy

Used only in non-infectious etiologies and after exclusion of active viral infection in the myocardium by PCR analysis of EMB samples. Indications include:

Virus-negative inflammatory (autoimmune) myocarditis

Giant-cell myocarditis (strong recommendation)

Eosinophilic myocarditis (strong recommendation)

Myocarditis associated with systemic autoimmune diseases (e.g., sarcoidosis)

Commonly used agents include corticosteroids (prednisone), azathioprine, mycophenolate mofetil, and cyclosporine [1].

4. Antiarrhythmic therapy and devices

Antiarrhythmic drugs are used for symptomatic arrhythmias. Implantable cardioverter-defibrillator (ICD) implantation is considered in patients with persistent LV dysfunction after at least 3 months of optimal therapy. A wearable defibrillator (vest-type device) may be used as a temporary option for 3–6 months [1].

5. Mechanical circulatory support (MCS)

In fulminant myocarditis with cardiogenic shock, MCS serves as a "bridge to recovery" or "bridge to transplantation." Recommended modalities include intra-aortic balloon pump (IABP) and veno-arterial ECMO (VA-ECMO) as the most effective support strategies [20–21].].

Table 5. Therapeutic recommendations for myocarditis according to ESC 2025

Therapeutic approach	Recommendation
Restriction of physical activity for 3–6 months	Recommended (Class II)
Standard heart failure therapy	Recommended (Class II)
Immunosuppressive therapy in virus-negative autoimmune myocarditis	Recommended (Class II)
Corticosteroids in giant-cell or eosinophilic myocarditis	Strong recommendation (Class I)
Antiviral therapy in confirmed viral etiology	Selective
Mechanical circulatory support in cardiogenic shock	Recommended

TREATMENT OF PERICARDITIS

First-line therapy: Aspirin or NSAIDs in combination with colchicine for at least 3 to 6 months (Class I A recommendation) [1–4].

Second-line therapy:

Corticosteroids are considered second-line treatment for refractory forms [2]. They are

used only when first-line therapy fails or is contraindicated.

Refractory pericarditis:

Interleukin-1 (IL-1) inhibitors represent a novel therapeutic option for treatment-refractory and/or recurrent pericarditis, particularly in patients with markedly elevated hsCRP levels (Class I A recommendation in highly inflammatory phenotypes) (Figure 4 and Table 6)..

Figure 4. Proposed algorithm for pharmacological treatment of pericarditis in adults (excluding interventional procedures and pericardiectomy). Adapted from: *Eur Heart J*, Volume 46, Issue 40, 21 October 2025, Pages 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192

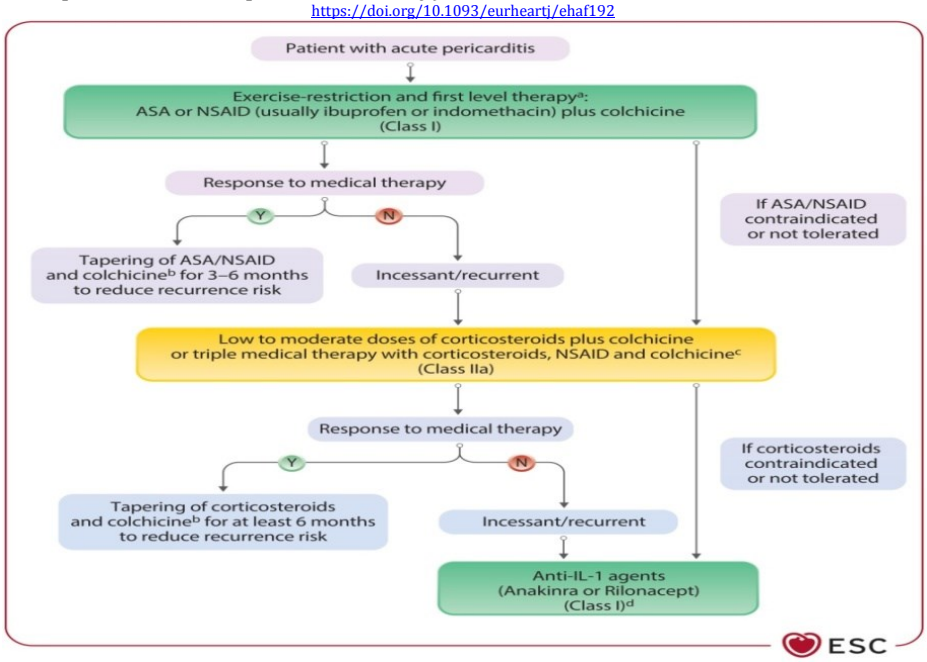


TABLE 6. Therapeutic protocol for the treatment of pericarditis. Adapted from: *Eur Heart J*, Volume 46, Issue 40, 21 October 2025, Pages 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192

Therapy	Dosing	Duration[a]	Tapering[a]
Aspirin[b]	750–1000 mg 3 times daily	1–2 weeks	Decrease by 250 mg every 1–2 weeks
Ibuprofen[b]	600–800 mg 3 times daily	1–2 weeks	Decrease by 200 mg every 1–2 weeks
Indomethacin	25–50 mg 3 times daily	1–2 weeks	Decrease by 25 mg every 1–2 weeks
Colchicine[b]	0.5 mg once daily (<70 kg or severe renal impairment) or 0.5 mg twice daily	3–6 months	Not required
Prednisone	0.2–0.5 mg/kg/day	2–4 weeks	Several months
Treatment for recurrences only:			
Azathioprine	Starting with 1 mg/kg/day then gradually increased to 2–3 mg/kg/day	Several months	Several months
IVIG	400–500 mg/kg i.v. daily for 5 days	5 days	Not required
Anakinra	1–2 mg/kg/day up to 100 mg/day in adults	At least 6 months/ >12 months	Needed (at least 3–6 months)/ unknown
Rilonacept[c]	320 mg once daily followed by 160 mg weekly		

TABLE 7. SUMMARY OF ESC RECOMMENDATIONS FOR THE TREATMENT OF INFLAMMATORY MYOPERICARDIAL SYNDROME (IMPS)

Recommendations	Class[a]	Level[b]
Management of symptoms		
NSAIDs (together with proton pump inhibition) should be considered in patients with associated symptoms of pericarditis to reduce symptoms.	IIa	C
Colchicine should be considered in patients with myopericarditis to reduce recurrences.[263]	IIa	B
Management of heart failure		
Adherence to the ESC HF guidelines is recommended in cases of myocarditis with LV systolic dysfunction and/or HF to reduce symptoms and to improve LV function.[12]	I	C
HF therapy should be considered in patients with myocarditis and LV systolic dysfunction for at least 6 months upon complete LV functional recovery to stabilize LV function.	IIa	C
Management of arrhythmias		
β-Blockers, with a continuation for at least 6 months, should be considered in patients with acute myocarditis, especially those with troponin elevation, to control symptoms and prevent arrhythmias.	IIa	C
Anti-arrhythmic treatment should be considered in post-myocarditis patients with recurrent, symptomatic VT to reduce arrhythmic burden.[58]	IIa	C
Immunosuppressive therapy		
Corticosteroids should be considered in patients with fulminant, non-infectious forms of myocarditis to stabilize the patients.	IIa	C
Corticosteroids may be considered in patients with acute myocarditis with impaired LVEF if refractory to standard HF therapy to stabilize patients.	IIb	C
Routine use of immunosuppressive therapy is not recommended in acute myocarditis with preserved LV function because no outcome benefit has been shown.	III	C

ESC, European Society of Cardiology; HF, heart failure; LV, left ventricle; LVEF, left ventricular ejection fraction; NSAID, non-steroidal anti-inflammatory drug; VT, ventricular tachycardia.
[a]Class of recommendation.
[b]Level of evidence.

SPECIAL POPULATIONS

Post-COVID myocarditis: May occur as a result of direct viral infection, systemic inflammatory response, or immune dysregulation (25–26). Diagnosis is often established by CMR, and therapy is mainly supportive. Post-COVID myocarditis and post-vaccination forms have been analyzed in several studies [23–28]. Rare cases of post-vaccination myocarditis after mRNA vaccines have been reported, most commonly in young males within several days after the second dose. The clinical course is usually mild, and the prognosis is favorable. The benefits of vaccination far outweigh the risks [27–30].

Children: Similar diagnostic criteria are applied, but caution is required with corticosteroid use due to potential effects on growth.

Pregnancy: Colchicine may be used for recurrence prevention in pericarditis (Class IIb C recommendation).

Elderly patients:

Dose adjustment of colchicine is necessary, along with monitoring of renal function.

PROGNOSIS AND FOLLOW-UP

Prognosis depends on initial clinical presentation and etiology. The most important predictor of adverse outcome is biventricular dysfunction. Most patients with mild disease achieve full recovery. In a minority of cases, progression to dilated cardiomyopathy and chronic heart failure may occur [14,15].

Follow-up after acute myocarditis is systematic over the first 6–24 months and includes: clinical assessment, ECG, Holter monitoring, biomarkers (troponin, CRP), echocardiography, exercise testing, and CMR in selected cases [1].

Prognosis is influenced by the degree of left ventricular dysfunction and the presence of fibrosis on CMR [6,10,11]. Diastolic dysfunction may persist even after clinical recovery [12–14].

DISCUSSION

The ESC 2025 guidelines represent a significant conceptual advance with the introduction of IMPS, but also raise several controversies.

The most debated issues include: Central role of CMR: Although CMR has become indispensable, its position as a "new gold standard" carries the risk of overdiagnosis and limited accessibility.

Controversial beta-blocker recommendation [1,6]: The recommendation for beta-blocker use in all myocarditis patients for at least 6 months, regardless of EF, is based on limited evidence and is one of the most debated points in the guideline.

Genetic testing approach: Also controversial, being more limited in ESC guidelines compared with broader recommendations in ACC/AHA documents [3,4].

Insufficient commitment to clarifying the etiopathogenesis in the MINOCA population: The guidelines miss the opportunity to refine the definition of IMPS in patients with MINOCA, where myocarditis is one of the most common final findings.

Differences from the American recommendations (ACC/AHA) [1,3]: The European approach is more conservative regarding genetic testing (only selected patients vs. all), earlier use of IL-1 inhibitors in pericarditis (later compared to ACC) and does not include microaxial pumps (e.g. Impella) in the mechanical support algorithms, unlike the American guidelines.

Table 8. Comparison of ESC 2025 and ACC/AHA 2024 guidelines

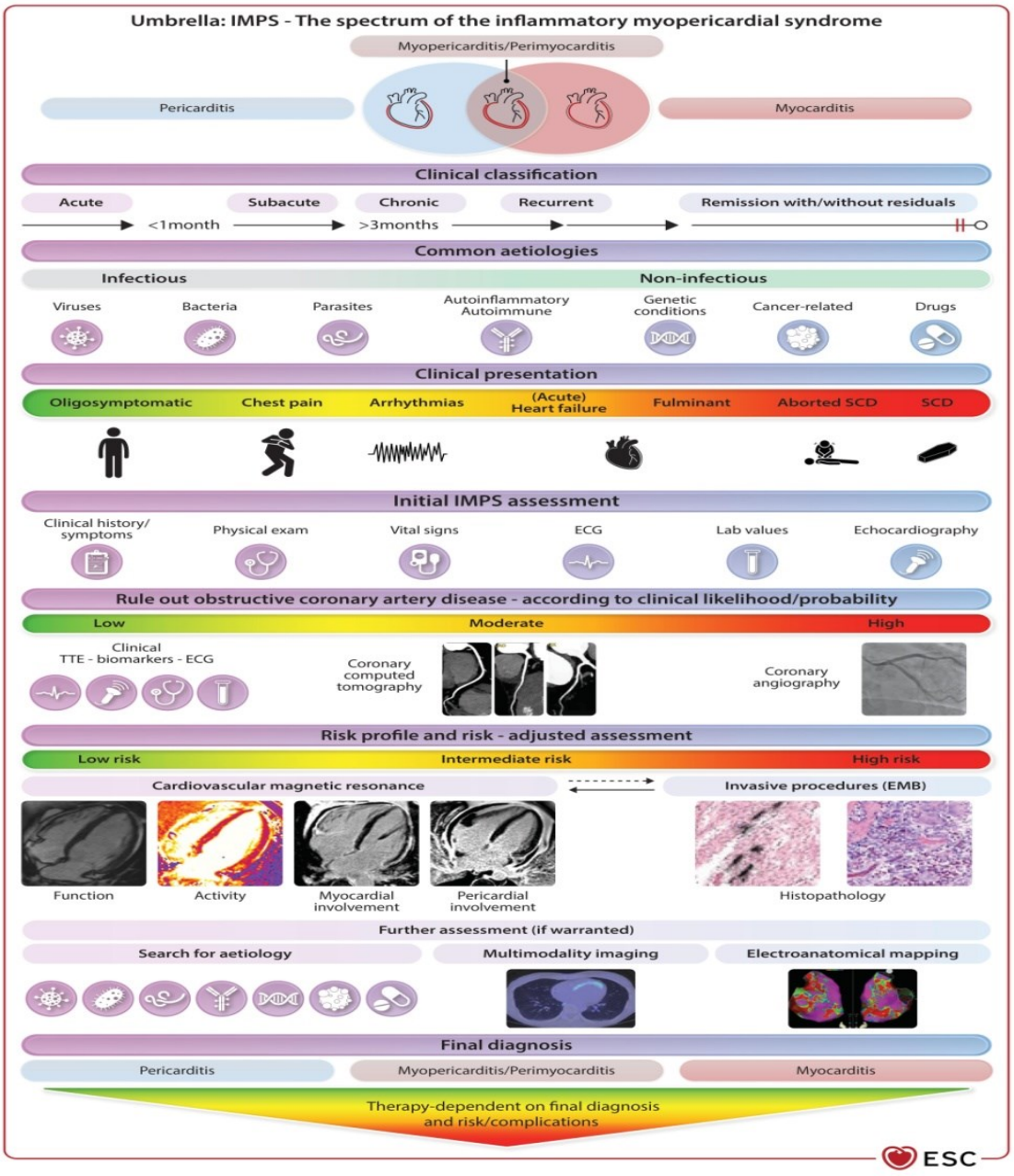
Topic	ESC 2025	ACC/AHA consensus
Genetic testing	Only in selected patients	Recommended in all patients with myocarditis
Beta-blockers	Recommended for ≥6 months in all patients	No universal recommendation
IL-1 inhibitors	More conservative approach (after corticosteroids)	Earlier use after failure of NSAIDs + colchicine
Mechanical support	Emphasis on VA-ECMO	More detailed inclusion of microaxial pumps (e.g., Impella)

The ESC 2025 guidelines introduce the IMPS concept, emphasize the role of CMR, and expand indications for EMB [1,10,17]. In the context of local clinical practice, the studies by the author of this literature review, Dr Dušan Bastać, significantly help bridge gaps in the practical application of diagnostic methods for inflammatory myopericardial syndrome in Serbia [12–14].

His research on diastolic stress testing and echocardiographic markers indicates that diastolic dysfunction and elevated left ventricular filling pressures may persist even after recovery of systolic function (LVEF). This finding is consistent with the concept of inflammatory cardiomyopathy and the HFpEF phenotype (heart failure with preserved ejection fraction) in these patients.

These observations highlight the need for careful, multimodal follow-up even in patients with normalized ejection fraction..

Figure 5. Central illustration of the ESC guidelines on myocarditis and pericarditis. Adapted from: *Eur Heart J*, Volume 46, Issue 40, 21 October 2025, Pages 3952–4041, https://doi.org/10.1093/eurheartj/ehaf192



CONCLUSION

The ESC 2025 recommendations and guidelines represent the most comprehensive document to date, unifying myocarditis and pericarditis into a single concept—inflammatory myopericardial syndrome (IMPS), as illustrated in the figure (Figure 5: Central illustration of ESC guidelines on myocarditis and pericarditis).

The emphasis is on a multimodal diagnostic approach, with a central role of cardiac magnetic resonance imaging (CMR), rational use of endomyocardial biopsy (EMB), selective application of immunosuppressive therapy, and overall personalization of treatment strategies.

Despite significant progress, many recommendations are still based on expert consensus, highlighting the need for further

high-quality research, particularly in areas such as optimal use of beta-blockers, novel antiviral therapies, genetic testing, and biological treatments.

The integration of domestic clinical experience shows that the guidelines are largely compatible with current practice, but also open new questions and research opportunities aimed at improving outcomes in patients with inflammatory heart diseases.

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