

Table 1. Causes of myocarditis/inflammatory cardiomyopathy

Infectious myocarditis

Viral

Bacterial

Staphylococcus, Streptococcus, Pneumococcus, Meningococcus, Gonococcus, Salmonella, Corynebacterium diphtheriae, Haemophilus influenzae, Mycobacterium (tuberculosis), Mycoplasma pneumoniae, Brucella

Spirochaetal

Borrelia (Lyme disease), Leptospira (Weil disease)

Fungal

Aspergillus, Actinomyces, Blastomyces, Candida, Coccidioides, Cryptococcus, Histoplasma, Mucormycoses, Nocardia, Sporothrix

Protozoal

Trypanosoma cruzi, Toxoplasma gondii, Entamoeba, Leishmania

Parasitic

Trichinella spiralis, Echinococcus granulosus, Taenia solium, Plasmodium falciparum

Rickettsial

Coxiella burnetii (Q fever), R. rickettsii (Rocky Mountain spotted fever), R. tsutsugamushi

Immune-mediated myocarditis

Allergens

Tetanus toxoid, vaccines, serum sickness

Drugs

penicillin, cefaclor, colchicine, furosemide, isoniazid, lidocaine, tetracycline, sulfonamides, phenytoin, phenylbutazone, methyl dopa, thiazide diuretics, amitriptyline

Alloantigens

Heart transplant rejection

Autoantigens

Infection-negative lymphocytic, infection-negative giant cell

Associated with autoimmune or immune-oriented disorders: systemic lupus erythematosus, rheumatoid arthritis, Churg-Strauss syndrome, Kawasaki's disease, inflammatory bowel disease, scleroderma, polymyositis, myasthenia gravis, insulin-dependent diabetes mellitus, thyrotoxicosis, sarcoidosis, Wegener's granulomatosis, necrotizing coronary vasculitis, rheumatic heart disease (rheumatic fever)

Toxic myocarditis

Drugs

Amphetamines, anthracyclines, cocaine, cyclophosphamide, ethanol, fluorouracil, lithium, catecholamines, hemetine, interleukin-2, trastuzumab, clozapine, immune check point inhibitors

Heavy metals

Copper, iron, lead (rare, more commonly cause intramyocyte accumulation)

Miscellaneous

Scorpion sting, snake, and spider bites, bee and wasp stings, carbon monoxide, inhalants, phosphorus, arsenic, sodium azide

Hormones

Phaeochromocytoma, vitamins: beri-beri

Physical agents

Radiation electric shock

Viruses associated with myocarditis and inflammatory cardiomyopathy

Viral tropism	Virus	Viral genome	Virulence	Treatment for associated heart disease
Cardiotropic	Adenoviruses	dsDNA	Virulent	IFN α or IFN β (?); direct-acting antiviral therapy (?); intravenous immunoglobulins (?)
	Enteroviruses (coxsackieviruses, echoviruses)	(+)ssRNA		
Vasculotropic	Parvovirus B19	ssDNA	Bystander: latent; low viral DNA copy numbers in cardiac tissue; with or without cardiac inflammation	No need for antiviral therapy
			Virulent: high viral DNA copy numbers in cardiac tissue (>500 viral DNA copies per microgram cardiac DNA) with cardiac inflammation or systemic infection	Intravenous injection of immunoglobulins (?)
Lymphotropic	Cytomegalovirus; Epstein–Barr virus; human herpesvirus 6	dsDNA	Bystander: latent; low DNA copy numbers in cardiac tissue; with or without cardiac inflammation	No need for antiviral therapy
			Virulent: high cardiac DNA copy numbers; with cardiac inflammation	Anti-herpesvirus drugs
Cardiotoxic	Hepatitis C virus; HIV; Influenza viruses	(+)ssRNA	Virulent: cardiac inflammation with viraemia	Direct-acting antiviral therapy
ACE2-tropic; cardiotoxic (?)	Coronaviruses (MERS-CoV, SARS-CoV, SARS-CoV-2)	(+)ssRNA	Virulent: viraemia; cardiac inflammation (?)	Potential treatments currently under investigation: remdesivir; hydroxychloroquine and azithromycin; darunavir and cobicistat; lopinavir–ritonavir; favipiravir; ribavirin; IFN α ; camostat mesylate

(?) indicates unclear, needs further investigation; ACE2, angiotensin-converting enzyme 2; dsDNA, double-stranded DNA; HIV, human immunodeficiency virus; MERS-CoV, Middle East respiratory syndrome coronavirus; SARS-CoV, severe acute respiratory syndrome coronavirus; ssDNA, single-stranded DNA; (+)ssRNA, positive-sense single-stranded RNA.

Tschope C, et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. *Nat Rev Cardiol.* 2020.

Table 2. Major myocarditis subtypes resulting in a fulminant presentation.

Subtype	H&E Findings	Clinical Manifestations	Treatment
Fulminant lymphocytic myocarditis	Extensive dense lymphocytic infiltrate with associated myonecrosis. May have occasional isolated multinucleated giant cells or eosinophils.	Acute heart failure rapidly leading to cardiogenic shock, conduction abnormalities, or ventricular arrhythmias/SCD. Chest pain.	Treatment is primarily supportive; circulatory support as needed to prevent MOSF. Some evidence that in the absence of cardiotropic viral genome by PCR, steroids may be helpful
GCM	Extensive mixed inflammatory infiltrate characterized by the presence of several multinucleated giant cells (usually present after 1–2 wk), eosinophils, monocytes, and macrophages in the absence of noncaseating granulomas. Edema and extensive myonecrosis often present.	Acute heart failure caused by systolic dysfunction, myocardial restriction, or both. Conduction abnormalities, including CHB and EMD; ventricular arrhythmias, including sustained VT/VF and SCD. Tends to comigrate with other autoimmune diseases.	Treatments consists of multimodality therapy and should be implemented after a tissue diagnosis has been confirmed. Usual therapy includes a combination of a high-dose steroids, a calcineurin inhibitor (such as cyclosporine), and an antimetabolite such as azathioprine. Cytolytic therapy (purified rabbit-derived polyclonal IgG directed at human thymocytes) used for suppression of life-threatening GCM has been reported.
Acute NEM	Extensive inflammatory infiltration of the myocardium with mononuclear cells and eosinophils. Associated myonecrosis or fibrosis. On EM, may see eosinophil degranulation and deposition of major basic protein.	Acute heart failure/cardiogenic shock. May present with a restrictive cardiomyopathy. Prothrombotic intracardiac state. Peripheral eosinophilia may or may not be present. Recent viral infection or new medication.	Identify potential precipitant, especially if a drug hypersensitivity reaction. High-dose steroids. Anticoagulation. Often presents with ST-segment elevations and chest pain mimicking an ST-segment-elevation myocardial infarction. Rapid angiography, EMB with subsequent circulatory support, and initiation of high-dose intravenous corticosteroids can be lifesaving.
ICI myocarditis	Newly identified lymphocytic myocarditis resulting from the introduction of novel chemotherapeutic agents that unleash inhibited antitumor T cells, which also may infiltrate and attack the myocardium. Histopathology consistent with lymphocytic infiltrate and myocardial necrosis.	Acute heart failure, cardiogenic shock, and atrial fibrillation developing soon after ICI therapy is started and generally more severe with combination ICI therapy. Typically occurs early in treatment and has a fulminant course.	Treatment includes immediate cessation of therapy, high-dose corticosteroids (1 g solumedrol intravenously daily for 3 d and then 2 mg/kg prednisone daily to start, followed by a slow wean) and initiation of an angiotensin receptor blocker or sacubitril/valsartan. May initially need MCS.

CHB indicates complete heart block; EM, electron microscopy; EMB, endomyocardial biopsy; EMD, electromechanical dissociation; GCM, giant cell myocarditis; H&E, hematoxylin and eosin; ICI, immune checkpoint inhibitor; IgG, immunoglobulin G; MCS, mechanical circulatory support; MOSF, multiorgan system failure; NEM, necrotizing eosinophilic myocarditis; PCR, polymerase chain reaction; SCD, sudden cardiac death; and VT/VF, ventricular tachyarrhythmia/ventricular fibrillation.

Kociol RD, et al. Recognition and Initial Management of Fulminant Myocarditis: A Scientific Statement From the American Heart Association. *Circulation*. 2020;CIR0000000000000745.

Table 3. ESC 2025 GL on myocarditis and pericarditis.

Terminology	Definition
IMPS	Umbrella term for inflammatory myocardial and pericardial syndromes
Myopericarditis	Predominant pericarditis ^a
Perimyocarditis	Predominant myocarditis ^b
Acute myocarditis	Duration of symptoms ≤ 4 weeks Fulminant if: • Acute onset²⁸ and haemodynamically unstable patients requiring inotropes or mechanical circulatory support
Complicated myocarditis	AM and ≥ 1 of the following: ²⁸ • LVEF $< 50\%$ on echocardiogram • Sustained ventricular arrhythmias • Advanced heart block • Heart failure • Cardiogenic shock
Acute pericarditis	Duration of symptoms ≤ 4 weeks
Subacute/ongoing myocarditis	Duration of symptoms > 4 weeks to ≤ 3 months
Subacute/incessant pericarditis ^c	Duration of symptoms > 4 weeks to ≤ 3 months
Chronic myocarditis/pericarditis	Duration of symptoms > 3 months
Inflammatory cardiomyopathy	Chronic myocarditis in association with cardiac dysfunction and ventricular remodelling with clinical phenotype of hypokinetic, either dilated or non-dilated cardiomyopathy with/without arrhythmogenic substrate
Recurrent myocarditis/pericarditis	New symptoms or disease activity after clinical remission
Remission without residuals	Regression/absence of symptoms, normalization of ECG, biomarkers, imaging abnormalities (echocardiography and CMR)
Remission with residuals	Regression/absence of symptoms, persistence of abnormalities on ECG, biomarkers and/or imaging (functional and/or structural abnormalities in echocardiography or CMR)

AM, acute myocarditis; CK-MB, creatine kinase muscle-brain type; CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; IMPS, inflammatory myopericardial syndrome; LVEF, left ventricular ejection fraction.

^aPatients with definite criteria for pericarditis and elevated biomarkers of myocardial injury (high-sensitivity troponin I or T, CK-MB fraction) without newly developed regional or global impairment of left ventricular function in echocardiography or CMR.

^bPatients with definite criteria for pericarditis and elevated biomarkers of myocardial injury (high-sensitivity troponin I or T, CK-MB fraction) with newly developed regional or global impairment of left ventricular function in echocardiography or CMR.

^cIncessant/ongoing pericarditis: the term incessant pericarditis describes patients with persistent symptoms without a symptom-free interval of > 4 weeks despite full-dose guideline-directed medical therapy (including corticosteroids), or those relapsing early during the tapering.

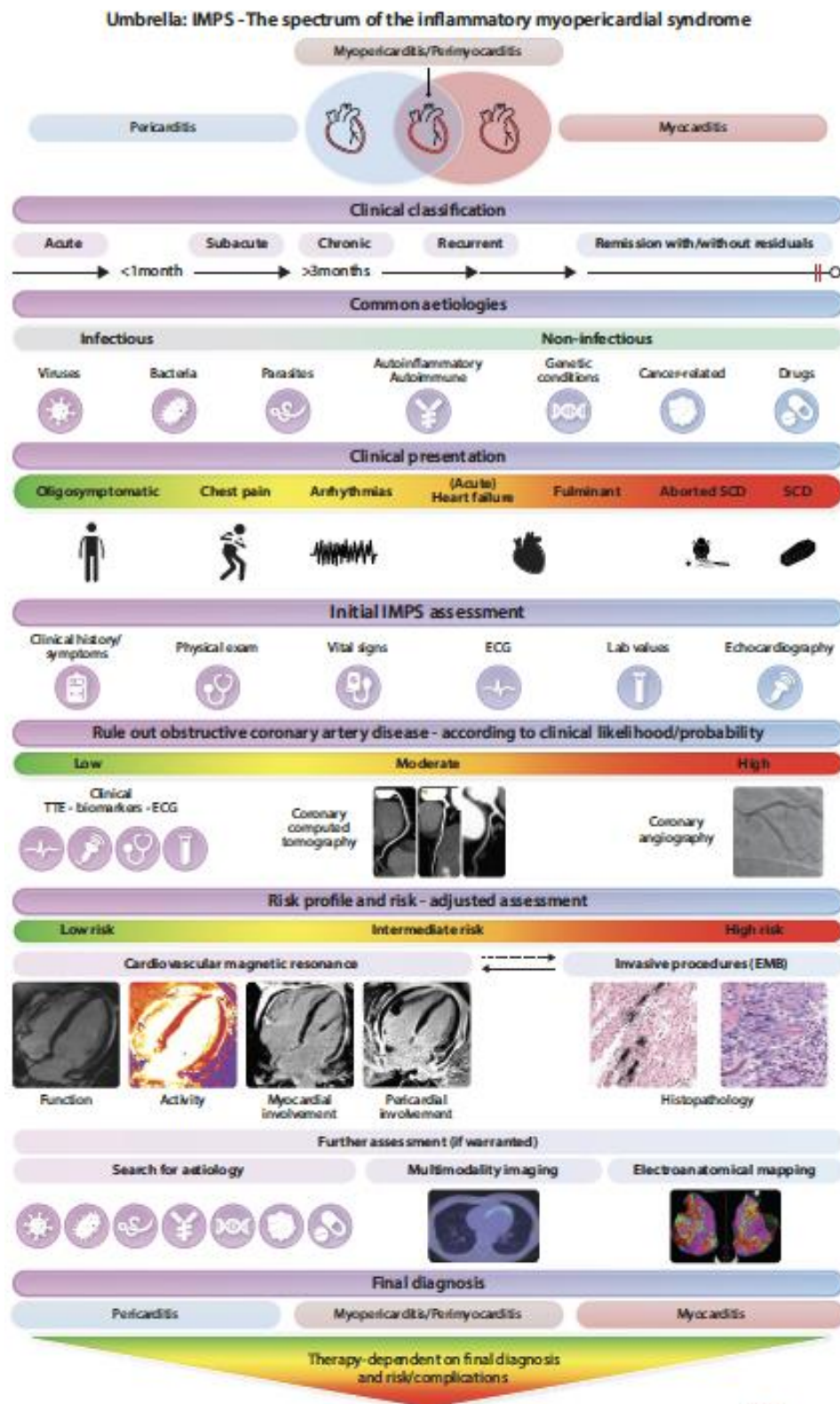


Figure 1. ESC 2025 GL on myocarditis and pericarditis. Central illustration of the ESC guidelines on myocarditis and pericarditis.

ECG, electrocardiogram; EMB, endomyocardial biopsy; IMPS, inflammatory myopericardial syndrome; SCD, sudden cardiac death; TTE, transthoracic echocardiography.

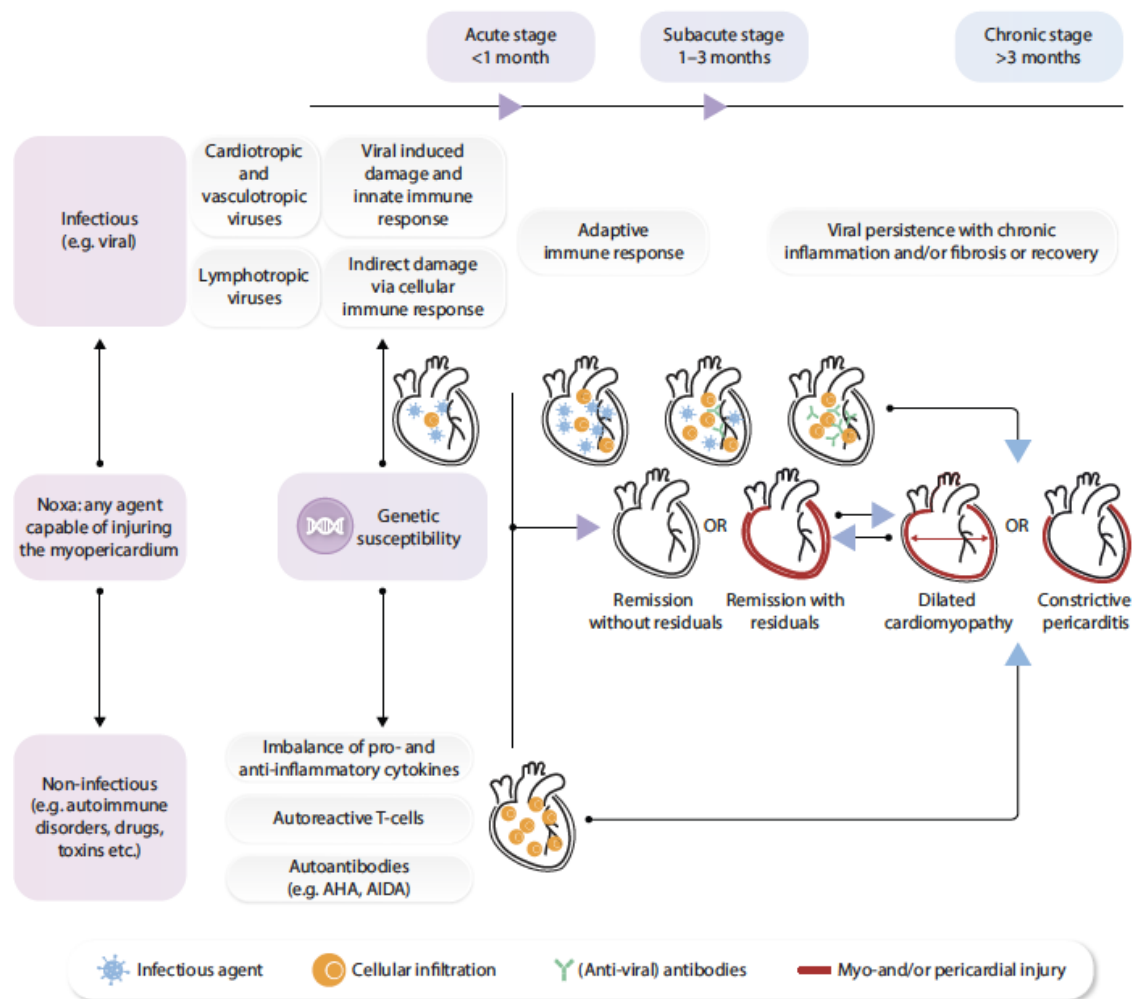


Figure 2. ESC 2025 GL on myocarditis and pericarditis. Stages of inflammatory myopericardial syndrome.

AHA, anti-heart antibodies; AIDA, anti-intercalated disc antibodies

Table 4. AHA/ACC 2021 GL on Chest Pain. Recommendations for Acute Chest Pain With Suspected Myopericarditis

In patients with acute chest pain and myocardial injury who have nonobstructive coronary arteries on anatomic testing, CMR with gadolinium contrast to distinguish myopericarditis from other causes, including myocardial infarction and nonobstructive coronary arteries (MINOCA).	I B-NR
In patients with acute chest pain with suspected acute myopericarditis, CMR if there is diagnostic uncertainty, or to determine the presence and extent of myocardial and pericardial inflammation and fibrosis.	I B-NR
In patients with acute chest pain and suspected myopericarditis, TTE to determine the presence of ventricular wall motion abnormalities, pericardial effusion, valvular abnormalities, or restrictive physiology.	I C-EO
In patients with acute chest pain with suspected acute pericarditis, noncontrast or contrast cardiac CT scanning to determine the presence and degree of pericardial thickening	IIb C-LD

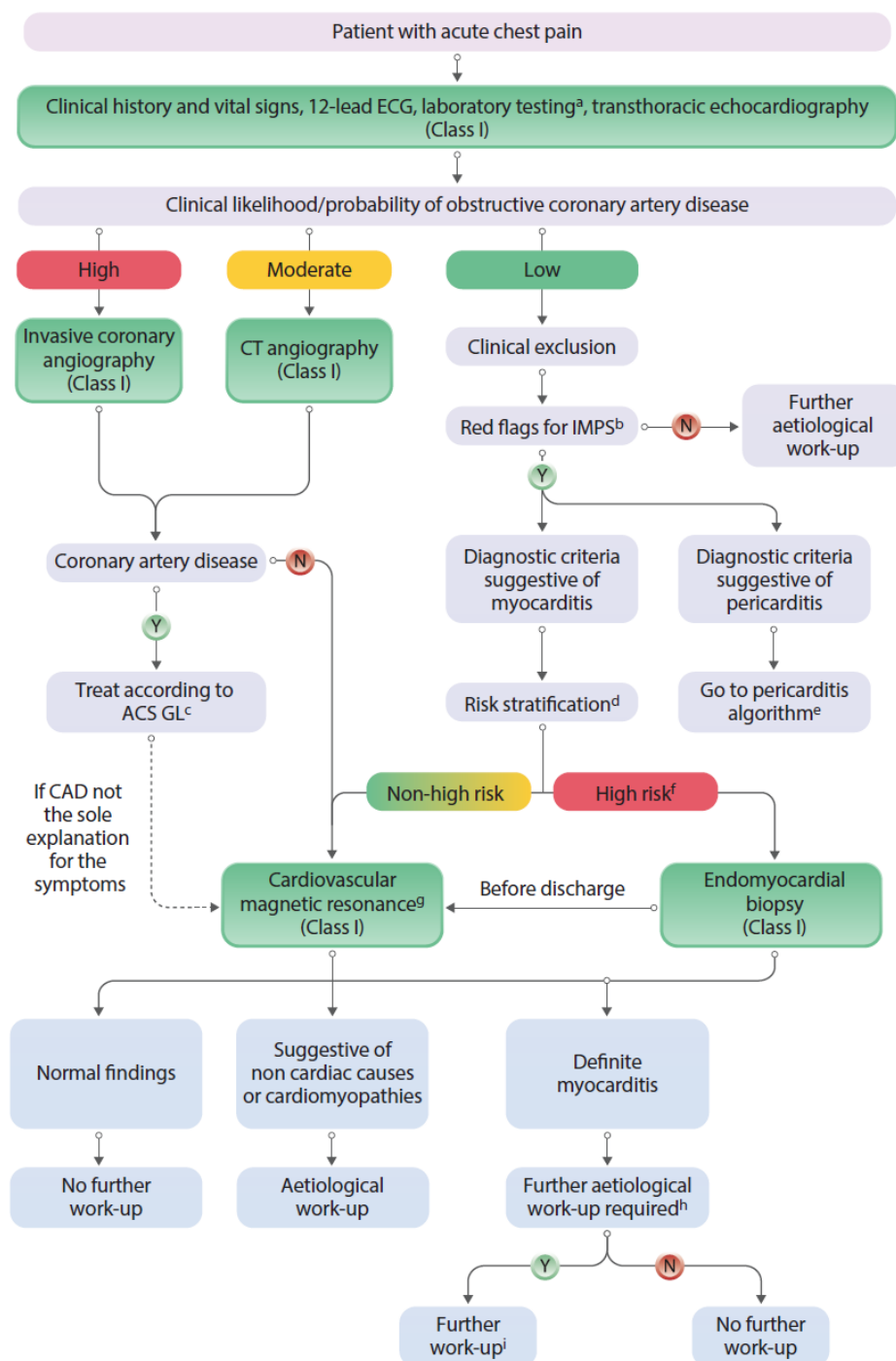


Figure 3. ESC 2025 GL on myocarditis and pericarditis Diagnostic algorithm for acute chest pain presentation.

ACS, acute coronary syndrome; CAD, coronary artery disease; CRP, C-reactive protein; CT, computed tomography; ECG, electrocardiogram; GL, guideline; IMPS, inflammatory myopericardial syndrome; N, no; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; Y, yes.

^aLaboratory testing including troponin, NT-proBNP, C-reactive protein, differential blood count.

^fConsider cardiovascular magnetic resonance. ^gConsider endomyocardial biopsy on a case-by-case basis. ^hDepending

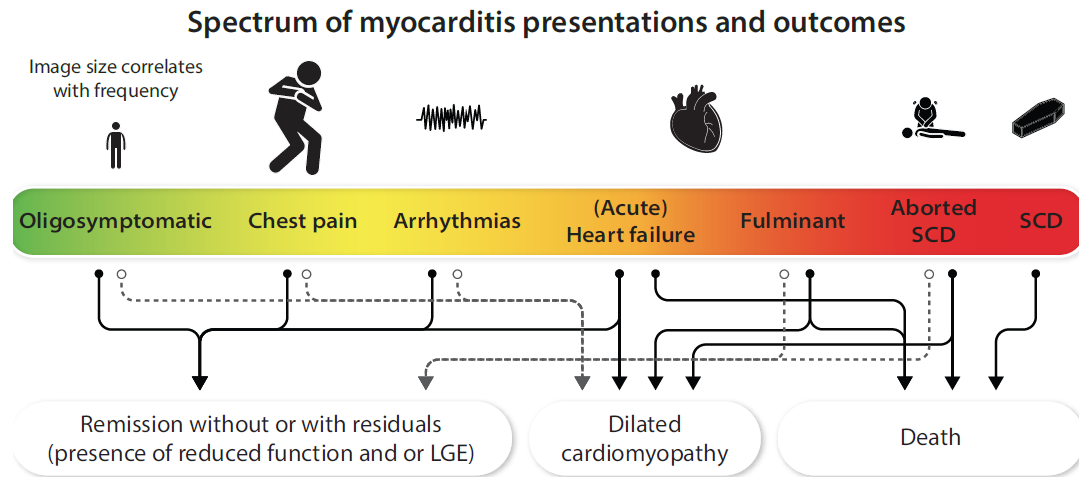


Figure 4. ESC 2025 GL on myocarditis and pericarditis. Spectrum of myocarditis presentations and outcomes.

LGE, late gadolinium enhancement; SCD, sudden cardiac death. Image size correlates with presentation frequency (e.g. chest pain being the most common). Uncomplicated cases of myocarditis (about 75% of unselected cases) usually have spontaneous remission. In acute myocarditis diagnosed by endomyocardial biopsy, up to 50% of patients have spontaneous recovery, but up to 25% may have a persistent stable cardiac dysfunction, while 10%–25% have a progressive ventricular dysfunction, which could lead to end-stage dilated cardiomyopathy, heart transplantation, or death.

Table 5. ESC 2025 GL on myocarditis and pericarditis.**Diagnostic criteria and classification for inflammatory myopericardial syndrome**

IMPS		
If diagnostic criteria for myocarditis and/or pericarditis are fulfilled ^a		
	Myocarditis	Pericarditis
Definite	Clinical presentation ^b and CMR- or EMB-proven	Clinical presentation ^b with >1 additional criterion
Possible	Clinical presentation ^b with at least 1 additional criterion CMR- or EMB-uncertain or not available	Clinical presentation ^b with 1 additional criterion
Unlikely/rejected	Only clinical presentation ^b without additional criteria	Only clinical presentation ^b without additional criteria
Additional criteria beyond clinical presentations ^b		
	Myocarditis	Pericarditis
Clinical ^b	Non-specific findings	Pericardial rubs
ECG ^c	ST-T changes	PR depression, widespread ST-segment elevation
Biomarkers	Troponin elevation	C-reactive protein elevation
Imaging ^d	Abnormal strain, wall motion, reduced EF Myocardial oedema and/or LGE (CMR findings)	New or worsening pericardial effusion Pericardial oedema and/or LGE (CMR findings)

CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; EF, ejection fraction; EMB, endomyocardial biopsy; IMPS, inflammatory myopericardial syndrome; LGE, late gadolinium enhancement; LLC, Lake Louise criteria.

Clinical presentations include chest pain or infarct-like symptoms, arrhythmias, heart failure, aborted sudden cardiac death.

Cardiovascular magnetic resonance categories: proven = 2 out of 2 updated LLC fulfilled; uncertain = only 1 out of 2 updated LLC fulfilled; rejected = negative CMR.

EMB categories: proven, uncertain, rejected (according to pathologist consensus).

aOne condition may be leading.

b rarely asymptomatic cases may be detected (e.g. drug-related with a distinct history, such as immune checkpoint inhibitors).

cIf ECG changes always consider/exclude myocarditis.

Histopathological criteria for myocarditis

Term	Predominant inflammatory cells	Myocyte necrosis	Infections PCR positive (viruses, etc.)
Active lymphocytic myocarditis	CD3 ⁺ T lymphocytes >7/mm ² , CD68 ⁺ macrophages	yes	yes/no
Persistent lymphocytic myocarditis	CD3 ⁺ T lymphocytes >7/mm ² , CD68 ⁺ macrophages	yes	yes/no
Resolved lymphocytic myocarditis	–	no	yes/no
Eosinophilic myocarditis (acute stage)	Eosinophils, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes	yes/no
Giant-cell myocarditis (acute stage)	Eosinophils, CD68 ⁺ giant cells, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes	no
Sarcoidosis	CD68 ⁺ giant cells, granuloma, CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages	yes/no	no

PCR, polymerase chain reaction.

Red flags for the clinical diagnosis of myocarditis and pericarditis**Myocarditis**

Recent or concomitant flu-like syndrome or gastroenteritis
Infarct-like chest pain
Palpitations
HF symptoms
ECG changes^a
Ventricular arrhythmias (isolated, complex)
Syncope
Haemodynamic instability
Elevated markers of myocardial lesion (hs-Tn, CK-MB elevation)
Elevated markers of HF (NT-proBNP)
Abnormal wall motion, increased wall thickness and/or impaired systolic function on imaging
CMR imaging with myocardial oedema and/or LGE

CK-MB, creatinine kinase muscle-brain type; CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; HF, heart failure; hs-Tn; high-sensitivity troponin; LGE, late gadolinium enhancement; NT-proBNP, N-terminal prohormone of brain natriuretic peptide.

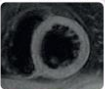
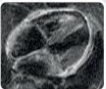
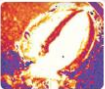
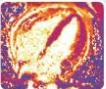
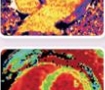
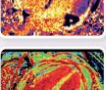
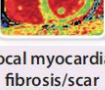
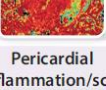
Criterion	Methods	Example images and pathology		Parameters for reporting	
				For myocarditis	For pericardial involvement
T2-based criterion	T2-weighted imaging or T2 mapping	Myocardial oedema 	Pericardial oedema 	• Presence, extent, and location of oedema (T2 weighted) • Regional high T2 SI or global high T2 SI (T2-weighted) • Regional or global increase of myocardial T2 times	• High signal intensity of the pericardium in T2-mapping or T2-weighted imaging
		Myocardial oedema/diffuse fibrosis 	Pericardial oedema/diffuse fibrosis 	• Description of focal increases • Regional or global increase of native myocardial T1 times • Regional or global increase ECV values	• High signal intensity of the pericardium in T1-mapping
T1-based criterion	Native T1 mapping/post-contrast T1 mapping (ECV)/T1-weighted imaging	Myocardial oedema/diffuse fibrosis 	Pericardial oedema/diffuse fibrosis 	• Presence, pattern, extent, and location of LGE (positive if areas with high SI in a nonischaemic distribution pattern) • Thrombi (if present) • Total LGE/LV mass (%) (no routine)	• High signal intensity of the pericardium in LGE images
	Late gadolinium enhancement	Focal myocardial fibrosis/scar 	Pericardial inflammation/scar 	• Regional wall-motion abnormalities • Cardiac function (e.g. LVEF, RVEF) and volume parameters	• Presence, composition, and extent of pericardial effusion • Haemodynamic relevance of pericardial effusion • Diameter of pericardial effusion
Supportive criterion	Cine imaging	Functional and wall motion abnormalities 	Haemodynamic compromise 		
Updated Lake Louise Criteria (LLC) for myocarditis					
CMR-proven myocarditis= 2 out of 2 updated LLC main criteria fulfilled		T2-based criterion Myocardial oedema	Abnormal T2-mapping or T2-weighted imaging	Pericardial abnormalities	
		Main criteria		Supportive criteria	
CMR-uncertain myocarditis= only 1 out of 2 updated LLC main criteria fulfilled		T1-based criterion Non-ischaemic myocardial injury	Abnormal T1-mapping, ECV or LGE	Systolic LV-dysfunction	

Figure 5. ESC 2025 GL on myocarditis and pericarditis.

Diagnostic criteria by cardiovascular magnetic resonance based on the updated Lake Louise criteria.

CMR, cardiovascular magnetic resonance;

ECV, extracellular volume; LGE, late gadolinium enhancement; LLC, Lake Louise criteria; LV, left ventricle; LVEF, left ventricular ejection fraction;

RVEF, right ventricular ejection fraction; SI, signal intensity ratio.³⁰ Presence of pericardial effusion suggests concomitant pericarditis.

Table 6. ESC 2025 GL on myocarditis and pericarditis. Clinical risk stratification to guide work-up in inflammatory myopericardial syndrome

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

AV, atrioventricular; CMR, cardiovascular magnetic resonance; HF, heart failure; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction; NSAID, non-steroidal anti-inflammatory drug; NYHA, New York Heart Association classification; WMA, wall motion abnormalities.

^aThese criteria do not lead directly towards endomyocardial biopsy; in these scenarios it is a case-by-case decision depending on the suspected underlying cause.

Table 7. ESC 2025 GL on myocarditis and pericarditis.**Recommendations for clinical evaluation of myocarditis and pericarditis**

Complete clinical evaluation, including history, physical examination, chest X-ray, biomarkers ^c , ECG, and echocardiography is recommended in all patients with a suspicion of myocarditis and/or pericarditis for the initial diagnostic assessment.	I	C
CMR is recommended in patients with the clinical suspicion of myocarditis (using updated LL criteria) ³⁰ and/or pericarditis for the non-invasive diagnosis of inflammatory reaction. ^{110,115,129}	I	B
Hospital admission is recommended for patients with high-risk pericarditis ^d for monitoring and treatment. ^{105,130}	I	B
Hospital admission is recommended for patients with moderate- to high-risk myocarditis ^d for monitoring and treatment.	I	C
EMB ^e is recommended in patients with high-risk myocarditis ^d and/or haemodynamic instability, and/or in patients with intermediate-risk myocarditis not responding to conventional therapy in order to detect a specific histologic subtype and to assess the presence of viral genome for treatment. ^{34,63,73,131}	I	C
Invasive coronary angiography or coronary CT, depending on clinical likelihood, is recommended in patients with IMPS if an acute coronary syndrome is suspected to rule out obstructive coronary artery disease.	I	C
Hospital admission should be considered for patients with low-risk myocarditis ^d for monitoring and treatment.	IIa	C
Pericardial or epicardial biopsy may be considered in relapsing pericardial effusion as part of the diagnostic work-up when the diagnosis cannot be reached with multimodality imaging and laboratory examinations.	IIb	C
Routine serology is not recommended in patients with myocarditis and/or pericarditis for the evaluation of viral aetiology except for hepatitis C, HIV, and Lyme disease.	III	C

CMR, cardiovascular magnetic resonance; CT, computed tomography; ECG, electrocardiogram; EMB, endomyocardial biopsy; HIV, human immunodeficiency virus; hs-TnI, high-sensitivity troponin I; hs-TnT, high-sensitivity troponin T; IMPS, inflammatory myopericardial syndrome; LL, Lake Louise; NT-proBNP, N-terminal prohormone of brain natriuretic peptide.

^cIncluding hs-TnT or hs-TnI, C-reactive protein, NT-proBNP.

^eContemporary evaluation of EMB should be based on histology, immunohistology, and molecular pathology for detection of viral infections in myocardial and blood samples

Basic and advanced level assessment

Basic assessment:
History: potential causes and triggers (viral infection of upper respiratory or gastrointestinal tract, toxins, drug use, medications), recurrent symptoms, family history of IMPS/cardiomyopathy/SCD, and systemic inflammatory/autoimmune diseases
Physical examination: assess clinical stability, symptoms (chest pain, HF symptoms, palpitations, syncope), malaise, general weakness and fatigue, pericardial friction rub, clinical symptoms/signs of CTP
ECG: PR-segment depression, ST/T-wave changes, AVB, and ventricular arrhythmias
Chest X-ray
Basic laboratory data: Markers of myocardial lesion (e.g. hs-TnT/TnI) Markers of systemic inflammation (e.g. C-reactive protein, ESR, WBC count) Markers of heart failure (e.g. NT-proBNP) Complete blood count (including eosinophilic count) Renal function and electrolytes (e.g. sodium, potassium, creatinine) Thyroid function (e.g. TSH) Hepatic function and additional testing (e.g. lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase)
Echocardiography including strain imaging
Advanced assessment usually after admission:
Coronary anatomy evaluation (if needed for differential diagnosis by invasive coronary angiography or coronary CT depending on the clinical likelihood of ACS)
CMR to assess signs of myocardial and pericardial inflammation and/or fibrosis
Arrhythmia screening depending on risk stratification (e.g. Holter-ECG)
Additional laboratory parameters guided by clinical suspicion (e.g. if therapeutic consequences are expected)
Dedicated genetic testing if indicated
CT to assess concomitant pleuropulmonary diseases
Specific myocarditis: EMB in high-risk cases and in intermediate-risk cases on a case-by-case decision to detect specific histology and some aetiologies if needed
Specific pericarditis: diagnostic pericardiocentesis when indicated ^a

ACS, acute coronary syndrome; AVB, atrioventricular block; CMR, cardiovascular magnetic resonance; CT, computed tomography; CTP, cardiac tamponade; ECG, electrocardiogram; EMB, endomyocardial biopsy; ESR, erythrocyte sedimentation rate; HF, heart failure; hs-TnT/TnI, high-sensitivity troponin T/I; IMPS, inflammatory myopericardial syndrome; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; SCD, sudden cardiac death; TSH, thyroid-stimulating hormone; WBC, white blood cell.

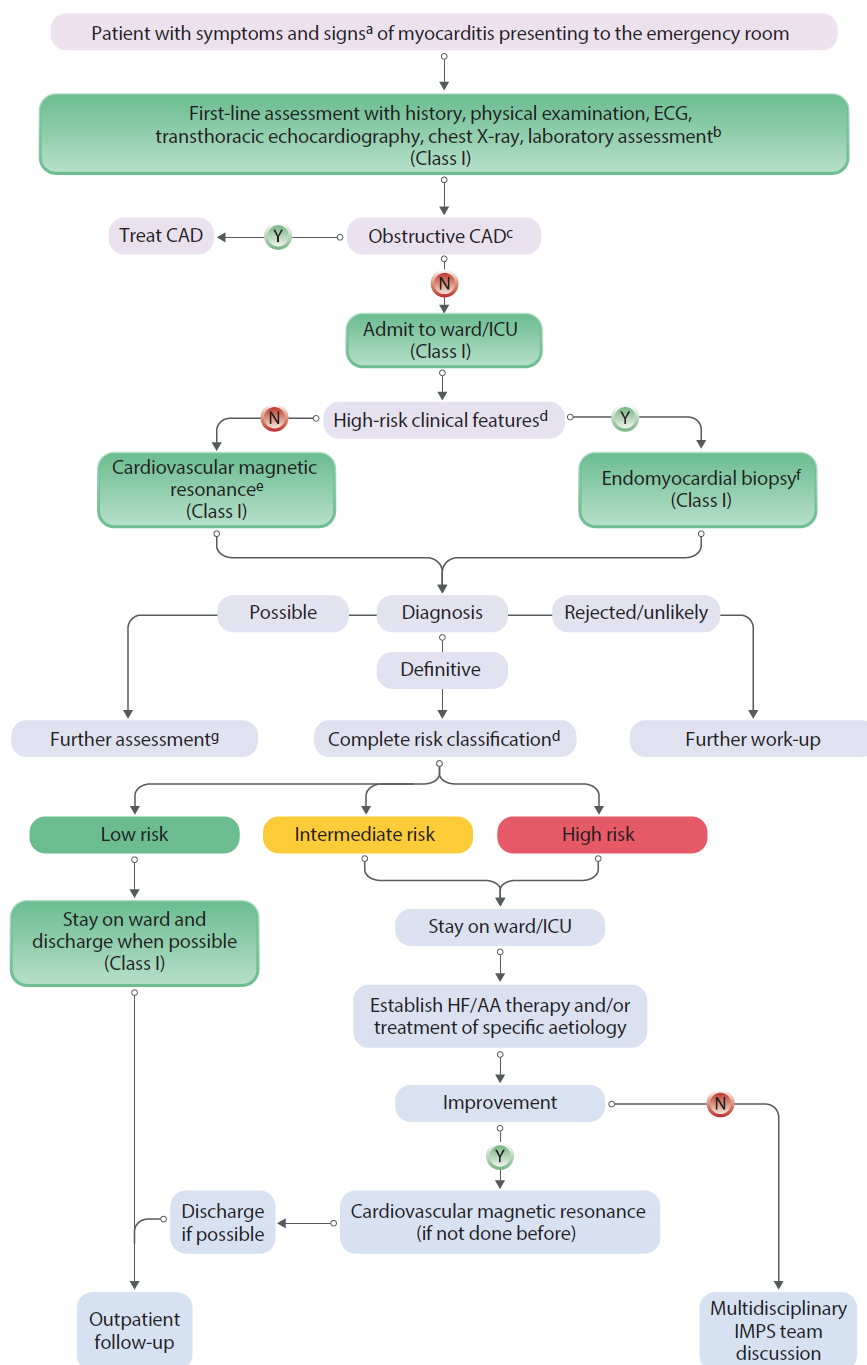


Figure 6. ESC 2025 GL on myocarditis and pericarditis. Diagnostic algorithm and triage for inpatient myocarditis.

AA, anti-arrhythmic therapy; CAD, coronary artery disease; CRP, C-reactive protein; ECG, electrocardiogram; HF, heart failure; ICU, intensive care unit; IMPS, inflammatory myopericardial syndrome; N, no; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; Y, yes.

^bLaboratory testing including troponin, NT-proBNP, C-reactive protein, differential blood count.
^cInvasive coronary angiography or computed tomography angiography depending on clinical likelihood/probability.

^eConsider endomyocardial biopsy. ^fConsider cardiovascular magnetic resonance.

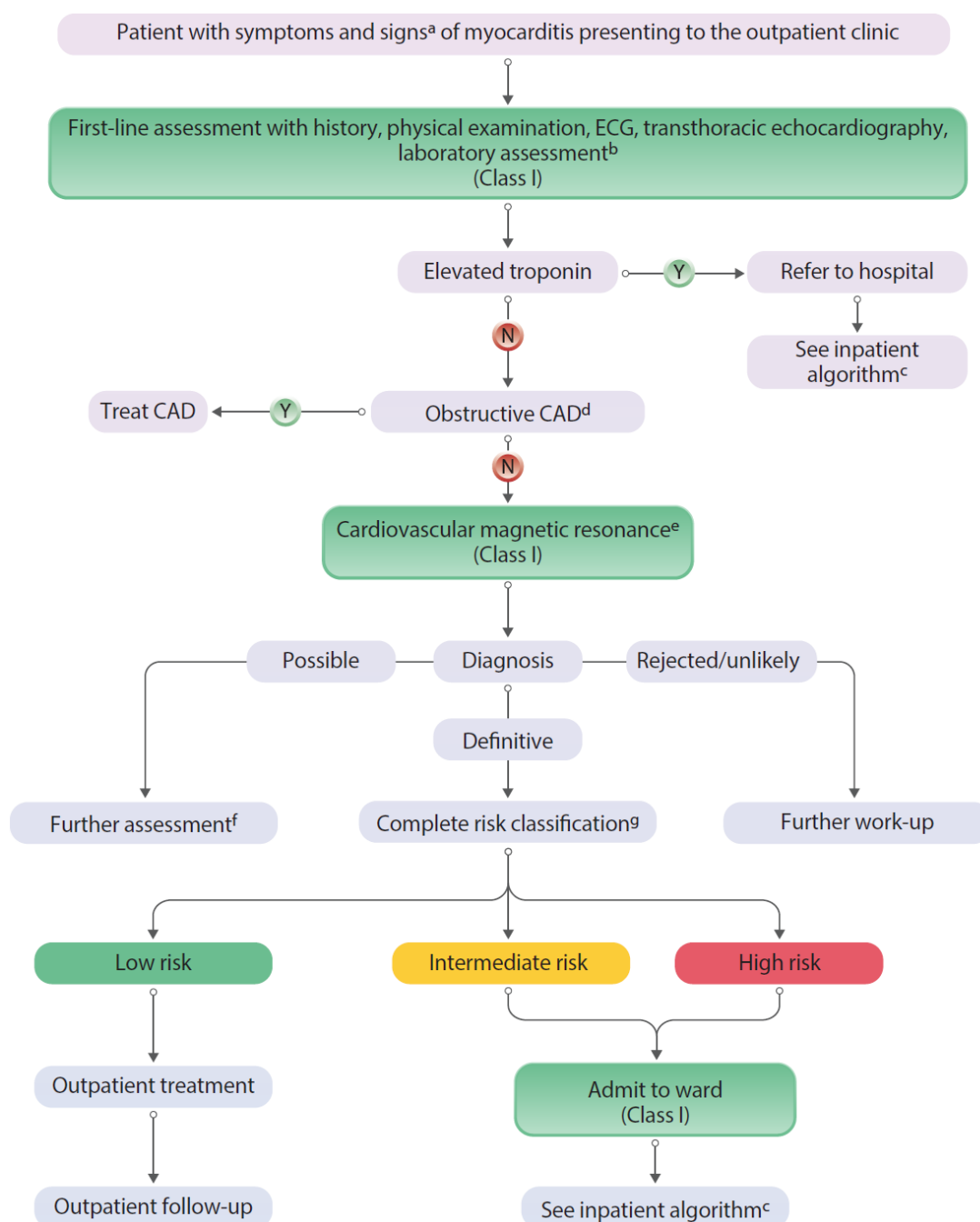
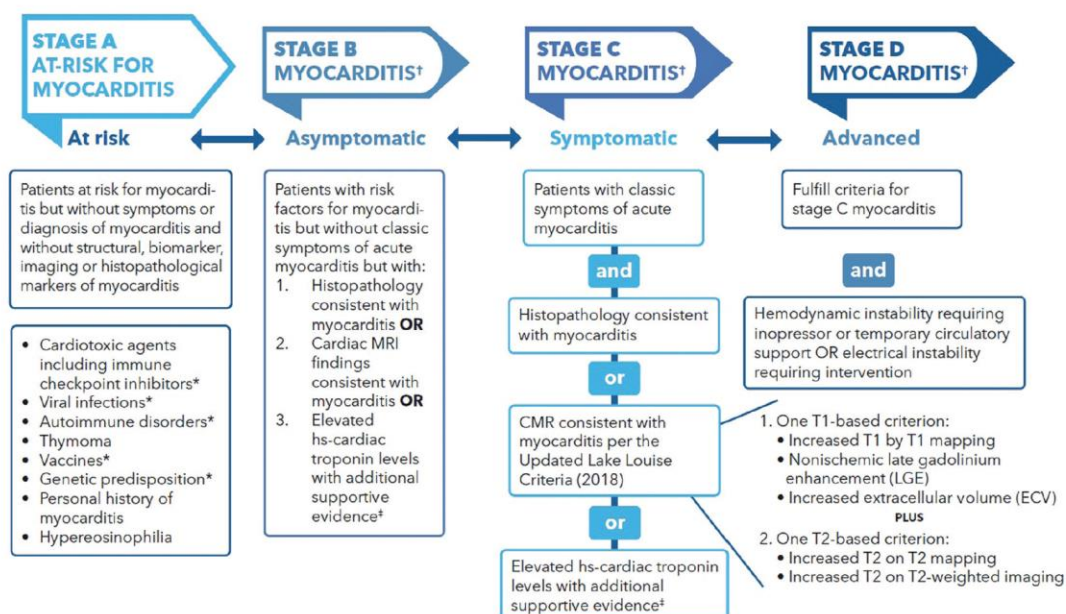


Figure 7. ESC 2025 GL on myocarditis and pericarditis. Diagnostic algorithm and triage for outpatient myocarditis.

CAD, coronary artery disease; CRP, C-reactive protein; ECG, electrocardiogram; N, no; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; Y, yes. ^bLaboratory testing including troponin, NT-proBNP, C-reactive protein, differential blood count. ^dInvasive coronary angiography or computed tomography angiography depending on clinical likelihood/probability. ^eDepending on clinical situation (heart failure, arrhythmias) admit to ward.



The spectrum of myocarditis is described as 4 stages: A (at-risk); B (asymptomatic); C (symptomatic myocarditis); and D (advanced myocarditis). There are 2 pivotal tests to diagnose stages B-D myocarditis: EMB and CMR. EMB, including histopathology, immunohistochemistry, and molecular search for infectious agents, is the gold-standard diagnostic test for myocarditis, allowing characterization of histotype and specific etiologies (including viral). EMB is associated with some risks due to its invasive nature and limitations due to sampling error. CMR is an attractive noninvasive strategy although it too has limitations, including reduced sensitivity depending upon clinical presentation, a delayed timing after onset of symptoms, and technical challenges due to patient breath-holding and irregular heart rhythms. The specificity of CMR diagnosis of myocarditis is enhanced when both T₁ and T₂ criteria are met, although isolated T₁ or T₂ abnormalities may be seen at times. In the appropriate clinical context (eg, therapy with an immune checkpoint inhibitor), an acute rise in troponin can be consistent with the diagnosis of myocarditis but the specificity of an elevated troponin with most other stage A risk factors is not adequate to make the diagnosis and one of the two pivotal tests would be needed. Further, myocardial infarction needs to be excluded (eg, by coronary angiography or absence of ischemic LGE on CMR) as the basis of an elevated troponin level in most contexts when considering the diagnosis of myocarditis. Unlike the staging system in HFrEF, patients with myocarditis can move from higher to lower stages*Those known to be associated with myocarditis. †Pericarditis may complicate stages B-D ("myopericarditis"). ‡The level of supportive evidence depends upon the clinical context, including which stage A risk factor is present. CMR = cardiac magnetic resonance; EMB = endomyocardial biopsy; HFrEF = heart failure with reduced ejection fraction; hs = high sensitivity; LGE = late gadolinium enhancement; MRI = magnetic resonance imaging.

Figure 8. ACC 2024 Decision Pathway on the Diagnosis and Management of Myocarditis. Proposed Stages of Myocarditis

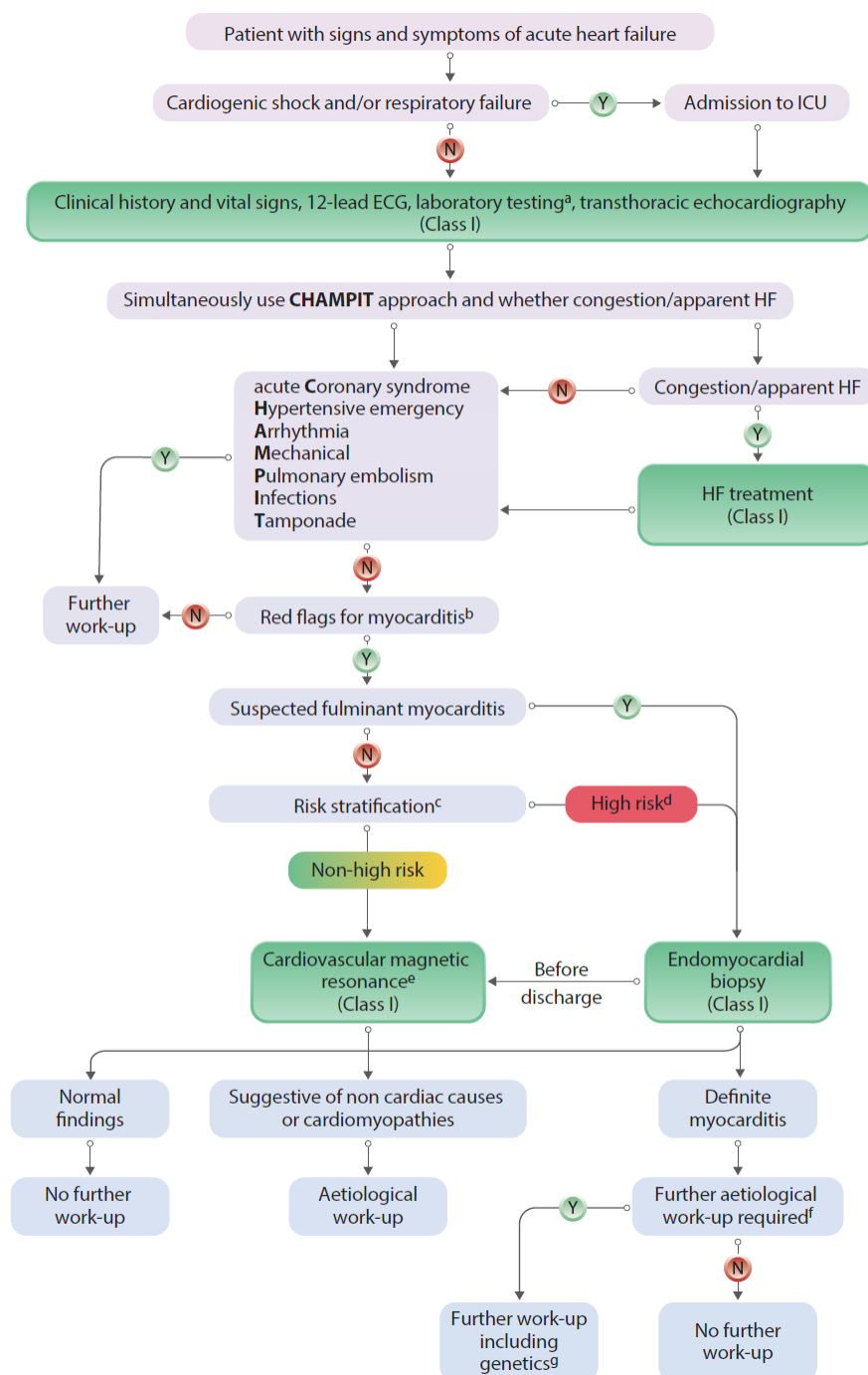


Figure 9. ESC 2025 GL on myocarditis and pericarditis. Diagnostic algorithm for acute heart failure presentation.

CRP, C-reactive protein; ECG, electrocardiogram; HF, heart failure; ICU, intensive care unit; N, no; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; Y, yes.

^aLaboratory testing including troponin, NT-proBNP, C-reactive protein, differential blood count.

^dConsider cardiovascular magnetic resonance.

^eIn stabilized heart failure patients, endomyocardial biopsy may be performed on a case-by-case basis.

^fDepending on the underlying cause and clinical need.

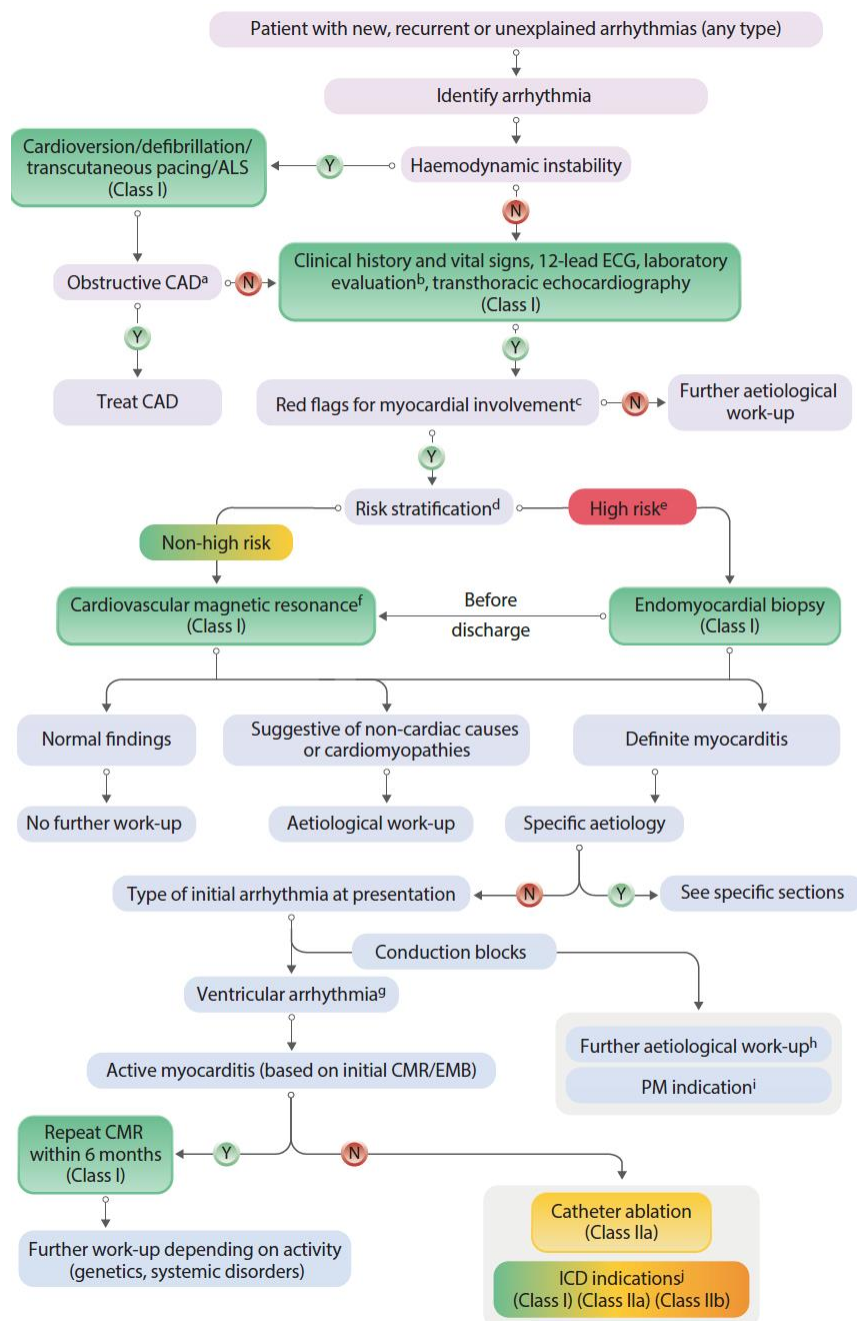


Figure 10. ESC 2025 GL on myocarditis and pericarditis. Diagnostic algorithm for arrhythmia presentation.

ALS, advanced life support; CAD, coronary artery disease; CMR, cardiovascular magnetic resonance; CRP, C-reactive protein; ECG, electrocardiogram; EMB, endomyocardial biopsy; ICD, implantable cardioverter-defibrillator; N, no; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; PM, pacemaker; Y, yes.

^aInvasive coronary angiography or computed tomography angiography depending on clinical likelihood/probability.

^bLaboratory testing including troponin, NT-proBNP, C-reactive protein, differential blood count.

^eConsider cardiovascular magnetic resonance.

^fConsider endomyocardial biopsy on a case-by-case basis.

Table 8. ESC 2025 GL on myocarditis and pericarditis. Recommendations for the use of cardiovascular magnetic resonance imaging

CMR is recommended in patients with suspected myocarditis to reach a clinical diagnosis and to determine the cause of acute myocardial injury, including assessment of oedema, ischaemia, and necrosis/fibrosis/scarring. ^{115,164,169–183}	I	B
CMR is recommended for follow-up at least within the first 6 months in patients with myocarditis to identify a healed or ongoing process, for risk stratification and personalized therapy, and to enable a return to exercise. ^{10,62,184–186}	I	C

Table 9. ESC 2025 GL on myocarditis and pericarditis. Recommendations for endomyocardial biopsy

EMB ^c is recommended in patients with high-risk myocarditis ^d , and/or haemodynamic instability, and/or in patients with intermediate-risk myocarditis not responding to conventional therapy in order to detect a specific histologic subtype and to assess the presence of viral genome for treatment. ^{34,63,73,131}	I	C
--	----------	----------

EMB, endomyocardial biopsy.

cContemporary evaluation of EMB should be based on histology, immunohistology, and molecular pathology for detection of viral infections in myocardial and blood samples

Table 10. ESC 2025 GL on myocarditis and pericarditis. Parameters for reporting by endomyocardial biopsy

Criteria	Parameters for reporting
Histology (paraffin-embedded EMB, at least 3 EMB)	Presence and extent of cardiomyocyte necrosis, inflammation, fibrosis
Immunohistology (paraffin-embedded EMB, at least 3 myocardial samples)	Presence, extent, localization, and typing of immune cells in the myocardium: CD3 ⁺ T lymphocytes, CD68 ⁺ macrophages (≥ 14 leucocytes/mm ² with T lymphocytes ≥ 7 cells/mm), ¹⁰ HLA-DR expression in immune cells and endothelial cells
Molecular pathology [infections: RNA later, snap-frozen tissue (1–2 EMB), paraffin-embedded EMB]	Presence, typing, and quantification of DNA/RNA from infectious agents by q(RT)-PCR Viruses: mainly enteroviruses, parvovirus B19, human herpesvirus 6, Epstein–Barr virus; <i>Borrelia</i> spp., <i>Trypanosoma cruzi</i> ^a
Blood	q(RT)-PCR for detection of systemic infections
Molecular pathology (genetics: RNA later, frozen tissue, paraffin-embedded EMB, blood)	NGS for detection of pathogenic variants in cardiac genes, traditionally associated with cardiomyopathies (especially desmosomal and sarcomeric/cytoskeletal genes)

DNA, deoxyribonucleic acid; EMB, endomyocardial biopsy; HLA-DR, human leucocyte antigen—DR isotype; NGS, next-generation sequencing; q(RT)-PCR, quantitative (real-time) polymerase chain reaction; RNA, ribonucleic acid.

Table 11. ESC 2025 GL on myocarditis and pericarditis. Recommendations for electro-anatomical mapping

Electro-anatomical mapping should be considered in cases of suspected myocarditis (especially cardiac sarcoidosis) to guide endomyocardial biopsy. ^{218,220,221,253,256–258}	IIa	C
---	------------	----------

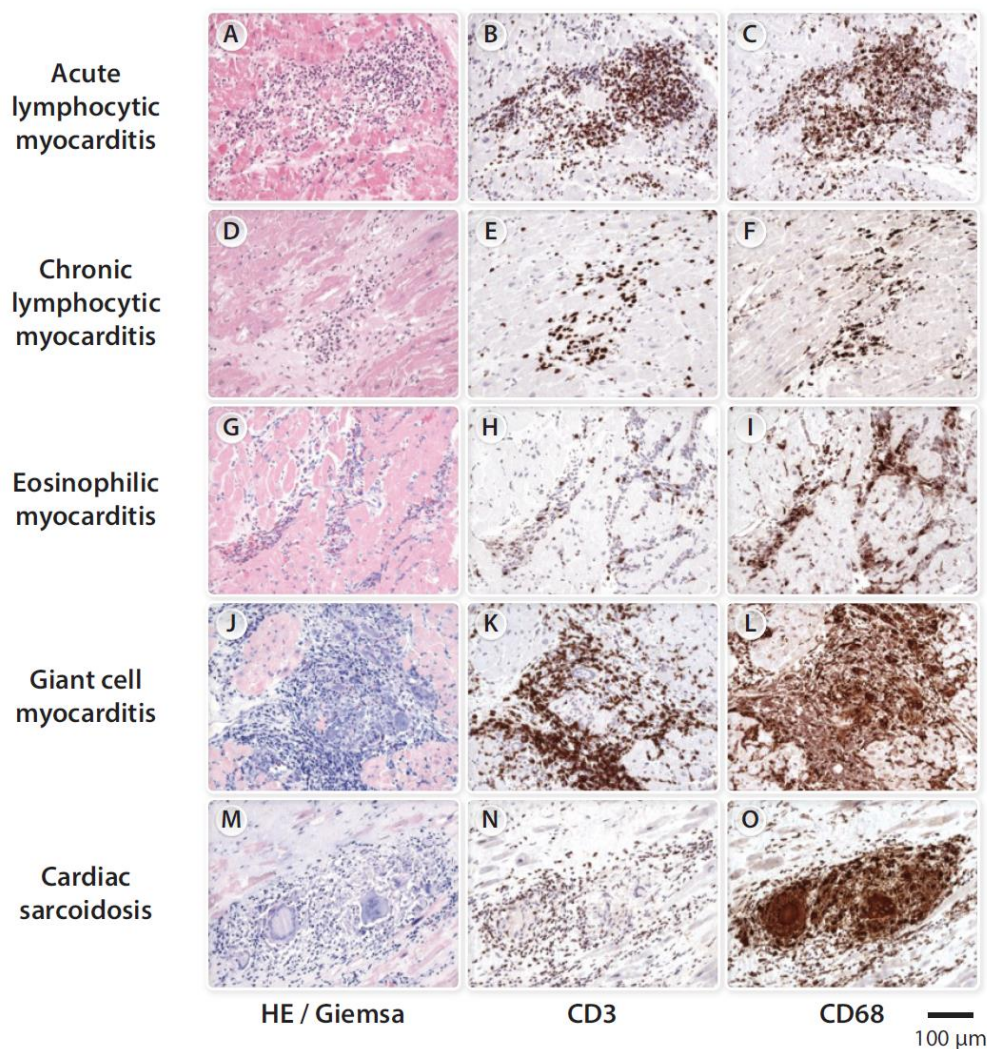


Figure 11. ESC 2025 GL on myocarditis and pericarditis. Histopathological findings in myocarditis.

HE, haematoxylin eosin. (A–C) Endomyocardial biopsy for acute lymphocytic myocarditis reveals cardiomyocyte necrosis (A, HE stain) and severe focal infiltration of CD3+ T cells (B) and CD68+ macrophages (C), as detected by immunohistochemistry.

(D–F) In chronic lymphocytic myocarditis, no cardiomyocyte injury is observed, but there is focal replacement fibrosis (D, HE) in the presence of CD3+ T cells (E) and CD68+ macrophages (F).

(G–I) In acute eosinophilic myocarditis, necrosis of cardiomyocytes is associated with numerous eosinophilic granulocytes (G, Giemsa stain), some CD3+ T cells (H), and numerous CD68+ macrophages (I).

(J–L) Areas with severe cardiomyocyte necrosis in the presence of extensive focal and diffuse infiltration of eosinophilic granulocytes (J, Giemsa stain), CD3+ T cells (K), and CD68+ macrophages and CD68+ giant cells (L) are typical of giant-cell myocarditis.

(M–O) Cardiac sarcoidosis is histologically characterized by the presence of granulomas, usually in the absence of cardiomyocyte necrosis (M, Giemsa stain), but with focal infiltration of CD3+ T cells (N) and numerous CD68+ macrophages and CD68+ giant cells (O).

Table 12. ESC 2025 GL on myocarditis and pericarditis. Recommendations for nuclear Medicine

Carb-free ¹⁸ F-FDG-PET or ¹⁸ F-FDG-PET/CT should be considered for the diagnostic work-up in patients with suspected myocarditis and/or pericarditis in whom echocardiography and CMR are inconclusive for the clinical diagnosis. ^{201,206}	IIa	C
---	------------	----------

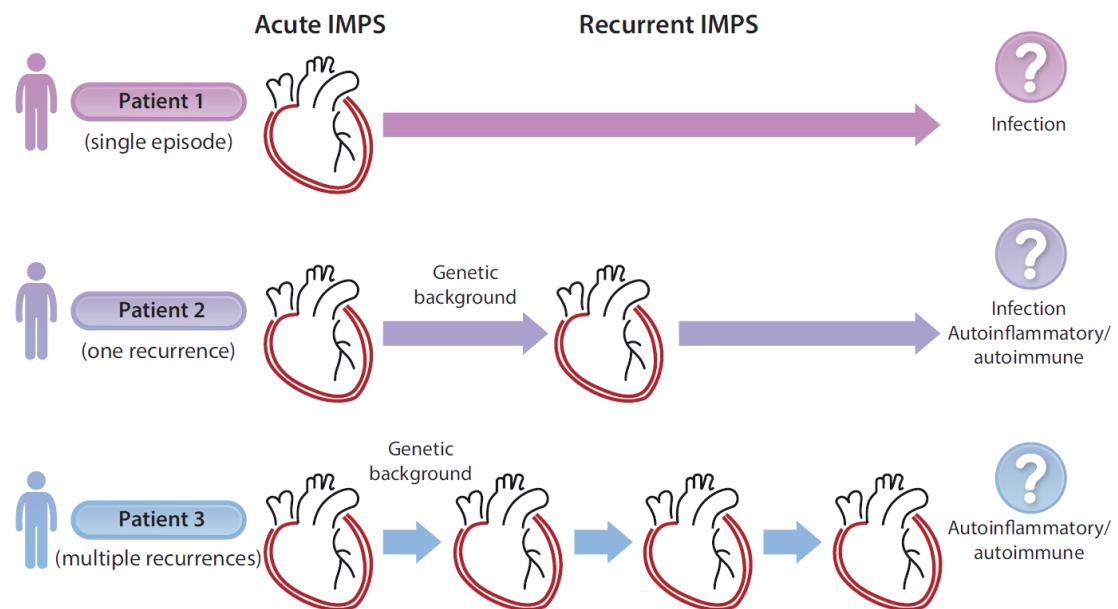
CMR, cardiovascular magnetic resonance; CT, computed tomography; 18F-FDG-PET, [18F]-fluorodeoxyglucose positron emission tomography.

Table 13. ESC 2025 GL on myocarditis and pericarditis. Recommendations for genetic testing

It is recommended to obtain family history including pedigrees in cases of recurrent IMPS to provide clues to the underlying aetiology, determine inheritance pattern, and identify relatives at risk.	I	C
Genetic testing should be considered in patients with definite myocarditis/pericarditis in cases of: ^{50,51,64,94,150} • family history of IMPS, inherited or suspected cardiomyopathy • severe ventricular arrhythmia^c • significant left/right LGE (e.g. ring-like pattern or septal LGE) or persistent LVEF systolic dysfunction • recurrent myocarditis or persistent troponin elevation • recurrent pericarditis with an inflammatory phenotype, refractory to conventional treatment^d, with the aim to detect an underlying genetic cause.	IIa	B

IMPS, inflammatory myopericardial syndrome; IL, interleukin; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction.

^dEspecially poor response to colchicine and anti-IL-1 agents.



IMPS, inflammatory myopericardial syndrome.

Figure 12. ESC 2025 GL on myocarditis and pericarditis. The different courses of inflammatory myopericardial syndrome and the interplay between genetic background inflammation and autoimmunity beyond the initial infectious trigger.

Table 14. ESC 2025 GL on myocarditis and pericarditis. Recommendations for autopsy

Comprehensive autopsy is recommended in all patients <50 years of age with SCD to evaluate the presence of acute myocarditis as a cause and to detect potential underlying inherited cardiac diseases. ^{87,243–246c}	I	B
Retaining samples suitable for DNA extraction and consulting a cardiac pathologist is recommended in cases of SCD, when an inherited cause is suspected, or the cause of death remains unexplained. ^{243,247c}	I	B

DNA, deoxyribonucleic acid; SCD, sudden cardiac death.

Table 15. ESC 2025 GL on myocarditis and pericarditis. Medical therapy in myocarditis

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.

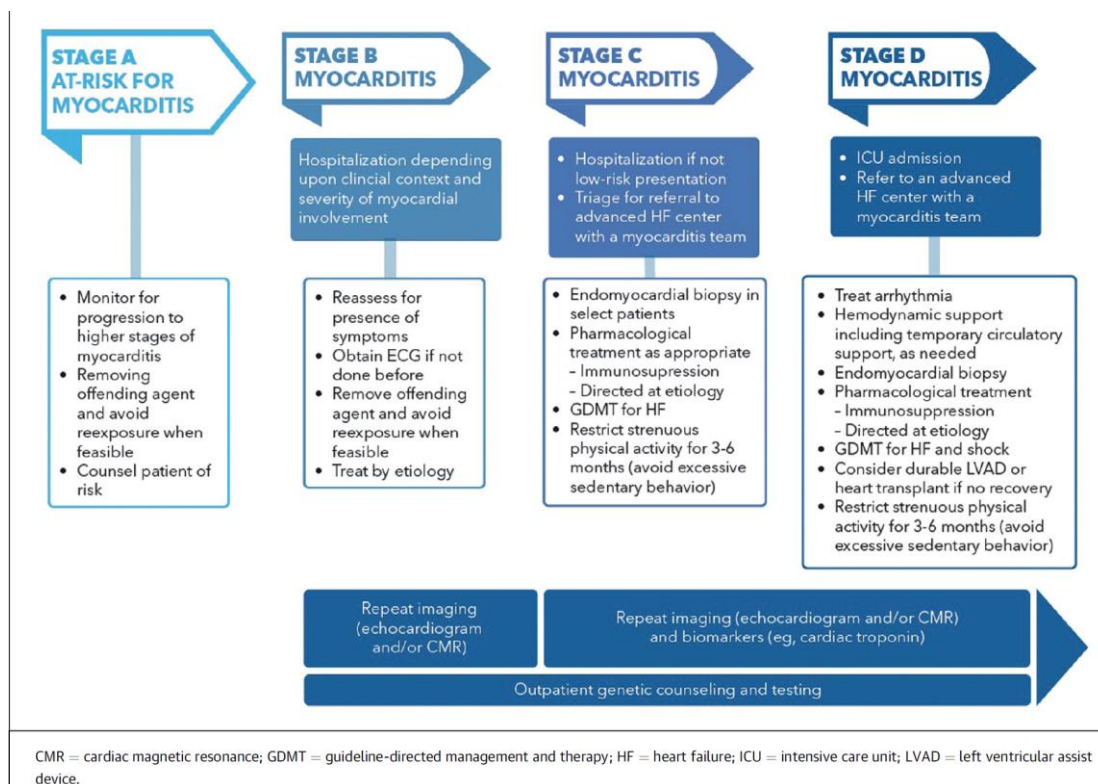


Figure 13. ACC 2024 Decision Pathway on the Diagnosis and Management of Myocarditis. Surveillance and Treatment by Stage of Myocarditis

Table 16. ESC 2025 GL on myocarditis and pericarditis. Therapy for specific forms of myocarditis

1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o. then tapered <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o.
2nd line therapy	Oral corticosteroids + azathioprine ^a or mycophenolate mofetil ^b , cyclosporine ^c , methotrexate ^d
3rd line therapy	IVIg ^e or plasmapheresis ^f
Eosinophilic myocarditis	
1st line therapy	Same as lymphocytic myocarditis + Treat EM-associated condition if identified
2nd line therapy	Same as lymphocytic myocarditis + Treat EM-associated condition if identified
3rd line therapy	–
Giant-cell myocarditis	
1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o. then tapered <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o. + immunosuppressive (azathioprine ^a or mycophenolate mofetil ^b , cyclosporine ^c)
2nd line therapy	Antithymocyte Globulin (ATG) ^g cyclophosphamide ^h , rituximab ⁱ
3rd line therapy	–
Cardiac sarcoidosis	
1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o., tapering from 40–60 mg daily <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o.
2nd line therapy	Methotrexate ^d (1st choice), or azathioprine ^a mycophenolate mofetil ^b , cyclophosphamide ^h
3rd line therapy	Infliximab ^j or adalimumab ^k , rituximab ⁱ
Lyme carditis	
1st line therapy	(a) Oral antibiotics (mild cases): – Doxycycline 100 mg b.i.d. (14–21 days) – Amoxicillin 500 mg t.i.d. (14–21 days) – Cefuroxime axetil 500 mg b.i.d. (14–21 days) (b) i.v. antibiotics (severe cases): – Ceftriaxone 2 g/day (14–21 days)
2nd line therapy	i.v. antibiotics: Cefotaxime (2 g q8h × 14–21 days) or Penicillin G (18–24 MU/day i.v. q4h × 14–21 day)
3rd line therapy	–
Chagas disease	
1st line therapy	Benznidazole 5–7 mg/kg/day in 2 doses for 60 days Nifurtimox 8–10 mg/kg/day in 3 doses for 60–90 days
2nd line therapy	–
3rd line therapy	–

ICI-induced myocarditis	
1st line therapy	Withdraw ICI, reassess <u>Non-severe:</u> methylprednisolone 500–1000 mg/day × 3 days, then taper with oral prednisone <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day × 3 days, then 1 mg/kg/day
2nd line therapy	If no response in 24–48 h: mycophenolate mofetil ^b , ATG ^g abatacept ^l , alemtuzumab ^m
3rd line therapy	Infliximab ^j or adalimumab ^k , rituximab ⁱ

ATG, anti-thymocyte globulins; b.i.d., twice daily; CTLA-4, cytotoxic T-lymphocyte antigen 4; EM, eosinophilic myocarditis; ICI, immune checkpoint inhibitor; IgG, immunoglobulin; IL, interleukin; i.v., intravenous; IVIG, intravenous immunoglobulins; p.o., by mouth; s.c., subcutaneous; t.i.d., three times daily; TNF- α , tumour necrosis factor alpha.

^aAzathioprine (immunosuppressant purine analog): 1–2 mg/kg per day p.o. (typically 100–150 mg daily, in 1–2 divided doses, main target: lymphocytes).

^bMycophenolate mofetil (immunosuppressant that inhibits inosine monophosphate dehydrogenase, main target: lymphocytes): 500–1000 mg p.o. b.i.d. (total 1–2 g/day).

^cCyclosporine (calcineurin inhibitor that prevents IL-2 transcription in activated T-cells): ~3–5 mg/kg/day p.o. (divided b.i.d.) adjusted to target trough levels ~150–250 ng/mL.

^dMethotrexate (antimetabolite that inhibits dihydrofolate reductase and other folate-dependent steps, reducing proliferation of active lymphocytes): 15–20 mg/week p.o. or s.c. (low-dose weekly, with folic acid supplementation).

^eIVIg (immunomodulatory therapy providing pooled IgG antibodies) = standard dose off-label 2 g/kg total dose, typically administered over 1 to 2 days; alternative dosing: 0.4 g/kg/day for 5 consecutive days (less commonly used in myocarditis but sometimes used in autoimmune settings).

^fPlasmapheresis (therapeutic plasma exchange that filters out and removes circulating autoantibodies, immune complexes, and inflammatory mediators) 3–5 sessions in 5–10 days.

^gAntithymocyte Globulin (ATG; polyclonal anti-T-lymphocyte antibody that causes profound T-cell depletion): ~1 mg/kg i.v., often given daily for 3–5 days.

^hCyclophosphamide (cytotoxic alkylating agent that crosslinks DNA in rapidly dividing cells, main target: lymphocytes): 600 mg/m² i.v. bolus on days 1, 15, and 30 (pulse therapy).

ⁱRituximab (monoclonal antibody against CD20 on B cells): 375 mg/m² i.v. weekly × 4 doses (1 month).

^jInfliximab (monoclonal antibody against TNF- α): 5 mg/kg i.v. at weeks 0, 2, 6, then every ~8 weeks (maintenance).

^kAdalimumab (anti-TNF- α fully human monoclonal antibody) 40 mg SC every week (or every 2 weeks, per clinical response).

^lAbatacept (CTLA-4 Ig fusion protein that binds CD80/86 on antigen-presenting cells, blocking the CD28 co-stimulatory signal required for full T-cell activation): 500 mg i.v. every 2 weeks × 5 doses (approximately 10 weeks).

^mAlemtuzumab (monoclonal antibody against CD52 on lymphocytes): 30 mg i.v. once (alternative: 15 mg i.v. daily for 2 days).

Main mechanisms of action:

- T-cell suppression (e.g., corticosteroids, cyclosporine, abatacept)
- B-cell depletion (rituximab)
- Cytokine inhibition (TNF- α blockers like infliximab, adalimumab)
- DNA synthesis inhibition (azathioprine, mycophenolate, methotrexate)
- Immunoglobulin replacement/modulation (IVIg)
- Plasma filtration (plasmapheresis)

Table 17. ESC 2025 GL on myocarditis and pericarditis. Recommendations for giant-cell myocarditis

EMB is recommended in patients with suspected GCM due to unexplained new-onset HF of up to 2 weeks associated with a normal or dilated left ventricle and new ventricular arrhythmias, second- or third-degree AVB, or failure to respond to usual care within 1 to 2 weeks to initiate specific treatment. ⁴⁰²	I	C
Combined immunosuppressive therapy is recommended in patients with a diagnosed GCM. ²¹⁷	I	C

AVB, atrioventricular block; EMB, endomyocardial biopsy; GCM, giant-cell myocarditis; HF, heart failure.

Table 18. ESC 2025 GL on myocarditis and pericarditis. Recommendations for immune checkpoint inhibitor-associated myocarditis

Diagnostic triage within 24 h is recommended ^c in patients with suspected myocarditis induced by ICI to initiate treatment rapidly. ^{495,496,501,504}	I	C
Immediate disruption of ICI and administration of high-dosage corticosteroids are recommended in patients with ICI-associated myocarditis in order to stop the inflammatory reaction and stabilize the patient. ⁵⁰⁴	I	C
Second-line immunosuppression treatment should be considered in patients with steroid-refractory ICI-associated myocarditis. ^{501,504}	IIa	C
Second-line immunosuppression treatment may be considered in patients with fulminant/severe ICI-associated myocarditis. ^{501,504}	IIb	C

ICI, immune checkpoint inhibitor.

Table 19. Arrhythmia management in inflammatory heart disease**ACC/AHA/HRS 2017 GL on VA/SCD. Myocarditis**

Referral to centers with mechanical hemodynamic support and advanced arrhythmia management of patients with life-threatening VT or VF	I-C-LD
ICD* in patients with giant cell myocarditis with VF or hemodynamically unstable VT	IIb-C-LD

*if meaningful survival >1 year is expected.

ESC 2025 GL on myocarditis and pericarditis. Recommendations for management of arrhythmias and prevention of sudden cardiac death in myocarditis

Pacing in myocarditis		
Temporary transvenous external pacing should be considered in patients with acute myocarditis and high-degree conduction disorders as a bridge to recovery. ^{86,317,335}	IIa	C
WCD in myocarditis		
A WCD should be considered for 3–6 months in patients with sustained ventricular arrhythmia during the acute phase of myocarditis as a bridge to recovery. ^{323,325,327–330}	IIa	C
Ablation in myocarditis		
Catheter ablation, performed in specialized centres, should be considered in post-myocarditis patients with recurrent symptomatic SMVT or ICD shocks in whom AAD are ineffective, not tolerated, or not desired. ⁵⁸	IIa	C
ICD in myocarditis		
Secondary prevention		
ICD implantation is recommended in patients with non-active ^c myocarditis and haemodynamically not-tolerated sustained VT to prevent SCD. ^{78,79,322,336}	I	C
ICD implantation should be considered in patients with non-active ^c myocarditis and haemodynamically tolerated sustained VT to prevent SCD. ^{78,79,322,336}	IIa	C
ICD implantation may be considered in patients with acute myocarditis and sustained VA (VT/VF) in the acute phase to prevent SCD. ^{71,79,89,222,323–325}	IIb	C
Primary prevention		
ICD implantation may be considered in patients with myocarditis after the acute phase (3–6 months) and persistent risk factors for VA ^d to prevent SCD. ^{89,332–334,336}	IIb	C

AAD, anti-arrhythmic drugs; CMR, cardiovascular magnetic resonance; ICD, implantable cardioverter-defibrillator; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction; NSVT, non-sustained ventricular tachycardia; PVS, programmed ventricular stimulation; SCD, sudden cardiac death; SMVT, sustained monomorphic ventricular tachycardia; VA, ventricular arrhythmias; VF, ventricular fibrillation; VT, ventricular tachycardia; WCD, wearable cardioverter-defibrillator.

cNon-active based on CMR evidence of activity (T2).

dNSVT, extensive LGE, unexplained syncope, positive PVS, reduced LVEF <50%.

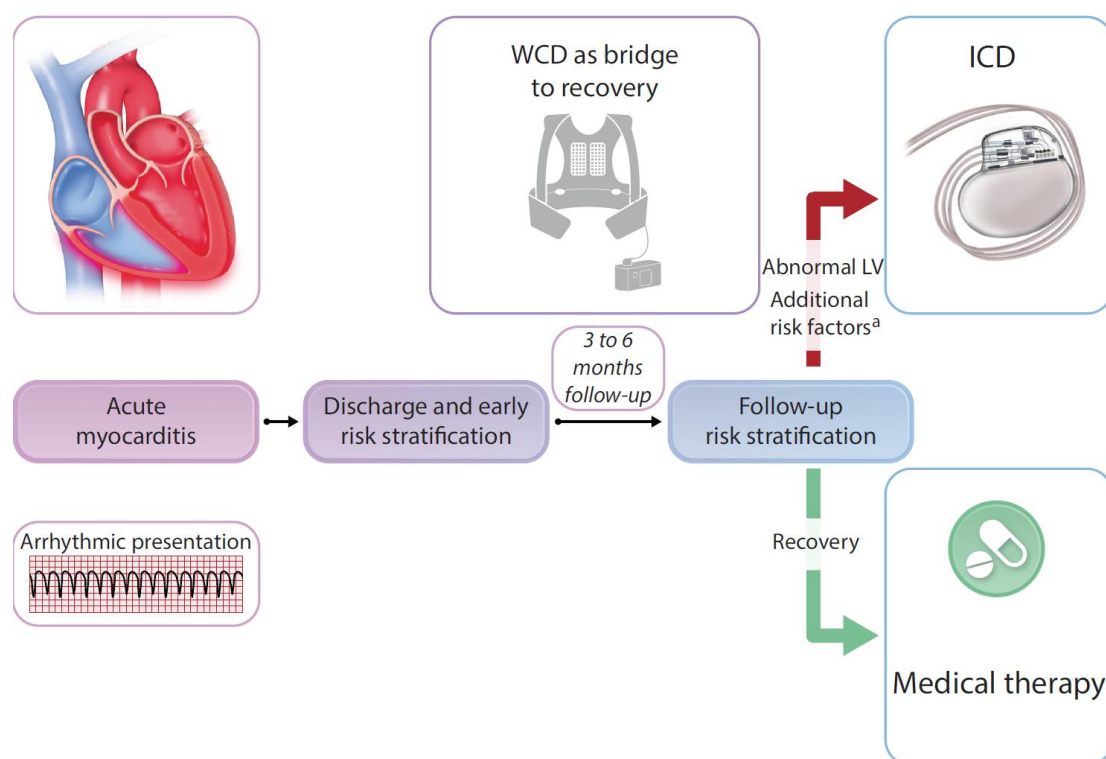


Figure 14. ESC 2025 GL on myocarditis and pericarditis. Follow-up and risk stratification after acute myocarditis with arrhythmic presentation.

ICD, implantable cardioverter-defibrillator; LGE, late gadolinium enhancement; LV, left ventricle; LVEF, left ventricular ejection fraction; NSVT, non-sustained ventricular tachycardia; PVS, programmed ventricular stimulation; WCD, wearable cardioverter-defibrillator. Rarely for a high risk, cardioverter-defibrillator implantation may be considered before discharge (see text for detailed discussion).

^aAdditional risk factors (at least 1, increasing risk and indication with >1 or more): NSVT, extensive LGE, unexplained syncope, positive PVS, reduced LVEF of <50%.

Table 20. ESC 2025 GL on myocarditis and pericarditis. Recommendations for interventional techniques including circulatory support in myocarditis

A timely and dedicated Shock Team discussion is recommended in patients with myocarditis in the presence of haemodynamic compromise, to decide on the need for escalation to MCS and to determine a long-term management plan.	I	C
Temporary MCS ^c should be considered in patients with myocarditis and cardiogenic shock or acute decompensation in chronic myocarditis to stabilize the patients.	IIa	C

IABP, intra-aortic balloon pump; MCS, mechanical circulatory support; PLVAD, percutaneous left ventricular assist device; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

cIABP, PLVAD, VA-ECMO.

Table 21. ESC 2025 GL on myocarditis and pericarditis.**Recommendations for
inflammatory cardiomyopathy**

Guideline-directed heart failure treatments are recommended in patients with inflammatory cardiomyopathy to improve and/or stabilize left ventricular function.	I	C
Specific medical therapy for the potentially underlying systemic disease is recommended in inflammatory cardiomyopathy.	I	C
Immunosuppressive therapy, guided by endomyocardial biopsy, should be considered in virus-negative inflammatory cardiomyopathies to suppress the autoimmune response. ^{34,131,551–555}	IIa	B

**Recommendations for risk stratification, complications, and outcomes of inflammatory
myopericardial syndrome**

Follow-up with clinical assessment, biomarkers ^c , ECG, exercise test, Holter-ECG monitoring, echocardiography, and CMR at least within 6 months after the index hospitalization is recommended in all patients with myocarditis to identify a potential progression or new risk factors. ⁶²	I	C
Long-term follow-up is recommended for patients with complicated myocarditis ^d to identify a potential progression or new complications. ^{28,74}	I	C
Long-term follow-up is recommended for patients with incessant or recurrent pericarditis to identify a potential progression and new complications. ^{104,347}	I	C

CMR, cardiovascular magnetic resonance; ECG, electrocardiogram.

Table 22. Physical activity**ESC 2020 GL on Sports. Recommendations for exercise in individuals with myocarditis**

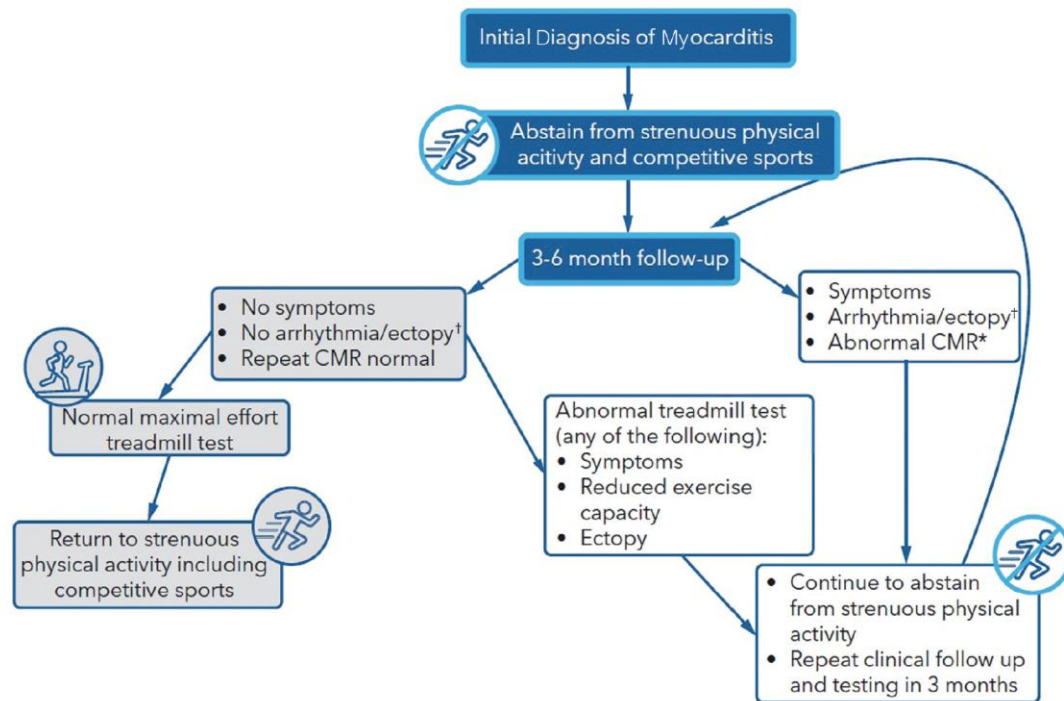
Comprehensive evaluation, using imaging studies, exercise stress test and Holter monitoring to assess the risk of exercise-related SCD.	IB
Return to all forms of exercise including competitive Sports, after 3-6 months in asymptomatic individuals, with normal troponin and biomarkers of inflammation, normal LV systolic function on echocardiography and CMR, no evidence of ongoing inflammation or myocardial fibrosis on CMR, good functional capacity, and absence of frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing.	IIa C
Among individuals with a probable or definitive diagnosis of recent myocarditis, participation in leisure-time or competitive sports while active inflammation is present is not recommended.	III C
Participation in moderate- to high-intensity exercise for a period of 3-6 months after acute myocarditis is not recommended.	III B
Participation in leisure exercise or competitive sports involving high intensity in individuals with residual myocardial scar and persistent LV dysfunction is not recommended.	III C

CMR = cardiovascular magnetic resonance; LV = left ventricular; SCD = sudden cardiac death; VA = ventricular arrhythmia.

ESC 2025 GL on myocarditis and pericarditis. Recommendations for physical activity and myocarditis/pericarditis

Restriction of physical exercise until remission, for at least 1 month, is recommended in athletes and non-athletes after IMPS using an individualized approach to accelerate recovery.	I	C
---	----------	----------

IMPS, inflammatory myopericardial syndrome.



*An abnormal CMR includes any of the following: reduced LVEF or RVEF; or evidence of nonischemic inflammation as described previously, including presence of LGE.
 †Assessed by 24-hour electrocardiographic monitoring. CMR = cardiac magnetic resonance; LGE = late gadolinium enhancement; LVEF = left ventricular ejection fraction; RVEF = right ventricular ejection fraction.

Figure 15. ACC 2024 Decision Pathway on the Diagnosis and Management of Myocarditis. Recommendations for Abstinence From Strenuous Physical Activity and Return to Competitive Sports Following Diagnosis of Myocarditis

Table 23. ESC 2025 GL on myocarditis and pericarditis. Follow-up in inflammatory myopericardial syndrome after discharge

		Within 1 month	Within 3–6 months	12 months	>1 year and long-term FU ^a
Clinical evaluation and ECG	Myocarditis	X	X	X	X
	Pericarditis	X	X	X	X
Biomarkers (TnI, C-reactive protein)	Myocarditis	X	X	(X)	(X)
	Pericarditis	X	X	(X)	(X)
Rhythm (stress and/or Holter-ECG)	Myocarditis	–	X	(X)	(X)
	Pericarditis	–	–	–	–
Imaging myocarditis	TTE		X ^b	X ^c	X ^c
	CMR		X ^b	X ^c	X ^c
Imaging pericarditis	TTE		X ^b	X ^c	X
	CMR		(X) ^b	(X) ^d	(X) ^d

CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; FU, follow-up; IMPS, inflammatory myopericardial syndrome; TnI, troponin I; TTE, transthoracic echocardiography.

All follow-ups should be adapted to the clinical situation and severity. In round brackets, optional testing according to clinical presentation [(X), case-by-case decision].

^aA long-term FU, e.g. after 2 years, is suggested only for complicated cases of IMPS, usually myocarditis.

^bIn complicated cases or if abnormal at 1 month, imaging should be repeated between 3 and 6 months.

^cIf abnormal at 6 months, imaging should be repeated within the next 6 months and/or in the next 12 months.

^dFollow-up proposed for uncomplicated cases of acute pericarditis. Long-term follow-up, tailored to the single patient, is recommended for high-risk cases.