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Relapsing Pericarditis: Prediction of Relapses, Risk Scores, and Role of Exercise Restriction

REVIEW

JOSEPH EL ROUMI, MD
JIBRAN IKRAM, MD
TOM KAI MING WANG, MBCHB, MD

*Author affiliations can be found in the back matter of this article

HOUSTON Methodist DEBAKEY HEART & VASCULAR CENTER

CME

ABSTRACT

Relapsing pericarditis (RP) is a chronic inflammatory disorder characterized by two or more episodes of acute pericarditis flares after a minimum of a 4-week symptom-free period. Recurrent pericarditis develops in approximately 15% to 30% of patients following an initial episode of acute pericarditis, and nearly half of these individuals experience subsequent recurrences. Although the course is heterogeneous, RP has substantial morbidity and impaired quality of life implications and is associated with pericardial complications, including cardiac tamponade and constrictive pericarditis. This review summarizes current knowledge about clinical perspectives, predictors, and risk stratification tools for relapses in RP and critically appraises the evolving role of exercise restriction as part of RP management. Many adverse prognosticators for RP in clinical, laboratory, multimodality cardiac imaging, and treatment factors have been identified from clinical trials, observational studies, and experiences of managing RP patients. Several risk scores have been recently developed to assist in risk stratification and treatment guidance for RP patients, such as the Athens, Torino, INFLA, and Klein scores. There is also growing evidence for exercise restriction strategies with a focus on practical individualized strategies in the multimodal treatment of RP that have been included in recent pericarditis guidance documents. Multicenter external validation and randomized trials remain necessary to assess the roles and performance of risk scores and exercise restriction in treating RP to improve their clinical outcomes.

CORRESPONDING AUTHOR:
Tom Kai Ming Wang, MBChB, MD
Center for Diagnosis and Treatment of Pericardial Diseases, Heart, Vascular and Thoracic Institute, Cleveland Clinic, Cleveland, Ohio, USA
wangt2@ccf.org

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INTRODUCTION

Acute pericarditis accounts for approximately 5% of nonischemic chest pain presentations in the emergency department,[1] with an estimated 1 in roughly 60,000 individuals in the United States affected.[2] Relapsing pericarditis (RP) is defined as a new episode of typical pericarditis signs and symptoms after a symptom-free interval of at least 4 to 6 weeks following an initial acute pericarditis episode.[1,3,4] RP occurs in approximately 15% to 30% of patients after a first episode of acute pericarditis and up to 40% to 50% after a second episode.[1,5,6] These patients experience repeated flares, emergency visits, and hospitalizations over several years, with substantial psychological distress, exercise limitation, and steroid-related side effects.[7] Understanding the risk factors—whether clinical, laboratory, imaging, or treatment-related—and synthesizing these would help guide risk stratification and management of RP patients.

Older randomized trials established colchicine for at least 3 to 6 months in combination with nonsteroidal anti-inflammatory drugs (NSAIDs) or high-dose aspirin as the first-line therapy for pericarditis.[4,8-11] Recent randomized trials in the last decade have demonstrated the high efficacy of anti-interleukin-1 (IL-1) agents for managing RP,[12-14] leading to a paradigm shift in practice and incorporating them as second- or third-line agents in contemporary guidelines,[4,8] while corticosteroids remain an alternative option in this setting, especially if the underlying etiology is autoimmune. Radical pericardiectomy continues to be the last treatment option, preferably performed at a high-volume experienced surgical center.[8,15] Moreover, key lifestyle modifications are integral to the management of pericarditis, particularly adherence to exercise restriction. Although supporting evidence has historically been sparse and largely extrapolated from patients with myocarditis, growing availability of wearable devices and objective activity metrics has renewed interest in the potential interaction between physical activity, subclinical inflammation, and the risk of future relapses.[8,16,17] As such, exercise restriction is now integral in guidance documents in the management of RP.

This review discusses contemporary understanding of the adverse prognosticators and risk stratification tools as well as the role of exercise restriction in the multimodal management of RP.

CLINICAL PERSPECTIVES

Acute pericarditis is a clinical diagnosis defined by the presence of characteristic pericardial chest pain in combination with at least two additional diagnostic criteria, as outlined by the American College of Cardiology (ACC) and European Society of Cardiology (ESC).[4,8] These criteria include a pericardial friction rub, typical electrocardiographic changes (diffuse ST-segment elevation and PR depression), new or worsening pericardial effusion, and evidence of systemic inflammation such as elevated C-reactive protein (CRP) or erythrocyte sedimentation (ESR), with supportive imaging findings on echocardiography or cardiac magnetic resonance (CMR) when available.[4,8] Based on symptom duration and disease course, pericarditis is classified as acute when symptoms last less than 4 to 6 weeks, subacute or incessant when symptoms persist beyond this period without a symptom-free interval, and chronic when inflammation continues for more than 3 months. RP is defined by the reappearance of pericardial symptoms after a documented initial episode, separated by a symptom-free interval of at least 4 to 6 weeks, accompanied by objective evidence of renewed pericardial inflammation.[4,8]

Recent epidemiological studies suggest that RP is associated with a 3-fold higher risk of pericardial effusion (PEff) and a 2-fold higher risk of cardiac tamponade (CTP) and constrictive pericarditis (CP).[6] However, a chronic recurrent course is associated with repeated emergency room visits and hospitalizations, long-term corticosteroid exposure and side effects (weight gain, osteoporosis, hyperglycemia, infection), impaired exercise capacity and quality of life, and mental health disturbances such as anxiety and depression.

Recurrent pericarditis is primarily rooted in auto-inflammatory mechanisms, reflecting dysregulation of the innate immune response rather than a classic adaptive autoimmune process. A central role has been attributed to inappropriate activation of the NLRP3 inflammasome, resulting in excess production of IL-1 and amplification of sterile inflammation within the pericardium. This IL-1–driven pathway offers a coherent biological framework that integrates the typical clinical presentation—episodic inflammatory flares often associated with fever and elevations in acute-phase reactants—with objective markers of disease activity. Systemic inflammatory burden is captured by CRP elevation, whereas CMR provides complementary tissue-level confirmation of active pericardial inflammation through findings such as T2-weighted edema and increased extracellular volume, even in the absence of a significant effusion. Importantly, failure of inflammatory markers to normalize, or persistence of CMR evidence of active inflammation, identifies a subgroup of patients at increased risk for relapse and corticosteroid dependence. This pathophysiologic model also underpins the robust clinical efficacy of IL-1–targeted therapies, which address the upstream driver of inflammation rather than

relying on nonspecific immunosuppression, supporting a more mechanism-based approach to risk stratification and treatment in recurrent pericarditis.[18]

PREDICTORS OF RELAPSE IN RP

Understanding the predictors of relapse is critical in the management of acute and recurrent pericarditis, as presence of one or more of these factors could signal greater severity and/or longer duration of disease. Predictors for relapse in RP can be divided into clinical, laboratory, imaging, and treatment-related factors as listed in Table 1.

CLINICAL PREDICTORS OF RELAPSING PERICARDITIS

Several clinical features have consistently been associated with an increased risk of relapse and progression to recurrent pericarditis, as summarized in the 2025 ESC Guidelines on Pericardial Diseases.[8] A higher inflammatory burden at initial presentation manifested by fever > 100.4°F, elevated inflammatory markers, and a subacute or incessant symptom onset rather than an abrupt presentation has been linked to a more complicated disease course and greater relapse risk. Large pericardial effusion, cardiac tamponade, failure to respond to NSAIDs, and presence of concomitant high-risk myocardial phenotype at presentation are similarly recognized high-risk features associated with adverse outcomes.[8] Importantly, prognosis differs across myocarditis-spectrum phenotypes: patients with myopericarditis and preserved left ventricular systolic function generally have a favorable long-term prognosis, whereas higher-risk features such as reduced ejection fraction, ventricular arrhythmias, or hemodynamic instability identify patients at increased risk of adverse outcomes.[19]

Demographic factors also appear to influence recurrence risk; studies have shown that younger age and female sex are independently associated with a higher likelihood of recurrent pericarditis despite acute pericarditis being more common in men, suggesting that sex-related differences in immune response rather than autoimmune disease alone may underlie this association.[20] Additionally, RP commonly presents with atypical or incomplete clinical features, which contributes to delayed or missed diagnoses in routine practice. Recurrent episodes may manifest with nonspecific chest discomfort, dyspnea, fatigue, or low-grade systemic inflammatory symptoms, often in the setting of absent or only modest biomarker elevation. Electrocardiographic abnormalities are frequently nondiagnostic, and pericardial effusion may be minimal or absent, particularly during recurrent flares. This heterogeneous presentation complicates diagnostic decision-making, especially in outpatient and emergency settings, underscoring the importance of maintaining a high index of suspicion and integrating clinical assessment with biomarkers and advanced imaging to identify active disease.[21,22]

CLINICAL PREDICTORS	LABORATORY PREDICTORS	IMAGING PREDICTORS	TREATMENT-RELATED PREDICTORS
Fever > 100.4°F at presentation	Elevated baseline CRP	Persistent pericardial LGE on CMR	Early or high-dose corticosteroid use
Subacute or incessant disease course	Delayed CRP normalization after treatment initiation	Persistent pericardial T2 hyperintensity (edema) on CMR	Rapid tapering of NSAIDs or corticosteroids
Large pericardial effusion	Elevated ESR	Large or recurrent pericardial effusion on echocardiography	Absence, under-dosing, or early discontinuation of colchicine
Cardiac tamponade at presentation	Neutrophilia or lymphopenia	Constrictive physiology (echo or CMR features)	Inadequate duration of anti-inflammatory therapy
Younger age	Elevated NLR	Pericardial thickening or calcification on CT	Failure to escalate therapy in refractory disease
Female sex	Elevated PIV	Myocardial involvement (myopericarditis) on CMR	Delayed initiation of IL-1 inhibition when indicated
Autoimmune or autoinflammatory disease	Elevated cardiac troponin (myocardial involvement)	Reduced LVEF or regional wall motion abnormalities	Poor treatment adherence
Post–cardiac injury syndromes	Elevated BNP/NT-proBNP	Effusive-constrictive pericarditis	Failure to identify and treat underlying etiology

Table 1 Established predictors of relapse in recurrent pericarditis. BNP: B-type natriuretic peptide; CMR: cardiac magnetic resonance; CRP: C-reactive protein; CT: computed tomography; ESR: erythrocyte sedimentation rate; IL-1: interleukin-1; LGE: late gadolinium enhancement; LVEF: left ventricular ejection fraction; NLR: neutrophil-to-lymphocyte ratio; NSAID: nonsteroidal anti-inflammatory drug; PIV: pan-immune-inflammatory value

Immune-mediated conditions further contribute to relapse risk, with autoimmune and systemic inflammatory diseases including systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), Sjögren's syndrome, and monogenic autoinflammatory syndromes being well-recognized contributors to persistent or recurrent inflammation.[23] Conversely, pericardial manifestations in RA span a broad clinical spectrum, ranging from incidentally detected, asymptomatic pericardial effusions to clinically overt acute or recurrent pericarditis, and may occur independently of active articular disease. Although subclinical pericardial effusion is the most common presentation, a subset of patients may develop symptomatic inflammatory pericarditis that can follow a relapsing course. Recognition of RA-associated pericardial disease is clinically relevant, as management often requires coordination between cardiology and rheumatology, with treatment strategies tailored to both pericardial inflammatory activity and systemic disease control, as highlighted in contemporary ACC guidance on rheumatic pericarditis.[5]

Moreover, in patients with SLE, interpretation of inflammatory biomarkers requires caution, as CRP levels are frequently normal or only modestly elevated during noninfectious lupus flares despite clinically active disease. This contrasts with other inflammatory conditions and reflects differences in cytokine signaling pathways that limit hepatic CRP production in SLE. Importantly, a disproportionately elevated CRP in a patient with SLE should raise concern for superimposed infection or active serositis, including pericardial involvement, rather than isolated immune-mediated disease activity. This diagnostic distinction is clinically relevant when evaluating patients with suspected lupus-related pericarditis, as reliance on CRP alone may underestimate inflammatory activity, whereas marked CRP elevation may prompt evaluation for alternative or concomitant etiologies. Recognition of this biomarker pattern, as emphasized in prior rheumatologic literature, can improve diagnostic accuracy and guide appropriate management.[24]

In addition, post-cardiac injury syndromes, including post-pericardiotomy and post–myocardial infarction pericarditis, are associated with higher recurrence rates due to sustained immune activation following myocardial or pericardial injury.[25] Collectively, these clinical predictors underscore the heterogeneity of recurrent pericarditis and support early risk stratification to guide monitoring intensity and therapeutic escalation.

LABORATORY MARKER PREDICTORS OF PERICARDITIS RELAPSE

Traditional inflammatory markers remain clinically informative for risk stratification and longitudinal management in pericarditis. Elevated baseline CRP and ESR at presentation reflect a higher inflammatory burden and have been consistently associated with an increased risk of recurrence.[8,26,27] Persistent elevation or delayed normalization of CRP after initiation of anti-inflammatory therapy is particularly prognostic; failure of CRP to normalize within the first 7 to 10 days identifies patients at higher risk of relapse and supports slower tapering and prolonged therapy.[26,27]

More recently, data from the Cleveland Clinic have expanded the prognostic role of biomarkers beyond traditional inflammation. In a large cohort of patients with idiopathic recurrent pericarditis, Yesilyaprak et al. demonstrated that higher inflammatory activity during active flares was associated with delayed clinical remission and inferior long-term outcomes, reinforcing the importance of biomarker intensity in predicting disease trajectory.[28] Importantly, this work laid the foundation for the Klein risk score, which integrates clinical features and imaging parameters, particularly the extent of late gadolinium enhancement (LGE) on CMR to stratify patients according to likelihood of remission and chronic disease evolution.[28]

Beyond CRP, leukocyte-derived indices have emerged as adjunctive predictors of relapse. Neutrophilia, lymphopenia, and an elevated neutrophil-to-lymphocyte ratio (NLR) have been associated with greater disease activity and adverse outcomes, including higher rates of tamponade and recurrence, with proposed high-risk thresholds such as NLR ≥ 5 in acute inflammatory presentations.[29] Composite immune indices, including the pan-immune-inflammatory value, which incorporates neutrophils, lymphocytes, monocytes, and platelets, have shown associations with disease severity and recurrence risk although external validation remains limited.[30] Laboratory evaluation should also extend beyond inflammatory markers when clinically indicated. Cardiac troponin elevation reflects myocardial involvement along the pericarditis–myocarditis disease spectrum and is useful for phenotypic classification and early risk assessment.[8,31] In the absence of ventricular dysfunction or electrical instability, myocardial involvement accompanying pericarditis is typically self-limited and does not, by itself, portend adverse long-term outcomes. By contrast, myocardial disease characterized by impaired systolic function, extensive myocardial involvement on advanced imaging, or clinically significant arrhythmias identifies a distinct subgroup with higher short-term risk and greater need for surveillance.[31] From a relapse standpoint, however, myocardial injury markers alone provide limited prognostic insight. Recurrent pericarditis is driven predominantly by ongoing pericardial inflammatory activity, and assessment of recurrence risk is therefore

best guided by longitudinal inflammatory markers and objective evidence of pericardial inflammation on imaging rather than isolated or transient myocardial biomarker elevation.[32]

Similarly, elevated natriuretic peptides—such as brain natriuretic peptide (BNP) and N-terminal proBNP—may suggest concomitant heart failure or evolving constrictive physiology; while values are often lower in isolated constriction compared with restrictive cardiomyopathy, abnormal elevations have been linked to adverse outcomes in pericardial syndromes with myocardial or hemodynamic involvement.[33] Together, these laboratory markers complement clinical and imaging findings and support a multidimensional approach to relapse risk stratification in recurrent pericarditis.

MULTIMODALITY IMAGING PARAMETERS ASSOCIATED WITH PERICARDITIS RELAPSE

Echocardiography

Transthoracic echocardiography (TTE) remains the first-line imaging modality for the evaluation of pericarditis and provides essential diagnostic and prognostic information. TTE allows systematic grading of PEff as trivial, small, moderate, or large based on end-diastolic echo-free space while also characterizing effusion distribution (circumferential vs loculated), echogenicity, and associated fibrinous strands, which may suggest inflammatory or malignant etiologies.[8,27,34] TTE is also central to the diagnosis of CTP, identifying hallmark features such as right atrial and right ventricular diastolic collapse, exaggerated respiratory variation in mitral and tricuspid inflow velocities, inferior vena cava plethora, and reduced cardiac output, and it plays a key role in guiding the timing and safety of pericardiocentesis.[8,27,34]

Beyond PEff assessment, TTE provides important prognostic insights through evaluation of myocardial and pericardial physiology. Regional wall-motion abnormalities (RWMAs) and reduced left ventricular ejection fraction (LVEF) may indicate concomitant myocardial involvement (myopericarditis), which is associated with a more complicated clinical course and warrants closer monitoring.[31] TTE also facilitates differentiation between constrictive and restrictive physiology, with features of constriction including interventricular septal bounce, marked respiratory variation in ventricular filling, annulus reversus, and hepatic vein diastolic flow reversal with expiration.[34] Importantly, TTE can identify distinct entities along the pericardial disease spectrum, including effusive-constrictive pericarditis and transient constrictive pericarditis, which have implications for prognosis and therapeutic decision-making.[4,8,34] In selected patients, stress echocardiography or cardiopulmonary exercise testing may further refine risk stratification, as reduced exercise capacity and lower achieved metabolic equivalents have been associated with worse functional status and outcomes in pericardial and constrictive syndromes.[4,8,27,34,35]

Computed Tomography

Computed tomography (CT) may provide supplementary information showing pericardial thickening and PEff (especially when echocardiographic windows are poor), which are some diagnostic but also potentially prognostic markers for RP. CT is also the best modality for assessing pericardial calcifications seen in chronic CP that portend worse prognosis, often needing diuretic symptom control and radical pericardiectomy. Importantly, CT also serves as a useful initial test in the evaluation of differential diagnoses of chest pain, including coronary artery disease, acute aortic syndromes, pulmonary emboli, pneumonia, and other thoracic pathologies while also having important roles in the preoperative evaluation for pericardiectomy surgery.

Cardiac Magnetic Resonance

CMR has become an indispensable and comprehensive tool for the assessment of pericardial diseases in terms of diagnosis, prognosis, treatment guidance, and surveillance and is recommended for RP evaluation.[4,8,27,36] Prior CMR studies largely assessed pericardial LGE using qualitative (visual) grading (eg, none/trace, mild, moderate, severe), and consistently showed that greater LGE burden reflects more active pericardial inflammation and identifies patients with a more "active" phenotype prone to recurrence or earlier relapse.[32,37] Building on these visual methods, later cohorts introduced semiquantitative approaches such as intensity relative to myocardium or blood pool and fully quantitative methods (eg, objective signal/intensity or volume-based metrics) to standardize LGE assessment; across these approaches, higher LGE severity has generally tracked with ongoing disease activity and higher relapse risk.[32,37] There is also a role for serial LGE and T2 imaging, whereby persistence of these abnormalities whether in the presence or absence of clinical remission are associated with RP relapse.[1,38] CMR should be emphasized not only for risk stratification but also as a critical diagnostic adjunct in patients with suspected recurrent/relapsing pericarditis when clinical criteria are equivocal or incomplete. In this setting, multiparametric CMR can provide objective evidence of active pericardial inflammation by demonstrating pericardial edema on T2-weighted (or STIR-based) imaging and pericardial late gadolinium enhancement (LGE)/delayed hyperenhancement, even when ECG findings are nondiagnostic and pericardial effusion is minimal or absent. Quantitative assessment of pericardial delayed

hyperenhancement has been shown to help identify patients with ongoing recurrences and supports improved diagnostic certainty in "silent" or atypical presentations, with downstream implications for treatment intensification and tapering decisions.[36,39]

More recently, the International Position Statement (IPS) and ACC concise clinical guidance have proposed a standardized pericardial LGE severity grading framework (optimized for phase sensitive inversion recovery/PSIR-LGE, ideally with fat suppression) to improve consistency across centers and enable longitudinal monitoring; adopting these criteria in routine clinical practice is encouraged to harmonize reporting, guide risk stratification, and support treatment escalation/de-escalation decisions in relapsing/recurrent pericarditis.[3,4]

In addition, several prognostically relevant features traditionally assessed by echocardiography in RP can be comprehensively evaluated by CMR. CMR allows accurate assessment of pericardial effusion size and distribution, while tissue characterization (T1/T2 signal, presence of fibrinous strands, loculations, or hemorrhagic features) provides insight into inflammatory activity and disease chronicity. CMR is also well-suited to evaluate constrictive physiology, demonstrating pericardial thickening, LGE, ventricular interdependence with septal bounce, inspiratory septal shift, and dissociation of intracardiac and intrathoracic pressures on real-time cine imaging. Finally, CMR is the reference standard for cardiac chamber size and ventricular function, and the presence of reduced LVEF or RWMAs accompanied by nonischemic myocardial LGE (mid-myocardial or subepicardial) supports the presence of high-risk concomitant myocarditis or myopericarditis, a phenotype associated with a more complicated clinical course and worse prognosis.[3,4,36]

TREATMENT-RELATED FACTORS FOR RP

Therapeutic selection and tapering strategies play a critical role in determining relapse risk in pericarditis. Early or high-dose corticosteroid exposure in idiopathic pericarditis is consistently associated with increased recurrence rates, likely due to impaired viral clearance and suppression of innate immune responses, predisposing to rebound inflammation upon withdrawal. In addition, abrupt or overly rapid tapering of NSAIDs or corticosteroids frequently precipitates disease flares, underscoring the importance of a gradual, individualized taper guided by clinical symptoms, inflammatory markers such as CRP, and, when available, CMR findings. Colchicine therapy, which reduces recurrence risk by approximately 50%, is recommended for most patients with acute or RP unless contraindicated; however, inadequate dosing, shortened treatment duration, or poor adherence is associated with higher relapse rates. Finally, failure to identify and address the underlying etiology, including autoimmune disease, malignancy, post-radiation pericardial injury, or infectious causes, can result in persistent or recurrent disease despite standard anti-inflammatory therapy.

Management of Higher Risk Patients

In the presence of one or more high-risk features, management of pericarditis should be intensified in accordance with ESC recommendations.[8] These patients generally warrant hospital admission for close monitoring, expedited diagnostic evaluation, and early assessment of treatment response rather than routine outpatient management. Initial therapy should be started promptly with full-dose first-line anti-inflammatory treatment (high-dose NSAIDs or aspirin plus colchicine), but lack of clinical or biochemical response within a short interval should prompt earlier escalation to second-line therapies rather than prolonged ineffective treatment.[8] High-risk patients often require a longer duration of therapy and slower tapering, guided by both symptom resolution and objective markers of inflammation. Follow-up should be more frequent, with inflammatory markers like CRP monitored monthly until normalization, and then at longer intervals (every 3 months), particularly in patients receiving advanced therapies such as IL-1 inhibitors. Clinical follow-up is recommended at 1- to 3-month intervals until sustained remission is achieved, and subsequently every 3 to 6 months thereafter. When an alternative or systemic etiology is suspected (eg, autoimmune, autoinflammatory, infectious, or malignant causes), early referral to rheumatology or other relevant specialties is advised to ensure a multidisciplinary, etiology-directed approach and to reduce the risk of relapse or complications.[8] Patients who have new chest pain should be promptly reviewed and inflammatory markers repeated to confirm diagnosis and treat as soon as possible.

RISK SCORES FOR RP

Risk scores play an important role in the risk stratification of patients with cardiovascular disease to help guide management decisions, including treatment options, durations, and surveillance. Four risk scores in pericarditis have recently been published, as summarized in Table 2 and below.[40]

ATHENS RECURRENCE SCORE

Developed in a cohort with first-episode acute pericarditis, this score incorporates six predictors of pericarditis recurrence: age, effusion size, platelet count, inferior vena cava collapse, in-hospital corticosteroid use, and heart

NAME OF SCORE	ATHENS RECURRENCE SCORE	TORINO PERICARDITIS SCORE	INFLA SCORE	KLEIN REMISSION SCORE
Author / Year	Lazarou et al., 2021	Imazio et al., 2021	Andreis et al., 2025	Yesilyaprak et al., 2024
Study population	~240 patients; first episode acute pericarditis	~500 patients; acute pericarditis at presentation	~200 patients; suspected acute pericarditis presenting with chest pain	~500 patients; recurrent or refractory pericarditis managed at tertiary center
Primary outcome predicted	Risk of pericarditis recurrence	Risk of complicated pericarditis (recurrence, tamponade, or constriction)	Diagnosis of pericarditis (inflammatory probability; indirect risk stratification)	Probability of achieving steroid-free clinical remission
Score parameters	Age; heart rate; platelet count; pericardial effusion size; inferior vena cava collapse; in-hospital corticosteroid use	Fever > 100.4°F; subacute/ incessant course; large pericardial effusion; cardiac tamponade; NSAID non-response; immunosuppression	CRP; white blood cell count; neutrophil count; platelet count; chest pain characteristics	Age; sex; number of recurrences; autoimmune etiology; heart rate category; pericardial LGE severity on CMR; left ventricular ejection fraction; colchicine use; corticosteroid exposure; DMARD use
Performance (AUC / C-statistic)	C-index ≈ 0.74 for recurrence prediction	C-index ≈ 0.75–0.80 for complicated pericarditis	AUC ≈ 0.80 for pericarditis diagnosis	C-index ≈ 0.80 for remission prediction

Table 2 Contemporary risk scores for risk stratification in pericarditis. AUC: area under the receiver operating characteristic curve; CMR: cardiac magnetic resonance; CRP: C-reactive protein; DMARD: disease-modifying antirheumatic drug; late gadolinium enhancement; NSAID: nonsteroidal anti-inflammatory drug

rate. Patients with low scores (0-7 points) had ~21% recurrence risk, whereas those with high scores (≥ 12 points) had ~70% recurrence risk, with good discriminative performance (c-index ~0.74).[38] This score is primarily applicable at the initial presentation of acute pericarditis, particularly in emergency department or inpatient settings, where they may aid in identifying patients at increased risk for recurrence or a complicated clinical course and inform decisions regarding closer monitoring or intensified initial therapy.

TORINO PERICARDITIS SCORE

Designed to predict the risk of "complicated pericarditis" (recurrence, CTP, or CP) at presentation, the Torino score combines the parameters of fever, subacute/incessant course, large pericardial effusion, cardiac tamponade, non-response to NSAIDs and immunosuppression to identify patients at particularly high risk who may warrant closer monitoring and more aggressive therapy.[41] Conversely to the Athens score, this score is most useful at the time of first acute pericarditis presentation, especially in hospitalized or emergency care settings, as tools to stratify recurrence risk and identify patients who may benefit from closer surveillance or more aggressive early management.

PERICARDITIS INFLA SCORE

This score integrates inflammatory biomarkers and clinical features including C-reactive protein, white cell, neutrophil and platelet counts, and chest pain characteristics to improve the diagnosis of inflammatory pericarditis in patients with chest pain and suspected pericarditis.[42] It also indirectly stratifies risk by quantifying inflammatory burden. The INFLA score was developed to support initial diagnostic discrimination, hence it derives its role by helping clinicians distinguish pericarditis from alternative etiologies of chest pain at presentation—whereas recurrence risk assessment relies on different clinical, biomarker, and imaging factors that evolve over time. Accordingly, the INFLA score should not be interpreted as a relapse-prediction instrument.

KLEIN REMISSION SCORE

Developed in a tertiary center cohort of patients with recurrent or refractory disease, this score uses a range of clinical, laboratory and imaging markers to predict the probability of achieving steroid-free clinical remission.[40] In a separate CMR-based model for recurrent pericarditis, ten variables were incorporated, including age, sex, number of baseline recurrences, autoimmune etiology, heart rate category, LGE severity, preserved LVEF, and exposure to colchicine, steroids, or disease-modifying antirheumatic drugs. Patients were stratified into low (≤ 3), intermediate, and high (≥ 8) risk groups, which were associated with different remission rates (higher risk has lower remission rates).[26,38,43,44]

The Klein score model is best suited for use in tertiary referral populations with recurrent or refractory pericarditis, where its primary objective is to estimate the probability of achieving steroid-free remission rather than to predict acute complications at initial presentation. By incorporating advanced disease-activity measures, particularly CMR markers of pericardial inflammation such as the extent of LGE, the model offers meaningful prognostic insight

and supports therapeutic decision-making in specialized centers. However, this reliance on advanced imaging and expert interpretation, while a clear strength in experienced settings, also limits its applicability and generalizability in broader clinical environments.

The apparent discrepancy between LVEF and relapse risk reflects differences in the populations and existing risk models. In the Klein score, preserved or higher LVEF was paradoxically associated with increased risk of recurrence, likely serving as a surrogate for predominantly inflammatory, non-myocardial disease, in which recurrent pericardial inflammation occurs in the absence of significant ventricular dysfunction. In contrast, prior studies have identified reduced LVEF or RWMAs as markers of myopericardial involvement, reflecting more extensive myocardial inflammation or injury, which has been associated with a higher risk of complicated clinical courses and recurrences. Thus, preserved LVEF in isolated pericarditis and reduced LVEF in myopericarditis may represent distinct pathophysiologic phenotypes, each associated with relapse risk through different mechanisms.[5,26]

FUTURE DIRECTIONS OF PREDICTORS AND RISK SCORES

Future directions in risk prediction for RP should focus on identifying novel and biologically grounded predictors that extend beyond traditional clinical and laboratory variables. Emerging inflammatory biomarkers (eg, high-sensitivity cytokine profiles), genetic susceptibility loci, and proteomic signatures may offer mechanistic insight into disease recurrence and treatment resistance. Advanced CMR techniques including tissue characterization with T1/T2 mapping, extracellular volume quantification, and emerging fingerprinting approaches hold promise for refining inflammatory phenotyping and temporal risk stratification. In parallel, machine-learning–based models that integrate multimodal data (clinical, imaging, laboratory, and longitudinal disease trajectories) may improve discrimination and calibration compared with conventional regression-based scores. However, refinement of existing risk scores must prioritize external validation across independent cohorts and healthcare systems to establish generalizability, reproducibility, and accuracy in predicting RP flares beyond the derivation populations.[3,4,40]

Importantly, future research must move beyond risk prediction toward risk-guided management strategies and demonstrable clinical benefit. Most existing RP risk scores were developed before the era of IL-1, limiting their applicability in contemporary practice. There is a critical need to identify predictors and develop risk models that specifically assess recurrence risk in patients receiving IL-1 blockade both among the rare patients experiencing flares while on therapy and, more commonly, among patients discontinuing anti-IL-1 agents. Comparative evaluation of different weaning strategies, including emerging data from the Cleveland Clinic rilonacept discontinuation experience, will be essential to inform personalized tapering approaches.[45] Ultimately, prospective studies are required to determine whether incorporating validated risk scores into treatment algorithms meaningfully alters clinical decision-making, optimizes therapy duration, and improves patient-centered outcomes such as flare reduction, steroid avoidance, and quality of life.

EXERCISE RESTRICTION

PHYSIOLOGICAL RATIONALE

Exercise imposes multiple hemodynamic and inflammatory stresses that can exacerbate active or residual pericardial inflammation. During physical activity, increased heart rate and cardiac output raise pericardial shear forces and friction, while elevated blood pressure and ventricular filling pressures heighten mechanical stress on inflamed pericardial layers. Concurrent sympathetic activation and catecholamine surges may further influence immune pathways and cytokine release. Moreover, vigorous exercise acutely elevates circulating IL-6 and other proinflammatory mediators, potentially amplifying subclinical pericardial inflammation. In predisposed individuals, these combined physiological and inflammatory responses may trigger recurrence or worsening of pericarditis.[46]

EMERGING DATA LINKING EXERCISE AND RELAPSE

Emerging data increasingly suggests an association between activity levels and the risk of pericarditis relapse, supporting a more cautious and individualized approach to exercise during recovery. Cardiac MRI studies indicate that patients who resume higher-intensity activity while still exhibiting LGE or T2 signal abnormalities experience higher recurrence rates, reinforcing the value of imaging-guided activity restriction.[47] Observational data from wearable devices further show that periods of increased physical exertion reflected by heart rate surges and decreases in heart rate variability may precede symptomatic flares, suggesting that mechanical and autonomic stress can unmask subclinical inflammation.[48] Clinically, athletes and highly active patients appear particularly vulnerable to relapse when returning to vigorous training before achieving complete inflammatory quiescence, underscoring the need for tailored guidance based on symptoms, biomarkers, and imaging.[1,49]

EXERCISE RESTRICTION IN THE ACUTE AND RELAPSING PHASE

During active inflammation, a conservative approach is highly recommended. Vigorous physical activity and all competitive sports should be restricted, while lighter exercise activities may be allowed once pain is controlled. Activities should be limited to walking and standard light-weight activities of daily living. Exercise limitation should continue until symptoms have completely resolved and inflammatory markers, particularly CRP, have normalized. In patients considered higher risk, such as those with multiple prior recurrences or residual imaging abnormalities, additional confirmation through CMR normalization or substantial improvement can provide further reassurance before liberalizing activity.[1,50,51]

REINTRODUCTION PHASE

After resolution of pericardial inflammation (complete symptom resolution, normalization of inflammatory markers such as CRP when available, the absence of active pericardial inflammation on imaging, particularly cardiac magnetic resonance), activity should resume gradually following a structured, stepwise progression, from low-intensity activities and gradually to moderate exercise. Exercise advancement should be individualized and guided by clinical tolerance, with close monitoring for recurrent chest pain, palpitations, or unusual fatigue.[52] Continuing colchicine and IL-1 inhibition when part of the therapeutic regimen during early reconditioning may reduce the risk of relapse.[1,53] Studies emphasize strict exercise restriction during any phase of active pericarditis and shift from a fixed timeline to a criteria-based return-to-activity approach. Patients may resume exercise only after complete symptom resolution, normalization of inflammatory markers (preferably high-sensitivity CRP), and absence of effusion or active inflammation on imaging. Athletes still require a minimum of 3 months before returning to competitive training. Non-athletes may resume earlier if all remission criteria are satisfied. Persistent subclinical inflammation warrants continued restriction due to the heightened recurrence risk.[1,3,4]

ATHLETES AND HIGH-RISK PATIENTS

Athletes and individuals with high-risk clinical or imaging features require heightened caution when considering return to exercise. In competitive athletes with myocarditis, contemporary international guidelines uniformly recommend complete exercise restriction for a minimum of 3 to 6 months, with return-to-play permitted only after resolution of symptoms, normalization of biomarkers, recovery of ventricular function, and absence of active inflammation or high-risk arrhythmias on cardiac testing. These recommendations are based on strong observational evidence linking exercise during active myocardial inflammation to adverse outcomes, including malignant arrhythmias and sudden cardiac death. Although pericarditis carries a lower arrhythmic risk profile than myocarditis, the shared inflammatory pathophysiology and growing evidence linking residual inflammation to relapse provide a rationale for extrapolating these myocarditis-based principles to selected pericarditis populations. Accordingly, competitive sports should generally be deferred for at least 3 months after the last pericarditis flare and longer in patients with persistent CMR abnormalities, multiple recent recurrences, or elevated risk scores. In such high-risk scenarios, a more prolonged restriction period and coordinated management involving sports cardiology and a dedicated pericardial disease specialist are recommended to ensure a safe, individualized return-to-play pathway.[1,50,52,54]

Moreover, management of higher-risk RPs should distinguish etiology-directed referral from complexity-based referral, as these pathways address fundamentally different clinical needs. Etiology-directed referral is appropriate when clinical, laboratory, or imaging features raise concern for an alternative or secondary cause of pericardial disease, including systemic autoimmune disorders (eg, systemic lupus erythematosus, rheumatoid arthritis), infection, malignancy, or post-cardiac injury syndromes, where disease-specific evaluation and treatment are required. In contrast, complexity-based referral applies to patients with refractory autoinflammatory recurrent pericarditis, characterized by steroid dependence, colchicine resistance or intolerance, frequent relapses, or persistent pericardial inflammatory activity on imaging.

In these patients, referral to specialized pericardial disease centers facilitates access to IL-1 pathway inhibition, advanced CMR–based inflammatory phenotyping, and structured anti-inflammatory tapering strategies. Importantly, relapse risk and overall disease burden in recurrent pericarditis are driven primarily by ongoing pericardial inflammatory activity rather than etiologic labels alone. Accordingly, incorporation of objective markers, including CRP trajectories and CMR findings of edema and LGE, into referral and treatment-escalation decisions aligns with current expert consensus and evolving clinical care pathways.[36]

FUTURE DIRECTIONS IN EXERCISE RESTRICTION

Emerging work increasingly supports a shift from uniform, prolonged activity restriction toward individualized, data-driven exercise “prescriptions.” Future research priorities

should aim to compare different exercise intensities and optimal timing of return to activity in patients with relapsing pericarditis—including prospective multicenter randomized trials, which are critically needed to establish safe thresholds for activity and to define their effects on relapse rates and patient-reported quality of life. The integration of wearable-derived metrics—such as heart rate patterns, step count, and heart rate variability into relapse prediction models may allow real-time titration of physical activity and early identification of physiological signatures preceding recurrence. Risk factors and scores in RP may also assist in the development of personalized exercise algorithms that replace traditional one-size-fits-all restrictions. This precision-guided strategy would represent a major step toward safer, more patient-centered rehabilitation in RP.

CONCLUSION

Contemporary management of recurrent pericarditis is best approached through an integrated framework that links mechanistic understanding, objective disease assessment, and individualized clinical decision-making. Recognition of RP as a predominantly IL-1–mediated autoinflammatory condition provides a unifying biological foundation for therapy, while inflammatory biomarkers and advanced imaging (mainly CMR) offer objective tools to confirm active disease and refine diagnostic certainty when clinical features are incomplete or atypical. Within this context, emerging risk stratification tools and imaging markers may help identify patients at increased risk for relapse, steroid dependence, or treatment resistance, although their performance in the era of widespread IL-1 inhibition remains incompletely validated. Similarly, while exercise restriction and graded return to activity are central components of management, prospective data guiding individualized exercise prescriptions based on inflammatory burden are lacking.

Historical "one-size-fits-all" approaches are being replaced by more nuanced, individualized strategies that account for patient-specific risk and disease activity. Risk stratification and wearable technologies, poised to enable personalized exercise prescriptions, can optimize the balance between relapse prevention and maintenance of physical and psychological well-being. Future studies integrating mechanistic biomarkers, imaging phenotypes, and patient-centered outcomes will be essential to validate risk models, refine exercise recommendations, and advance a truly personalized approach to recurrent pericarditis care.

KEY POINTS

- Relapsing pericarditis (RP) occurs in 15% to 30% of patients after a first episode and between 40% and 50% after a second episode, leading to substantial morbidity and impaired quality of life.
- Clinical (fever, subacute course, autoimmune disease), laboratory (C reactive protein, neutrophil-to-lymphocyte ratio, pan-immune-inflammatory value), imaging (persistent cardiac magnetic resonance late gadolinium enhancement/T2 abnormalities) and treatment factors are established predictors of RP flares.
- Novel risk scores including the Athens, Torino, INFLA, and Klein scores for predicting RP relapse or remission were recently developed.
- Exercise restriction is an important pillar in the management of RP and moving towards individualized strategies
- Future research should focus on external validation of risk scores, integration of novel markers, and comparisons of exercise prescription studies in prospective multicenter trials.

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COMPETING INTERESTS

The authors have no competing interests to declare.

AUTHOR AFFILIATIONS

Joseph El Roumi, MD orcid.org/0009-0005-8594-3407
Center for Diagnosis and Treatment of Pericardial Diseases, Heart, Vascular and Thoracic Institute, Cleveland Clinic, Cleveland, Ohio, USA

Jibran Ikram, MD orcid.org/0009-0003-6653-0661
Center for Diagnosis and Treatment of Pericardial Diseases, Heart, Vascular and Thoracic Institute, Cleveland Clinic, Cleveland, Ohio, USA

Tom Kai Ming Wang, MBChB, MD orcid.org/0000-0001-5570-9402
Center for Diagnosis and Treatment of Pericardial Diseases, Heart, Vascular and Thoracic Institute, Cleveland Clinic, Cleveland, Ohio, USA

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