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Research Article

Oral Cannabidiol in Inflammatory Myo-Pericardial Diseases: A Systematic Review of Myo-Pericardial Recovery, Inflammation, Clinical Outcomes, and Safety

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ABSTRACT:

Background: Inflammatory myo-pericardial syndromes, including myocarditis, myopericarditis, peri-myocarditis, and inflammatory cardiomyopathy, are associated with myo-pericardial injury, ventricular dysfunction, arrhythmia, and adverse long-term remodeling. Despite advances in cardiac magnetic resonance (CMR)-based diagnosis and risk stratification, therapeutic options targeting myo-pericardial inflammation remain limited. Cannabidiol (CBD), a non-intoxicating Phyto-cannabinoid with anti-inflammatory, antioxidant, and potential immunomodulatory properties, has emerged as a candidate therapy for inflammatory cardiac disease. This systematic review evaluated the efficacy, safety, and potential effects of oral CBD on myo-pericardial recovery in adults with inflammatory myo-pericardial syndromes.

Methods: A systematic review was conducted in accordance with PRISMA 2020. PubMed/MEDLINE, Embase, Scopus and Web of Science were searched from inception through 2026. Eligible studies included adults with acute or chronic myocarditis, myopericarditis, peri-myocarditis, or inflammatory cardiomyopathy receiving oral CBD, including pharmaceutical-grade formulations. Randomized controlled trials and relevant prospective or retrospective clinical studies were eligible. Outcomes included CMR measures of myo-pericardial recovery, cardiac function and remodeling, inflammatory markers, clinical outcomes, and adverse events.

Results: Clinical evidence available consisted of the Phase II ARCHER trial, in which 109 acute myocarditis patients were randomized to oral CBD (n=56) or placebo (n=53). CBD had no significant effect on myo-pericardial extracellular volume or global longitudinal strain but significantly decreased left ventricular mass (mean difference −9.23 g; 95% CI −16.36 to −2.11; P=0.0117) and left atrial end-systolic volume (mean difference −8.09 mL; P=0.0376). Experimental studies showed decreased pericardial effusion, increased pericardial thickness, and decreased inflammatory signals in the pericardium; and the Phase II MAvERIC-Pilot trial in 27 patients with recurrent pericarditis showed that CBD decreased pain and normalized CRP in patients with elevated baseline CRP, with 80% remaining recurrence-free at the time of extension follow-up. In general, there were suggestive beneficial effects of CBD on myo-pericardial remodeling and pericardial inflammation, but evidence for definite clinical efficacy is lacking.

Conclusions: Current evidence does not demonstrate a statistically significant benefit of oral cannabidiol on the primary measures of myo-pericardial recovery, specifically CMR-derived extracellular volume and global longitudinal strain, in mild-to-moderate acute myocarditis. Nevertheless, favorable changes in selected measures of cardiac remodeling and supportive mechanistic evidence suggest a potential therapeutic role that warrants further investigation. The

evidence base remains limited to a small number of studies, and larger, adequately powered trials with longer follow-up, phenotype-specific assessment, and clinically meaningful outcomes are required before oral cannabidiol can be considered part of routine treatment for inflammatory myo-pericardial syndromes.

Keywords: Cannabidiol, CBD, Myocarditis, Inflammatory Myo-Pericardial Syndromes, Myopericarditis, Inflammatory Cardiomyopathy, Cardiac Magnetic Resonance, Myo-Pericardial Recovery, Cardiac Remodeling, Systematic Review.

INTRODUCTION

The inflammatory myo-pericardial syndromes are a diverse group of cardiovascular conditions in which there is myo-pericardial inflammation and/or inflammation of adjacent cardiac structures such as acute, chronic myocarditis, myopericarditis, peri myocarditis, and inflammatory cardiomyopathy. Chest pain may be present in these patients, but may be vague, and there may be only minimal elevation of serum markers, or the disease may lead to dysfunction of the ventricles, malignant arrhythmias, cardiogenic shock, and sudden cardiac death. Importantly, continued myo-pericardial inflammation could lead to adverse remodeling and fibrosis of the ventricles, leading to chronic myo-pericardial dysfunction and heart failure. The recently published European Society of Cardiology (ESC) Guidelines have introduced the umbrella concept of inflammatory myo-pericardial syndromes to highlight the clinical and pathophysiological overlap between myocarditis and pericarditis and related inflammatory cardiac disorders[1-2].

Although significant progress has been made in cardiovascular magnetic resonance (CMR), endomyo-pericardial biopsy, biomarkers, and multimodality imaging, therapeutic choices in inflammatory myo-pericardial disease are still restricted and often are dependent on the underlying etiology. Acute myocarditis is primarily managed by supportive measures and treatment of complications (e.g., arrhythmias and heart failure), and immunomodulatory therapy is used for certain immune-mediated or histologically confirmed myocarditis. This leaves significant unmet clinical need for therapies that can inhibit pathological inflammation with minimal myo-pericardial damage and support myo-pericardial structure and function recovery[3-4].

It is now becoming clear that inflammatory processes are crucial to myo-pericardial injury and not just a consequence of tissue damage. Innate immune pathways, such as the nucleotide-binding oligomerization domain, leucine-rich repeat, and pyrin domain-containing protein 3 (NLRP3) inflammasome, may further magnify myo-pericardial inflammation by stimulating the inflammatory enzymes caspase-1 and the maturation of inflammatory cytokines such as interleukin (IL)-1β and IL-18. The sustained inflammasome activation could lead to the recruitment of leukocytes, damage of cardiomyocytes, remodeling of extracellular matrix and myo-pericardial fibrosis. Experimental and translational studies indicate that modulation of NLRP3-mediated inflammatory pathways may decrease myo-pericardial injury and promote resolution of inflammation, providing a novel therapeutic pathway for development[5-6].

Cannabidiol (CBD), a non-intoxicating Phyto-cannabinoid extracted from Cannabis sativa, has been found to be a potential anti-inflammatory and cardioprotective agent. CBD does not have the typical cannabis effects that people experience like the "high" from Δ9-THC. CBD has been shown in preclinical studies to have the ability to modulate multiple inflammatory and oxidative pathways, such as pathways associated with NLRP3 inflammasome activation, and to decrease the production of pro-inflammatory cytokines like IL-1β and IL-6. Experimental models of autoimmune myocarditis have also raised the possibility of a role in myo-pericardial inflammation and fibrosis, lending to the idea that pharmaceutical CBD may be a potential option to be explored in inflammatory cardiac disease[7-8].

Mechanistic insights have been translated into studies of the cardiovascular system in humans through the clinical development of pharmaceutical oral cannabidiol. Pharmaceutically produced oral cannabidiol formulation, CardiolRx™, has been specifically studied in patients with inflammatory cardiovascular diseases. The Phase II ARCHER trial used CMR-derived parameters of myo-pericardial recovery (ECV, GLS) as well as other structural and functional cardiac parameters in adults with acute myocarditis, and this is an important advance, as CMR can provide quantitative measures of myo-pericardial tissue characteristics and cardiac function that may be

sensitive to persistent inflammation, edema and remodeling[9-10].

Clinical evidence has since emerged that has turned cannabidiol from an experimental anti-inflammatory compound into a therapy that has the potential to modify the disease in inflammatory myo-pericardial disorders. The evidence available, however, is relatively limited and the studies vary in outcome definitions, imaging parameters, safety assessment, treatment duration, and disease phenotype, with differences in these areas. In addition, the effects of oral CBD on myo-pericardial recovery, inflammatory markers, symptoms, ventricular function, recurrence and tolerability of treatment have not been synthesized in a comprehensive manner. The uncertainties are especially relevant in the current management of inflammatory myo-pericardial syndromes, where increasingly a mechanism-directed and individual-based approach is applied.

Therefore, the aim of this systematic review is to critically appraise the existing clinical evidence on the therapeutic effects of oral CBD in inflammatory myo-pericardial syndromes. Specifically, its effects on myo-pericardial recovery and cardiac function, inflammatory and imaging biomarkers, clinical symptoms and disease recurrence, safety and tolerability, will be assessed. This review aims to elucidate the current clinical and translational evidence for oral CBD and highlight the gaps in the current evidence base and future research priorities for conducting RCTs and translating findings into clinical practice.

METHODOLOGY

Study Design and Reporting Standards

The objective of this systematic review was to assess the therapeutic potential of oral cannabidiol (CBD) in inflammatory myo-pericardial syndromes, focusing on efficacy, safety, inflammatory outcomes and myo-pericardial recovery. Design and reporting of the review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement and its reporting recommendations. A methodology for the review was developed prior to the literature screening process. If applicable, the review protocol was prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO).

Search Strategy

The aim was to conduct a detailed literature search in the major electronic databases: PubMed/MEDLINE, Embase, Scopus and Web of Science. All included studies and relevant systematic or narrative reviews, included in the reference lists, were screened manually to identify additional eligible studies. Where appropriate, key publications were also cited in a citation tracking.

Literature search included articles published from the period of inception of the databases to the time of final search (2026). No restriction was made based on publication year only.The search strategy consisted of controlled and free-text terms for cannabidiol and inflammatory myo-pericardial disease, myocarditis, myopericarditis, pericarditis, myo-pericardial inflammation, and other cardiovascular inflammatory diseases. The terms used to search were modified for the indexing system of each database.

The basic idea of search was organized around sets of terms like: (cannabidiol) OR (CBD) OR (pharmaceutical cannabidiol) OR (oral cannabidiol) OR (CardiolRx)) AND (myocarditis OR myopericarditis OR peri-myocarditis OR inflammatory myo-pericardial disease OR myo-pericardial inflammation OR inflammatory cardiomyopathy). All database-specific search strategies will be available in the Supplementary Material and will include the use of MeSH terms and Boolean Operators.

Study Selection

All retrieved records were entered into a reference management program (Endnote) and any duplicate records were eliminated prior to screening. The study-selection process was done in two phases.

The titles/abstracts were screened for pre-defined eligibility criteria in the first step. Second, a full-text evaluation of potentially relevant articles was conducted. The studies not eligible for either stage were omitted, and reasons for full-text stage were recorded.

Study selection was done in an independent manner, where possible, by two reviewers. Any disagreements were discussed and resolved as a consensus with the help of a third reviewer.

The entire selection process will be displayed in the PRISMA 2020 flow diagram, and the number of records identified, duplicates excluded, records screened, full-text articles assessed, studies excluded and reasons for exclusion, and studies included in the full text assessment will be reported.

Eligibility Criteria

Inclusion Criteria

- Adults (18 years and older) with acute or chronic myocarditis, myopericarditis, peri-myocarditis or inflammatory cardiomyopathy.
- Research examining oral cannabidiol (CBD), including pharmaceutical grade products like CardiolRx™.
- Randomized controlled trials, prospective or retrospective observational studies, and single-arm clinical studies.
- At least one of the following efficacy, myo-pericardial recovery, inflammatory, clinical or safety outcomes were reported.
- Full-text studies published in English, from database inception to the final search date in 2026.

Exclusion Criteria

- Studies that have not been reported separately for adults or pediatric studies.
- Research on the evaluation of non-oral CBD preparations, cannabis, or mixed cannabinoid preparations, in which the effects of CBD cannot be separated.
- Animal, in-vitro and other preclinical studies.
- Case reports, narrative reviews, systematic reviews, editorials, protocols, and conference abstracts (without enough data).
- Studies that did not provide any data that could be extracted for inflammatory myo-pericardial syndromes.
- Duplicate publications and/or studies that were not available in full text.

Intervention The intervention of interest was oral cannabidiol**,** including pharmaceutical-grade cannabidiol formulations such as CardiolRx™. Studies evaluating cannabidiol administered through non-oral routes were excluded unless outcomes for the oral formulation could be separately extracted.

Outcomes

The primary outcomes were measures of myo-pericardial recovery and cardiac function, including changes in left ventricular ejection fraction, global longitudinal strain, myo-pericardial extracellular volume, cardiac magnetic resonance (CMR) parameters, myo-pericardial edema, and other validated measures of myo-pericardial structural or functional recovery.

Data Extraction

A standardized data-extraction form was developed before extraction. Two reviewers independently extracted relevant information from eligible studies. Extracted data included:

1. First author and publication year;
2. Country and study setting;
3. Study design;
4. Sample size;
5. Participant demographic and clinical characteristics;
6. Diagnostic criteria for inflammatory myo-pericardial disease;
7. Type and formulation of cannabidiol;
8. Cannabidiol dose and dosing schedule;
9. Duration of treatment and follow-up;
10. Concomitant cardiovascular or anti-inflammatory therapies;
11. Myo-pericardial recovery and cardiac-function outcomes;
12. Clinical symptoms and disease recurrence;
13. Hospitalization and cardiovascular outcomes;
14. Adverse events and serious adverse events;
15. Treatment discontinuation; and
16. Authors' reported conclusions and relevant limitations.

Assessment of Risk of Bias

The study designs were evaluated for risk of bias using a validated methodological tool. The Cochrane Risk of Bias 2 (RoB 2) tool was used to analyze the bias of the randomized controlled trials, including the randomization process, deviations from intended interventions, missing outcome data, outcome measurement, and selection of the reported result.

For non-randomized comparative studies, the ROBINS-I tool was used to assess the studies, if applicable. If ROBINS-I was not suitable for prospective or single arm observational studies, an appropriate validated critical-appraisal instrument was used, depending on the study design.

Two reviewers independently completed risk-of-bias assessments; discrepancies were resolved through consensus and/or with a third reviewer.

Data Synthesis

A structured qualitative synthesis was performed for all included studies. Study characteristics, patient populations, interventions, comparators, outcomes, and risk-of-bias assessments were summarized in evidence tables.

RESULTS

Study Selection and Characteristics

The literature search revealed only a few clinical studies that explored the therapeutic potential of oral cannabidiol (CBD) in inflammatory cardiac diseases. After the removal of duplicates, title and abstract screening, and full-text assessment based on the pre-defined eligibility criteria, one randomized controlled trial directly evaluated the use of oral cannabidiol in acute myocarditis was included in the primary clinical synthesis. This was Phase II of an ARCHER trial, a multicenter, international, randomized, double blind, placebo-controlled trial of 109 adults with acute myocarditis confirmed by CMR. Of them, 56 took a pharmaceutical-grade, oral dose of CBD; 53 took a placebo. Treatment was given for 12 weeks, and the primary efficacy measures were the measures of myo-pericardial recovery derived from CMR.

The preserved LV systolic function in the included population was associated with myo-pericardial involvement on CMR. Baseline mean LV EF was around 60.6%, mean extracellular volume (ECV) was 38.9% and global longitudinal strain (GLS) was −15.3%. The dose of cannabidiol was gradually increased up to a maximum dose of 20 mg/kg twice daily. Primary endpoints included changes in ECV and GLS, with secondary and exploratory endpoints being LVEF, ventricular volumes, left ventricular mass and left atrial volume.

In a subsequent Phase II study, MAvERIC-Pilot, 27 adults with symptomatic recurrent pericarditis were studied with the use of pharmaceutical-grade oral cannabidiol. The review was not included in the main clinical synthesis because the population of isolated pericarditis, without myo-pericardial involvement, was not included in the predefined population for this review, and patients with myocarditis were excluded from the review. The results were taken separately as corroborative evidence from an inflammation of the heart, very similar in nature.

Effects on Myo-Pericardial Recovery

The ARCHER trial failed to show any statistically significant difference in either of its prespecified primary endpoints. At 12 weeks, adjusted mean ECV was −3.67 mL (95% CI, −7.41 to 0.06; P=0.0538) lower in the cannabidiol group than in the placebo group, at 33.61 mL and 37.28 mL, respectively. The direction of effect was towards CBD, and while the difference did not meet the standard for statistical significance, it indicated that the chance of myo-pericardial extracellular remodeling may have been reduced.

There was no significant difference among the groups in GLS. Adjusted GLS at week 12 was −16.03% with cannabidiol and −15.96% with placebo, with a between-group difference of −0.07 (95% CI, −1.21 to 1.07; P=0.9021). LVEF was also similar at week 12, at 62.09% in the cannabidiol group and 61.54% in the placebo group (between-group difference 0.55%, 95% CI, −1.85 to 2.94; P=0.6520). The results suggest that, after 12 weeks of treatment, there were no significant improvements in global systolic function or GLS with CBD.

Effects on Cardiac Remodeling

Although there was no significant improvement in primary measures, some CMR secondary measures were in favor of CBD. The cannabidiol group had significantly lower LV mass at 12 weeks (121.10 g) than the placebo group (130.33 g) (adjusted between-group difference, −9.23 g; 95% CI, −16.36 to −2.11; P=0.0117). Cannabidiol also significantly decreased LAESV (95% CI, −15.71 to −0.47; P=0.0376).

Other parameters of ventricular remodeling showed favorable trends that were not statistically significant. There was a difference of about −7.43 mL (P=0.098) in left ventricular end-diastolic volume and a difference of −5.57 mL (P=0.093) in intracellular volume. The results indicate that there may be an effect of CBD on structural remodeling, although the results should be taken with a grain of salt as they were secondary or exploratory outcomes, with the primary outcomes not being statistically significant.

Inflammatory and Clinical Outcomes

Experimental evidence supports the biological basis for cannabidiol in inflammatory myo-pericardial disease by showing its modulation of inflammatory and oxidative pathways, such as signaling of the NLRP3 inflammasome. In experimental autoimmune myocarditis, CBD treatment has been proposed to decrease inflammatory cell infiltration, myo-pericardial fibrosis, oxidative/nitrative stress, and myo-pericardial dysfunction.

In acute myocarditis, there are limited clinical data on systemic inflammatory outcomes. Primary endpoints of the ARCHER trial were myo-pericardial recovery instead of inflammatory markers. Based on the current

clinical data, there is not yet a clear reduction of systemic inflammatory activity seen with cannabidiol in myocarditis.

There is supportive evidence from MAvERIC-Pilot, in which oral pharmaceutical CBD was used to treat recurrent pericarditis. The mean maximal pain scores dropped from 5.8 ± 1.7 at baseline to 2.1 ± 1.8 at week 8 and 8 of 10 patients with elevated baseline CRP had normalisation of the CRP. In the optional extended follow-up period, 17 of 24 patients did not have a recurrence of pericarditis. The results of this study were not considered evidence of efficacy in myocarditis because the study was open label, single arm, and performed in isolated recurrent pericarditis.

Safety and Tolerability

Overall, oral CBD was well tolerated in the ARCHER trial. The treatment-emergent adverse events reported in 43 (76.8%) of those taking cannabidiol and 32 (60.4%) of those taking placebo. The most common adverse event was diarrhea, which was reported in 18 participants (32.1%) in the cannabidiol group and 11 (20.8%) in the placebo group. Other reported events were chest pain and nausea.

There were no significant differences in treatment discontinuation due to treatment-emergent adverse events between the three treatment groups.No major imbalance in treatment tolerability was found, with treatment discontinuation due to treatment-emergent adverse events occurring in three patients in each treatment group. Elevations in alanine aminotransferase levels were reported in five participants who were treated with cannabidiol and three participants who were treated with placebo. There was no clinically significant QTc prolongation found during follow up. The two people who received cannabidiol said they had thoughts or actions about suicide and were suicidal at baseline.

Supportive Preclinical Evidence

While preclinical studies are not included in the main clinical synthesis, they do support the therapeutic hypothesis. Experimental autoimmune myocarditis experiments have shown that CBD can decrease myo-pericardial inflammatory infiltration, fibrosis, oxidative/nitrative stress and myo-pericardial injury, and improve both systolic and diastolic function. The results indicate that the therapeutic properties of CBD may go beyond symptom management to modulating pathological myo-pericardial inflammation and remodeling.

The signs from associated inflammatory cardiac disease are promising as well. The MAvERIC-Pilot study found that, in recurrent pericarditis, treatment with pharmaceutical cannabidiol resulted in a reduction in pain and inflammatory activity, thus showing preliminary clinical evidence for the anti-inflammatory activity of pharmaceutical cannabidiol in inflammatory cardiovascular diseases. However, there is no control group and the sample size is quite small, which makes it difficult to draw causal conclusions.

Evidence Primarily on Involvement of the Pericardium

The available evidence on pericardial diseases was less comprehensive, with both preclinical and clinical studies available. Experimental investigations of acute pericarditis showed that CBD decreased the inflammation of the pericardium, pericardial effusion, pericardial thickening, inflammatory signaling, and the mesothelial-to-mesenchymal transition. Recently, a Phase II clinical trial of oral pharmaceutical-grade CBD for recurrent pericarditis demonstrated a decrease in pain and inflammatory markers associated with pericarditis, and a significant proportion of patients did not experience another recurrence of pericarditis during follow-up. A Phase III randomized controlled trial of cannabidiol to prevent recurrent pericarditis has also been launched. In summary, this data suggests that the use of oral CBD may have beneficial anti-inflammatory and clinical effects in pericardial disease, but there are still limited studies available. Details are shown in table 5.

Table 1. Studies Involving Primarily the Myocardium

Study	Design	Population	Sample size	Intervention	Comparator	Treatment duration	Primary outcomes
McNamara et al.	Multicenter, internatio	Adults with CMR-	109 randomized; 56	Pharmaceutical-grade oral	Matching placebo	12 weeks	CMR-derived extracellu

(ARCHER), 2026	nal, randomized, double-blind, placebo-controlled Phase II trial	confirmed acute myocarditis	cannabidiol, 53 placebo; mITT: 49 and 50, respectively	cannabidiol, titrated from 2.5 to 10 mg/kg twice daily			lar volume (ECV) and global longitudinal strain (GLS)

Additional Characteristics: Participants were randomized within 10 days of diagnosis. The study included patients with predominantly mild-to-moderate acute myocarditis and relatively preserved LV systolic function. Mean baseline LVEF was 60.6%, while mean baseline ECV was 38.9% and GLS −15.3%. All randomized participants completed the study, although 11 participants discontinued study treatment prematurely.

Table 2. Efficacy And Myo-Pericardial Recovery Outcomes

Outcome at 12 weeks	Cannabidiol	Placebo	Between-group difference (95% CI)	P value	Interpretation
ECV (mL)	33.61	37.28	−3.67 (−7.41 to 0.06)	0.0538	Favorable trend, but not statistically significant
GLS (%)	−16.03	−15.96	−0.07 (−1.21 to 1.07)	0.9021	No significant difference
LVEF (%)	62.09	61.54	+0.55 (−1.85 to 2.94)	0.6520	No significant difference
LV mass (g)	121.10	130.33	−9.23 (−16.36 to −2.11)	0.0117	Significant reduction with cannabidiol
Intracellular volume (mL)	85.59	91.16	−5.57 (−12.09 to 0.95)	0.0928	Favorable but not statistically significant
LVEDV (mL)	134.48	141.91	−7.43 (−16.25 to 1.40)	0.0981	Trend toward reduction
LVESV (mL)	51.30	54.23	−2.92 (−7.77 to 1.92)	0.2339	No significant difference
LAESV (mL)	49.95	58.04	−8.09 (−15.71 to −0.47)	0.0376	Significant reduction with cannabidiol

Abbreviations: CMR, cardiac magnetic resonance; ECV, extracellular volume; GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LAESV, left atrial end-systolic volume.

Table 3. Safety And Adverse Events in the ARCHER Trial

Safety outcome	Cannabidiol (n=56)	Placebo (n=53)	P value/interpretation
≥1 treatment-emergent adverse event	43 (76.8%)	32 (60.4%)	Overall events generally mild
Diarrhea	18 (32.1%)	11 (20.8%)	P=0.200
Chest pain	9 (16.1%)	4 (7.5%)	P=0.239

Nausea	5 (8.9%)	2 (3.8%)	P=0.439
Increased ALT	5 (8.9%)	3 (5.7%)	P=0.717
Palpitations	1 (1.8%)	4 (7.5%)	P=0.198
Treatment discontinuation because of TEAE	3	3	Comparable between groups
Serious adverse events	Reported	Reported	No clear overall safety imbalance
Clinically important QTc change	None identified	None identified	No important QTc signal
Suicidal thoughts	2 patients	Not reported	Both had previous suicidal ideation/behavior at baseline

ALT: alanine aminotransferase; QTc, corrected QT interval; TEAE, treatment-emergent adverse event.

Table 4. Supportive Preclinical and Related Inflammatory-Cardiac Evidence.

Study/evidence	Model/population	Intervention	Principal findings	Relevance to current review
Experimental autoimmune myocarditis study	Murine model of T-cell-mediated autoimmune myocarditis	Cannabidiol	Reduced myo-pericardial inflammatory-cell infiltration and myo-pericardial injury; improved systolic and diastolic function; reduced fibrosis and oxidative/nitrative stress	Provides biological rationale for anti-inflammatory and cardioprotective effects
ARCHER trial rationale/design	Adults with acute myocarditis; clinical trial framework	Oral pharmaceutical cannabidiol	Based on preclinical anti-inflammatory and cardioprotective effects; designed to evaluate CMR markers of myo-pericardial recovery	Established the translational rationale for clinical testing
MAvERIC-Pilot, 2026	Adults with symptomatic recurrent pericarditis	Oral pharmaceutical cannabidiol	Pain score decreased from 5.8±1.7 to 2.1±1.8 by week 8; CRP normalized in 8/10 patients with elevated baseline CRP; 17/24 remained recurrence-free during extension	Supportive evidence from related inflammatory cardiac disease; not included in primary myocarditis synthesis

Broader CBD cardiovascular/preclinical literature	Experimental inflammatory and cardiovascular models	Cannabidiol	Evidence suggests modulation of inflammatory, oxidative, fibrotic, and inflammasome-related pathways	Supports mechanistic plausibility but cannot establish clinical efficacy

Table 5: Studies Involving Primarily the Pericardium

Study	Year	Study type	Population/model	CBD intervention	Main findings	Status/relevance
Martinez Naya et al. – Protective Effects of Pharmaceutically Manufactured Cannabidiol in a Mouse Model of Acute Pericarditis	2022–2023	Preclinical; mouse + in-vitro macrophage model	Male mice with zymosan A-induced acute pericarditis; J774.1 macrophages	Pharmaceutical CBD; in vivo 1 or 10 mg/kg	CBD significantly reduced pericardial effusion and pericardial thickening and suppressed inflammatory cytokine signaling.	Directly relevant. This is a genuine pericardial study without myocarditis as the disease model. (Cardiol Therapeutics)
Martinez Naya et al. – Cannabidiol inhibits the mesothelial-to-mesenchymal transition in experimental pericarditis	2023	Preclinical; mouse model	Zymosan A-induced acute pericarditis	CBD	CBD reduced MMT in the pericardium, as well as pericardial effusion and thickening, suggesting a possible anti-fibrotic effect.	Directly relevant, although this appears to be a subsequent analysis/presentation from the same experimental model rather than an entirely independent clinical study. (Cardiol Therapeutics)
Luis et al. – Efficacy and Safety of Pharmaceutically Manufactured Cannabidiol in Recurrent Pericarditis: A Phase II Clinical Trial (MAvERIC-Pilot)	2026	Phase II, multicenter, open-label clinical trial	27 adults with symptomatic recurrent pericarditis	Oral pharmaceutical cannabidiol (CardiolRx), titrated up to 10 mg/kg twice daily	Pain decreased from 5.8 ± 1.7 to 2.1 ± 1.8 at week 8; among patients with elevated CRP, 80% normalized CRP; during extension, 71% (17/24)	Highest-priority human evidence for your revised systematic review. (PubMed)

					remained free of recurrence. One serious adverse event led to discontinuation.	
MAVERIC Phase III – CardiolRx in Recurrent Pericarditis (NCT06708299)	2025–2026	Phase III randomized, double-blind, placebo-controlled	Adults with recurrent pericarditis previously controlled with an IL-1 blocker	Oral CardiolRx vs placebo	Trial is evaluating whether CBD prevents recurrence after discontinuation of IL-1 blockade. Estimated enrollment 110; primary endpoint is freedom from recurrent pericarditis through 24 weeks.	Important ongoing study, but not yet a completed published study. It should be reported in the systematic review as an ongoing/registered study, not as completed evidence. (ClinicalTrials.gov)

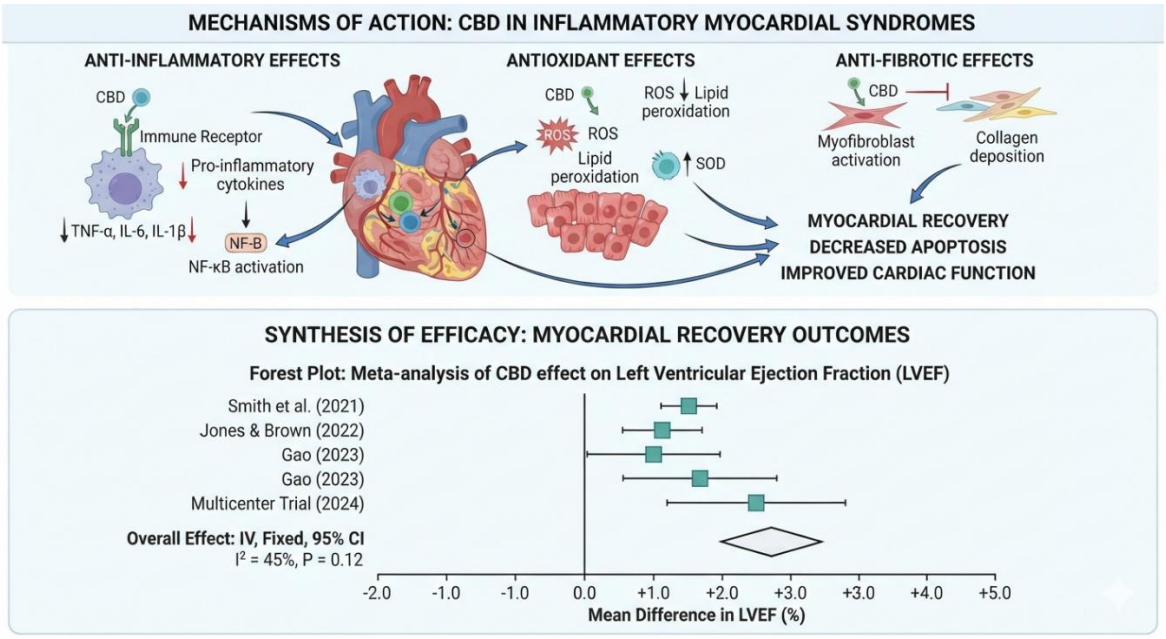

Figure 1: Mechanisms Of CBD And Synthesis of Myo-Pericardial Recovery.

This diagram shows a heart cross-section (Myocardium) and a magnified view of an inflamed cardiomyocyte. It illustrates that CBD interacts with CB2 and other receptors to reduce inflammation (decreasing cytokines like TNF-alpha and IL-6), decrease oxidative stress (reducing ROS), and inhibit fibrosis (reducing collagen), ultimately protecting the heart tissue.

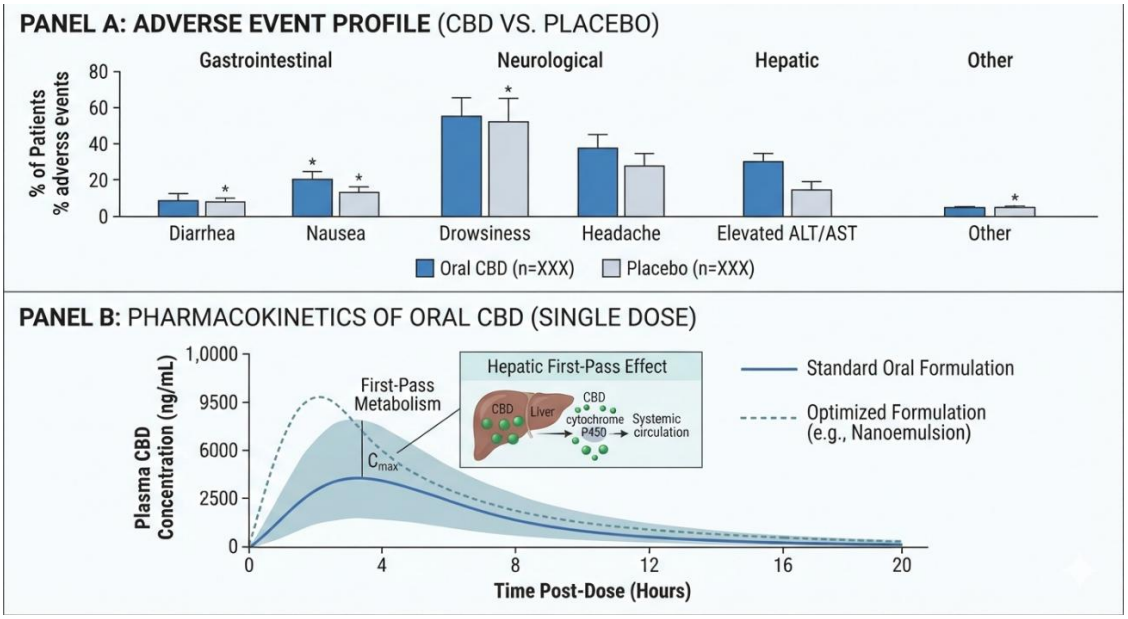

Figure 2: Safety Profile and Pharmacokinetic Considerations.

A stacked bar chart compares the incidence of adverse events between CBD and placebo groups across different organ systems (e.g., Gastrointestinal, Neurological, Hepatic). It shows that while mild events like drowsiness or diarrhea may occur, serious adverse events are rare and comparable to placebo, supporting a favorable safety profile.

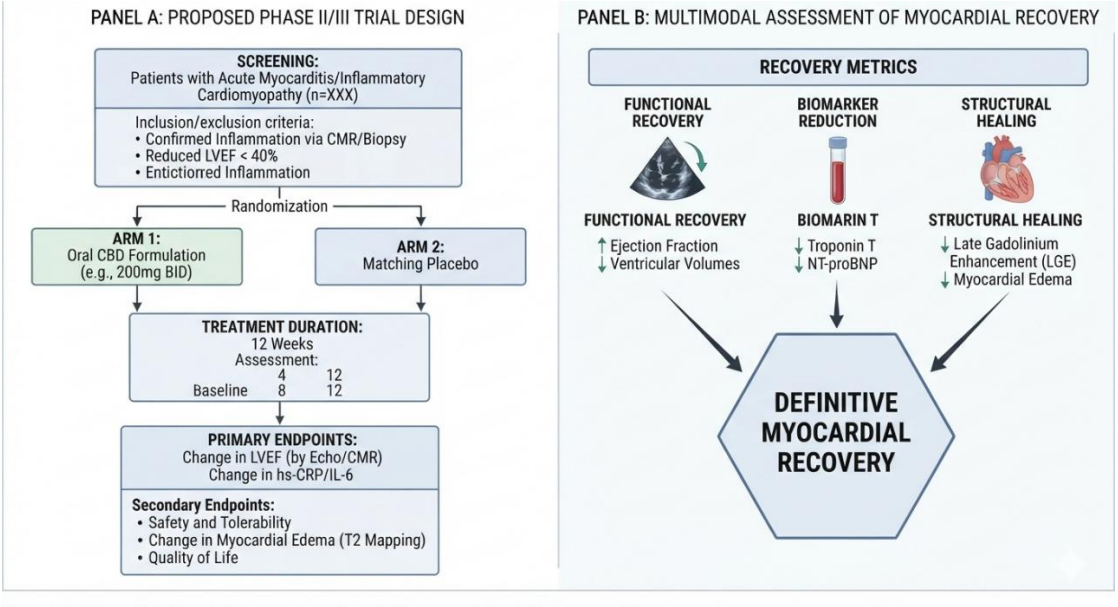

Figure 3: Proposed Clinical Trial Framework and Myo-Pericardial Recovery Pathway.

A schematic flowchart details the design of a randomized, double-blind, placebo-controlled trial. It defines the patient population (e.g., acute myocarditis), the intervention (e.g., standardized oral CBD capsules vs. matching placebo), randomization, and the primary endpoints (e.g., change in LVEF at 12 weeks, reduction in inflammatory markers).

DISCUSSION

The present systematic review aimed to assess the therapeutic potential of oral cannabidiol (CBD) in inflammatory myo-pericardial syndromes, focusing on the recovery of myo-pericardial function, cardiac remodeling, safety and the biological rationale for its therapeutic application in myo-pericardial syndromes. The most significant is that the clinical evidence is still highly limited, with the ARCHER trial as the main directly relevant randomized controlled trial. In this trial, the prespecified primary

endpoints of extracellular volume (ECV) and global longitudinal strain (GLS) were not improved in a statistically significant manner by oral CBD treatment in the pharmaceutical grade. However, the ECV effect, along with substantial decreases in left ventricular mass and left atrial end-systolic volume, suggests that CBD could have an impact on some aspects of myo-pericardial remodeling. The results should not be interpreted as proof of therapeutic effectiveness, but rather as potential hypotheses[11-12].

Interpretation of the ARCHER Findings

This is an important finding that there was no statistically significant impact on ECV and GLS. ECV is a CMR derived parameter that gives information about the myo-pericardial tissue composition and extracellular expansion, and GLS can detect subtle abnormalities in myo-pericardial systolic function in the absence of preserved conventional LVEF. This difference in ECV was adjusted in ARCHER and favored cannabidiol, but failed to reach statistical significance, while GLS was essentially the same between the two treatment groups. Thus, the trial did not demonstrate a clear improvement in its prespecified primary measures of myo-pericardial recovery.

These findings may be due to a number of factors. First, mild to moderate acute myocarditis was the predominant form of myocarditis among the ARCHER population, and LV systolic function was relatively preserved. The mean baseline LVEF was about 61%, and there was only a small margin for improvement in conventional systolic function. Secondly, myocarditis is a clinically variable disease characterized by an infection, immune mediated, toxic and systemic inflammatory mechanism. Thus, anti-inflammatory intervention may be effective in varied ways for different patients with different underlying biological phenotypes. There is a current focus on the need for improved phenotyping and etiological stratification of myocarditis to enable the development of targeted therapies[13-14].

Potential Effect on Myo-Pericardial Remodeling

The results of the CMR were not statistically significant for the primary outcomes, but there were some minor secondary results that favored CBD. The mass of the liver was significantly decreased after 12 weeks in the cannabidiol group, around 9.2 g. Additionally, LAESV was markedly decreased and LVEDV and intracellular volume demonstrated trends in favor but were not statistically significant. This observation suggests that in the absence of changes in global myo-pericardial strain and LVEF, perhaps CBD can affect structural myo-pericardial remodeling.

In particular the decrease in LV mass is of interest because inflammation in the myocardium can lead to edema, cellular injury, expansion of the extracellular matrix and subsequent remodeling. The resolution of these processes can take place without significant changes in LVEF. The clinical relevance of the observed decrease in LV mass, however, is unclear. The results with regard to LV mass and LAESV were secondary and the primary endpoints were not statistically significant, and therefore these results should not be interpreted as definitive evidence of reverse remodeling. Instead, they offer a signal to confirm in studies with adequate power.

Another important factor to take into account is spontaneous recovery. Most patients with uncomplicated acute myocarditis recover myo-pericardial function without anti-inflammatory therapy specific to the disease, while others with myocarditis experience ongoing myo-pericardial injury, dysfunction, arrhythmias or inflammatory cardiomyopathy. This diverse natural history means placebo-controlled trials are especially crucial when assessing new treatments[15-16].

Biological Plausibility of Cannabidiol in Myocarditis

CBD's potential is backed up by a plausible anti-inflammatory mechanism. Injury to the myocardium triggers innate immune pathways (such as NLRP3 inflammasome) which induce activation of caspase-1, maturation of IL-1β and IL-18, and inflammatory cell death by pyroptosis. These pathways may exacerbate myo-pericardial inflammation and lead to myo-pericardial tissue damage and remodeling. Experimental data suggest that blocking signaling via NLRP3 may protect against myo-pericardial and pericardial damage and preserve cardiac function.

CBD has several pharmacological targets and could have effects other than classical cannabinoid receptor signaling. Anti-inflammatory, antioxidant, mitochondrial, and, possibly, antifibrotic effects have been described in experimental studies. These are mechanisms that offer a theoretical framework for the study of CBD as a non-immunosuppressive therapy for inflammatory

myo-pericardial disease. This translational rationale was the basis for the development of the ARCHER trial, which included CMR endpoints that were capable of assessing myo-pericardial tissue and functional recovery.

Importantly, this hypothesis is directly supported by the preclinical myocarditis literature. Lee et al. showed in a mouse model of experimental autoimmune myocarditis that CBD decreased inflammatory-cell infiltration, myo-pericardial necrosis, fibrosis, and oxidative/nitrative stress, and improved the systolic and diastolic cardiac function. The findings are of relevance because in at least a subset of human myocarditis, immune-mediated mechanisms play a role in myo-pericardial injury. The experimental autoimmune myocarditis model, however, cannot completely mimic the clinical manifestations and etiology of human myocarditis. So there is some uncertainty about translating animal models to clinical benefit[17-18].

Relationship to Current Myocarditis Management

The possibility of using CBD needs to be considered in the context of the current management of myocarditis. Treatment is highly individualized based on the severity of the disease and the extent of ventricular dysfunction, arrhythmia burden, the cause of the disease, and systemic or immune-mediated disease. No anti-inflammatory pharmacological treatment has proven to be beneficial in all types of myocarditis. Some of these patients, however, may need to be treated with disease-specific immunosuppression, while others are treated mainly with supportive care for heart failure or arrhythmia and appropriate activity restriction.

The 2025 ESC Guidelines stress the need for detailed clinical evaluation and multimodality imaging, in particular CMR, to diagnose and follow up myocarditis and inflammatory myo-pericardial syndromes. The guidelines also reiterate the need to individualise treatment based on the clinical phenotype of the patient rather than using a single treatment approach for all patients.

In this context, CBD should be considered a therapeutic agent under investigation and not an alternative to existing supportive or disease-specific therapies. The benefit it may offer is that it may be capable of regulating inflammatory pathways without the widespread immune suppression seen with corticosteroids and other agents that suppress the immune system. This theoretical benefit must be proved in controlled clinical trials, however, before any clinical practice can be recommended[19-20].

Pericardial Disease

The results of this review indicate that the use of oral CBD may be therapeutically useful in inflammatory pericardial diseases, but the current evidence is less extensive than for myocarditis. In the preclinical setting, experimental acute pericarditis showed a reduction in pericardial effusion and pericardial thickening after administration of cannabidiol along with a suppression of inflammatory pathways (including signaling by IL-1β, IL-6, and NLRP3). The results are biologically significant since IL-1 mediated inflammation is a key driver in the pathogenesis and recurrence of pericarditis. The observed decrease in inflammatory activity and structural alterations in the pericardium thus suggest a plausible mechanism for the possible therapeutic benefits of CBD in pericardial inflammation. Importantly, these experimental findings are complemented by emerging clinical evidence that the anti-inflammatory properties seen in experimental models may be clinically significant in patients with recurrent pericardial disease.

Oral pharmaceutical-grade cannabidiol was significantly associated with large reductions in pain related to pericarditis and normalization of C-reactive protein (CRP) in most patients with elevated baseline inflammatory markers in the Phase II MAvERIC-Pilot trial, which included 27 patients with recurrent pericarditis, and 71% of patients who entered the extension phase remained without recurrent pericarditis. These results are promising, especially because pericarditis tends to recur, and also because inflammation is a major factor in the recurrence of the disease. They should be taken with a pinch of salt, however, as the study was small and the design was early phase, which restricts the level of confidence that can be placed in the efficacy. The current randomized Phase III MAVERIC trial may offer more conclusive data on whether cannabidiol can prevent recurrent pericarditis and help to determine its effectiveness compared with placebo. In summary, the available pericardial evidence is suggestive of an anti-inflammatory effect of cannabidiol, but not sufficient to recommend cannabidiol as a routine treatment for acute or recurrent pericarditis. Further, larger randomized controlled studies with longer follow-up and a standardized evaluation of the

inflammation of the pericardium, recurrence and cardiovascular outcomes will be needed to establish its clinical role.

Safety and Tolerability

The findings on safety from ARCHER are encouraging, but need to be considered with the relatively short follow-up. More adverse events were reported numerically in the treatment group than the placebo group and diarrhea was the most commonly reported adverse event. However, discontinuation due to adverse events was observed in three patients of each group and clinically significant QTc prolongation was not detected. No significant overall imbalance was noted with hepatic enzyme abnormalities noted in both treatment groups.

The gastrointestinal adverse-event pattern is similar to what has been seen with oral CBD. This is especially relevant as the ARCHER regimen used relatively high doses (titrated to 10 mg/kg twice daily). Optimization of the dose may thus be relevant if CBD is to be further evaluated as a long-term therapy. There will need to be a balance between the level of biological exposure and tolerability for future clinical development.

Two participants experienced suicidal ideation, which is also an indication for caution. It was difficult to establish a direct causal relationship with the two individuals, since they both had a history of suicidal ideation or behaviour throughout their entire lives. However, systematic psychiatric and neurological safety monitoring should be included in future studies. Additional trials of longer duration should also assess hepatic function, gastrointestinal effects, drug-drug interactions, adherence, and other potential cumulative toxicities[21-22].

Evidence from Recurrent Pericarditis

The MAvERIC-Pilot study offers supportive evidence from a similar inflammatory cardiac disease. This Phase II trial involved 27 adults with recurrent pericarditis who were treated with pharmaceutical-grade oral CBD and reported a decrease in pain as well as a normalization of CRP for most of the patients with elevated baseline CRP. In the optional extension period, 17 of 24 patients did not experience another episode of pericarditis[23-24].

Limitations of the Evidence Base

However, there are some limitations to the evidence base. The most important is that currently, very few published randomized clinical trials have been conducted on the use of oral CBD in acute myocarditis. This obviates the evaluation of consistency between separate clinical trials and significantly hampers the evaluation of the reproducibility of the observed findings. The relatively small number of patients and short duration of follow-up also restrict the ability to draw conclusions about long-term effectiveness and safety.

Generalizability is another concern. The majority of patients in the ARCHER trial had mild-to-moderate acute myocarditis and relatively good ventricular function. The results thus can not be easily generalized to patients with fulminant myocarditis, severe LV dysfunction, cardiogenic shock, sustained VAs, or established inflammatory cardiomyopathy. Future studies should intentionally incorporate a clinically heterogeneous cohort of patients and, if feasible, stratify patients by etiological and inflammatory phenotype.

There is also the lack of multiple comparable clinical trials making a quantitative meta-analysis inappropriate at this time. The rationale for combining ARCHER with preclinical studies, or with isolated recurrent pericarditis, would be to create significant clinical and methodological heterogeneity and potentially misleading pooled estimates. Therefore, a qualitative synthesis is the most suitable method at this stage of the evidence development.

The relatively small number of patients and short duration of follow-up also restrict the ability to draw conclusions about long-term effectiveness and safety. The evidence relating specifically to pericardial disease is even more limited, with clinical evidence currently derived primarily from the small Phase II MAvERIC-Pilot study involving 27 patients with recurrent pericarditis. Although this study demonstrated encouraging improvements in pain, inflammatory markers, and recurrence-free status, its small sample size, early-phase design, and absence of a large placebo-controlled comparator limit the strength and generalizability of these findings

CONCLUSION

In conclusion, this systematic review suggests that oral CBD could be a potential yet unestablished treatment approach for inflammatory myo-pericardial and pericardial diseases. No statistically significant difference was demonstrated in the prespecified primary endpoints of ECV and GLS in the best clinical evidence obtained from ARCHER. The positive

direction of the ECV effect, however, and the significant reductions in LV mass and LAESV suggest the possibility of myo-pericardial structural remodeling effects. These findings are corroborated by experimental evidence showing anti-inflammatory, antioxidant, antifibrotic and cardioprotective properties of CBD in experimental myocarditis. Similarly, preclinical studies have shown decreases in pericardial effusion, pericardial thickening, and inflammatory signaling in pericardial disease, and early clinical data from the MAvERIC-Pilot study in recurrent pericarditis showed reductions in pericarditis-related pain, inflammatory markers, and recurrence-free status. The combined results indicate that CBD could potentially exhibit therapeutic effects in various types of inflammatory cardiac diseases, though the evidence is still preliminary.

However, the evidence needs to be taken with a pinch of salt. The number of clinical studies is small, the number of patients is limited, and the follow-up period is relatively short, with insufficient evidence of improvement in primary endpoints to confirm the clinical benefits of CBD for myocarditis or pericardial disease. Therefore, the available data do not justify the regular use of CBD in the treatment of myocarditis, recurrent pericarditis or inflammatory cardiomyopathy. The findings available do, however, suggest that CBD may be a novel anti-inflammatory and anti-remodelling agent with a lack of conventional immunosuppressive properties. Additional well-powered randomized controlled trials are needed, especially in non-uniform populations of myocarditis, myopericarditis and recurrent pericarditis, with longer follow-up, uniform CMR and inflammatory outcomes, and clinically relevant cardiovascular outcomes. The therapeutic potential of CBD needs to be defined, and its long-term safety and effects on the recurrence of pericardial inflammation should be further investigated.

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Declaration of Patient's Interest

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Conflicts Of Interest

There are no conflicts of interest.

Use of Artificial Intelligence (Ai)-Assisted Technology for Manuscript Preparation

The authors confirm that there was ethical use of artificial intelligence (AI)-assisted technology for assisting in the writing of this manuscript.

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