



Perioperative and Surgical Management of Constrictive Pericarditis

REVIEW

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ABSTRACT

Constrictive pericarditis represents a form of severe diastolic heart failure characterized by impaired diastolic filling due to a rigid, noncompliant pericardium. Surgical pericardiectomy is the definitive treatment for chronic or refractory subacute constrictive pericarditis; however, outcomes vary widely depending on disease etiology, chronicity, myocardial involvement, end-organ dysfunction, and surgical technique. This review summarizes contemporary best practices for the surgical management of constrictive pericarditis, with emphasis on patient selection, timing of intervention, operative approach, and perioperative considerations.

Accurate differentiation between inflammatory, transient constrictive phenotypes and irreversible fibrotic disease is central to therapeutic decision-making. Etiology-based risk stratification, assessment of hepatic and renal dysfunction, and careful evaluation of myocardial involvement provide critical prognostic information. Multimodality imaging plays a central role in diagnosis, surgical planning, and identification of patients most likely to benefit from intervention. Radical pericardiectomy is associated with superior functional recovery compared with partial resection, whereas radiation-associated disease, mixed constrictive–restrictive physiology, and advanced end-organ dysfunction are consistently linked to worse outcomes. High-volume centers of excellence in pericardial disease are critical in optimizing results after radical pericardiectomy.

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INTRODUCTION

Constrictive pericarditis (CP) is characterized by impaired diastolic filling due to a rigid, noncompliant pericardium that limits ventricular expansion and produces heart failure physiology despite preserved systolic function.¹ CP can arise from either irreversible pericardial fibrosis and/or calcification, termed chronic constrictive pericarditis, or from potentially reversible pericardial inflammation, termed subacute constrictive pericarditis. Distinguishing these entities is essential because chronic CP typically requires surgical pericardiectomy, whereas, subacute CP can have transient constrictive phenotype that may resolve with anti-inflammatory therapy.^{2,3} Another important entity to highlight is effusive-constrictive pericarditis (ECP)—a phenotype in which constrictive physiology persists after removal of pericardial fluid, historically identified by persistent elevation of right atrial or pericardial pressures and now more commonly recognized by echocardiographic features following pericardiocentesis.^{4,5}

Etiologies of constrictive pericarditis vary geographically and strongly influence disease severity and operative risk. Tuberculosis remains the leading cause in endemic regions, whereas idiopathic disease, prior cardiac surgery, and mediastinal radiation predominate in nonendemic settings.³ Delayed referral is particularly consequential in these high-risk etiologies, as prolonged constriction leads to hepatic congestion, renal dysfunction, malnutrition, and myocardial atrophy. These factors adversely affect surgical outcomes yet are often attributed to advanced disease stage rather than operative risk at high-volume centers. Since the first successful pericardiectomies in the early 20th century, outcomes have improved substantially with advances in imaging, perioperative care, and standardized radical pericardiectomy techniques.^{2,6,7}

Despite these advances, significant variability persists in diagnostic pathways, timing of referral, and operative approach across institutions. This review aims to summarize current best practices for the surgical management of CP, focusing on patient selection, timing of intervention, operative techniques, and postoperative care.

PATHOPHYSIOLOGY AND ETIOLOGY RELEVANT TO SURGERY

The pericardium is a double-walled sac composed of an outer fibrous pericardium and an inner serous pericardium, the latter divided into parietal and visceral (epicardial) layers.^{8,9} The fibrous pericardium anchors the heart to surrounding structures and limits acute cardiac distension.^{10,11} Pericardial reflections around the pulmonary veins form the transverse

and oblique sinuses, which are technically challenging regions during pericardiectomy.^{7,9} The phrenic nerves, which course laterally along the pericardial surfaces, must be identified and preserved to prevent diaphragmatic dysfunction.^{2,9} The right coronary artery and right ventricular free wall lie immediately beneath the anterior pericardium and may be injured in calcific or adherent disease. Mobilization of the posterior pericardium over the left atrium and pulmonary veins is often the most challenging aspect of surgery due to fibrosis or calcification.¹²

CP results from varying degrees of pericardial inflammation, fibrosis, and calcification that progressively reduce pericardial compliance.¹³ Constrictive physiology is defined by two interrelated hemodynamic mechanisms: dissociation between intrathoracic and intracardiac pressures and exaggerated ventricular interdependence. Reduced transmission of intrathoracic pressure to the cardiac chambers during inspiration decreases left ventricular filling while augmenting right ventricular filling, resulting in discordant respiratory changes in ventricular stroke volumes and characteristic Doppler findings.^{1,14} CP comprises distinct clinical subtypes, including transient constrictive pericarditis, effusive-constrictive pericarditis, and chronic fibrotic constrictive pericarditis, each with different underlying mechanisms and therapeutic and surgical implications. Transient constrictive pericarditis is driven by active pericardial inflammation and edema and usually resolves with anti-inflammatory therapy.^{15,16} ECP represents an important clinical entity in which constrictive physiology persists despite removal of pericardial fluid, reflecting involvement of the visceral pericardium. Although pericardiocentesis may provide transient symptomatic relief, persistent constriction often necessitates surgical pericardiectomy, with outcomes influenced by the extent of visceral pericardial and myocardial involvement.^{4,17,18}

CP develops infrequently after acute pericarditis. In a prospective cohort of 500 patients followed for a median of 6 years, Imazio et al. reported an overall CP incidence of 1.8%, with an extremely low risk in idiopathic or viral etiologies (0.48%) and a substantially higher risk in non-idiopathic forms (8.3%) such as neoplastic, post-pericardial injury, or bacterial pericarditis.¹⁹ Globally, tuberculosis remains the leading cause of CP in endemic regions.^{16,20,21} In contrast, most cases in North American and European countries are idiopathic or related to prior cardiac surgery or mediastinal radiation.¹⁷ Radiation-associated disease represents a particularly high-risk surgical substrate, often involving visceral constriction and concomitant myocardial, valvular, and mediastinal pathology.^{16,17,22} These etiology specific differences in disease substrate translate into marked differences in operative risk and long-term outcomes following pericardiectomy, as summarized in [Figure 1](#).

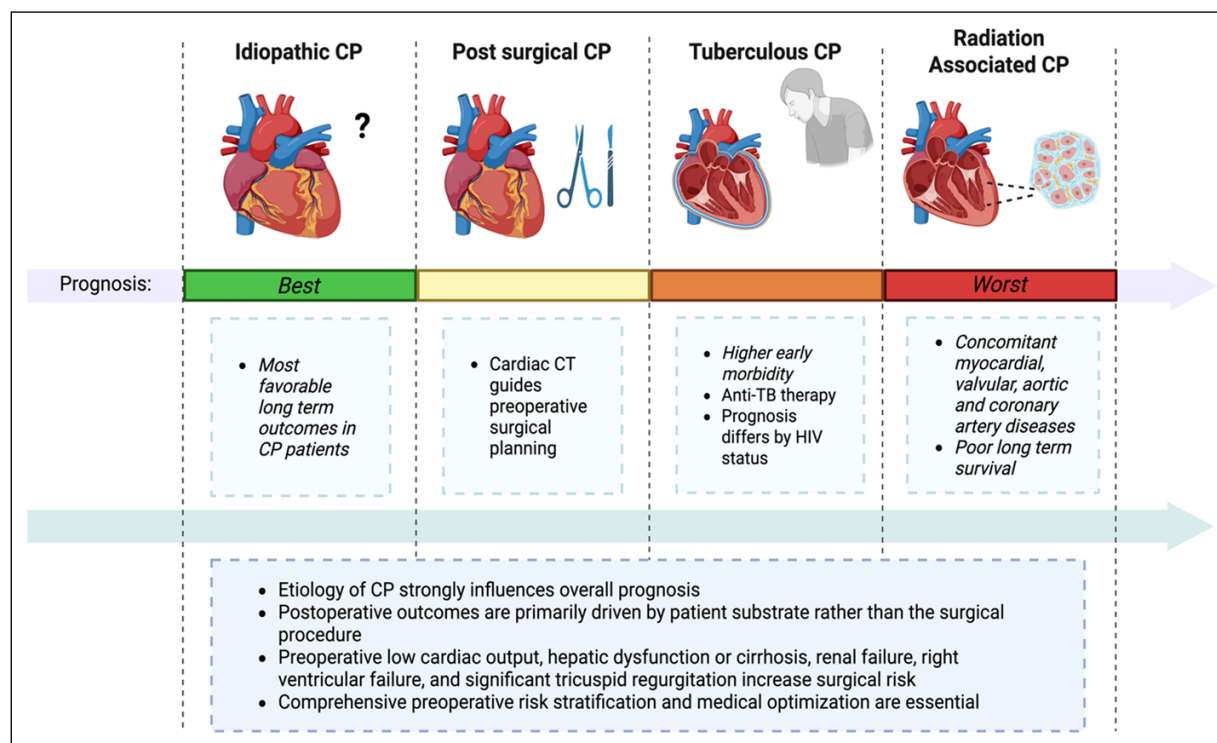


Figure 1 Etiology-based risk stratification and outcomes after pericardiectomy for constrictive pericarditis. (Created in BioRender with permission; Harake L, 2026; <https://BioRender.com/lI02ak0>). CP: constrictive pericarditis; CT: computed tomography, TB: tuberculosis

PREOPERATIVE EVALUATION: CLINICAL AND MULTIMODALITY IMAGING

Careful clinical assessment combined with multimodality imaging and comprehensive hemodynamic evaluation is central to the diagnosis, phenotyping, and preoperative assessment of CP. The differential diagnosis includes restrictive cardiomyopathy (RCM), severe tricuspid regurgitation, pulmonary arterial hypertension, and noncardiac causes of systemic congestion such as intrinsic liver disease.

CONFIRMING CONSTRICTIVE PHYSIOLOGY

Multimodality imaging forms the cornerstone of diagnostic confirmation and therapeutic decision-making in CP.²³ The two defining hemodynamic hallmarks of constriction, dissociation between intrathoracic and intracardiac pressures and exaggerated ventricular interdependence, are most readily identified on transthoracic echocardiography through assessment of ventricular filling and interaction.¹ The Mayo Clinic echocardiographic criteria define constrictive physiology using four core features: respirophasic septal shift, exaggerated respiratory variation in mitral inflow, expiratory hepatic vein diastolic flow reversal, and preserved or increased medial mitral annular e' velocity. High diagnostic accuracy is achieved when septal shift is present with either medial $e' \geq 9$ cm/s or a hepatic vein

expiratory diastolic reversal ratio ≥ 0.79 .²⁴ Importantly, not all criteria must be present, and diagnosis should be made in the appropriate clinical context.²⁵

Speckle-tracking strain imaging provides complementary information and can increase diagnostic sensitivity.^{14,15,26} CP is typically associated with preserved global longitudinal strain and a characteristic regional pattern of reduced lateral wall strain with relative preservation of medial strain, reflecting tethering of the left ventricular free wall by the constrictive pericardium.²⁷ Improvement in tissue Doppler velocities and longitudinal strain over serial examinations correlates with clinical recovery in transient inflammatory CP during anti-inflammatory therapy, providing a useful tool for longitudinal assessment.

COMPUTED TOMOGRAPHY FOR ANATOMIC DEFINITION AND SURGICAL PLANNING

Cardiac computed tomography (CT) provides high-resolution assessment of pericardial anatomy and is particularly useful for surgical planning. CT can identify pericardial thickening and/or calcification if present. In surgically confirmed cohorts, pericardial calcification is present in approximately one-third of patients, while pericardial thickening is more common.¹³ Beyond diagnosis, CT delineates the distribution of dense calcification and defines the relationship between the pericardium, coronary arteries, bypass grafts, and adjacent mediastinal

structures, information that is essential when planning pericardiectomy, particularly in patients with prior cardiac surgery or extensive calcium burden.^{7,23}

CARDIAC MAGNETIC RESONANCE IMAGING FOR PHENOTYPING AND TIMING

Cardiac magnetic resonance (CMR) imaging provides comprehensive evaluation of pericardial structure, inflammation, and functional consequences and plays a key role in guiding therapeutic strategy.²⁸ While echocardiography often provides a more complete hemodynamic assessment, CMR uniquely characterizes pericardial tissue and helps distinguish inflammatory from fibrotic disease. T2-weighted imaging identifies pericardial edema, reflecting active inflammation, whereas late gadolinium enhancement, particularly with phase-sensitive inversion recovery and fat suppression, highlights increased pericardial vascularity associated with inflammation

and neovascularization. Quantitative assessment of pericardial LGE has prognostic significance; greater degrees of delayed hyperenhancement independently predict clinical improvement with anti-inflammatory therapy and provide incremental discrimination beyond symptoms and inflammatory markers.^{29,30} These features allow CMR to identify an inflammatory constrictive phenotype and guide the timing of surgery versus continued medical therapy. A representative case illustrating inflammatory and fibrotic substrates relevant to surgical decision-making is shown in [Figure 2](#).

INVASIVE HEMODYNAMIC ASSESSMENT

Invasive hemodynamic assessment is reserved for cases in which noninvasive evaluation is equivocal or discordant. Although historically considered the diagnostic gold standard, catheterization findings are load dependent and operator sensitive. Classic features include elevated

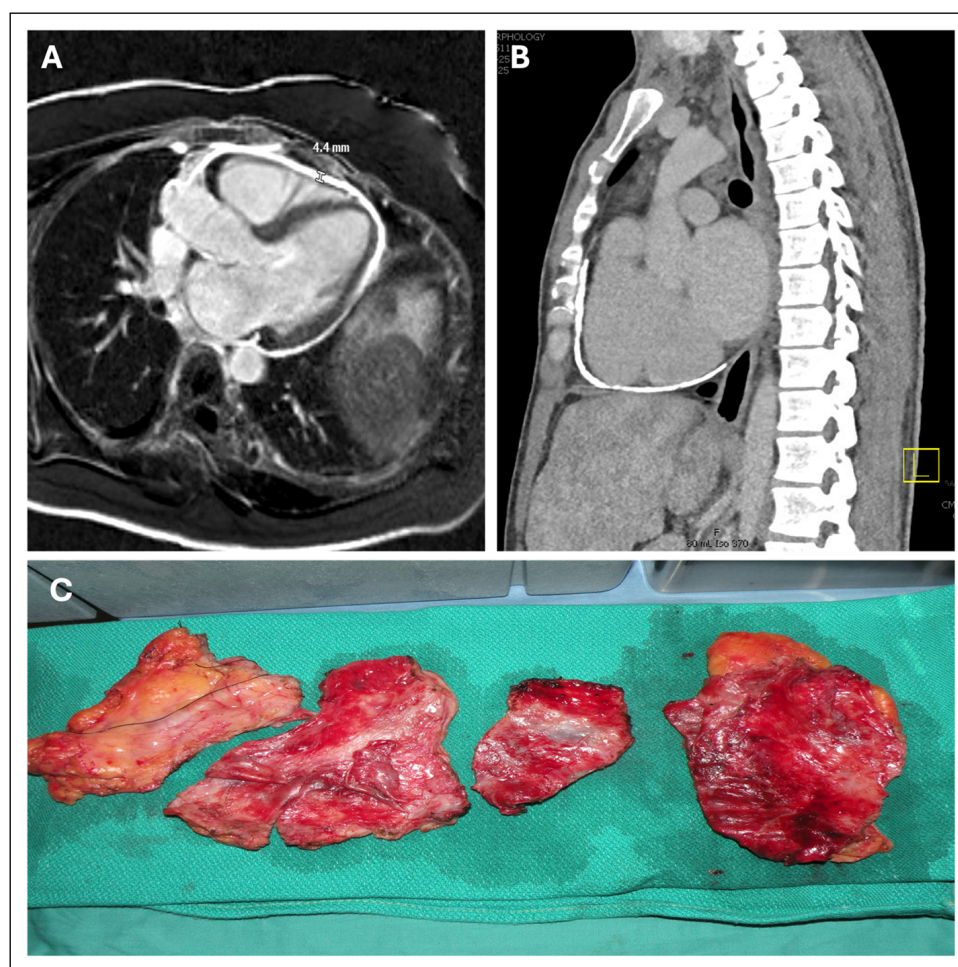


Figure 2 Representative imaging and pathologic substrates guiding surgical management of constrictive pericarditis. **(A)** Cardiac magnetic resonance imaging using phase-sensitive inversion recovery demonstrating pericardial late gadolinium enhancement with increased pericardial thickness, consistent with an inflammatory constrictive phenotype that may be potentially reversible with medical therapy. **(B)** Cardiac computed tomography showing severe circumferential “eggshell” pericardial calcification characteristic of chronic fibrotic constriction. **(C)** Gross surgical specimen following pericardiectomy demonstrating markedly thickened and fibrotic pericardium.

and near-equalized diastolic filling pressures, a prominent right atrial y descent, and a ventricular dip-and-plateau pattern; however, these findings lack specificity and may also be observed in restrictive cardiomyopathy, and many contemporary patients with constrictive pericarditis have concomitant pulmonary hypertension.^{31,32}

Contemporary invasive assessment relies on dynamic respiratory indices, with dissociation of intrathoracic and intracardiac pressures demonstrated by a ≥ 5 mm Hg respiratory gradient between pulmonary capillary wedge and left ventricular pressures and enhanced ventricular interdependence identified by discordant respiratory variation in simultaneous right and left ventricular pressure waveforms; a systolic area index ≥ 1.1 further supports constrictive pericarditis.^{33,34}

DISTINGUISHING CP FROM SEVERE TR AND RCM

In most patients, transthoracic echocardiography is sufficient to distinguish constrictive pericarditis from severe tricuspid regurgitation or restrictive cardiomyopathy.^{35,36} Hepatic vein Doppler is central to this distinction: expiratory diastolic flow reversal supports constriction, whereas inspiratory reversal reflects impaired right ventricular compliance as seen in restrictive cardiomyopathy.¹ RCM is

further characterized by markedly reduced mitral annular e' velocities, biatrial enlargement, pulmonary hypertension, and ventricular concordance on simultaneous biventricular pressure recordings, while severe tricuspid regurgitation is suggested by right ventricular and annular dilation, systolic hepatic vein flow reversal, and prominent c-v waves on right atrial tracings.³⁵ When these entities coexist, most commonly in radiation-associated heart disease, a multimodality approach is required to identify the dominant physiology, as patients with substantial restrictive myocardial involvement derive limited hemodynamic or symptomatic benefit from pericardiectomy. The integrated role of clinical assessment, multimodality imaging, and invasive hemodynamics in preoperative evaluation is summarized in [Table 1](#).

RISK STRATIFICATION AND PREOPERATIVE OPTIMIZATION

Risk stratification in CP is essential to identify patients most likely to benefit from pericardiectomy and to minimize perioperative morbidity and mortality. Importantly, surgical risk is driven primarily by disease etiology, chronicity, end-

MODALITY	KEY FINDINGS IN CONSTRICTIVE PERICARDITIS	DIAGNOSTIC VALUE	ROLE IN SURGICAL DECISION-MAKING
Clinical Assessment	Right-sided heart failure, systemic congestion, preserved systolic function	Establishes pretest probability and excludes noncardiac causes of congestion	Identifies disease severity, comorbidities, and operative risk
Transthoracic Echocardiography	Respirophasic septal shift, exaggerated respiratory variation in mitral inflow, expiratory hepatic vein diastolic flow reversal, preserved or increased medial mitral annular e' velocity	Cornerstone for confirming constrictive physiology using Mayo Clinic criteria	Differentiates CP from RCM and severe TR and guides need for further testing
Speckle-Tracking Strain Imaging	Preserved global longitudinal strain with reduced lateral wall strain and relative medial preservation	Improves diagnostic sensitivity and phenotyping	Serial improvement supports reversibility in inflammatory or transient CP
Cardiac Computed Tomography	Pericardial thickening or calcification, distribution of calcium, relationship to coronary arteries and bypass grafts	Defines anatomic extent of disease	Essential for surgical planning and risk stratification
Cardiac Magnetic Resonance Imaging	Pericardial edema on T2 imaging, late gadolinium enhancement indicating inflammation, quantitative LGE burden	Differentiates inflammatory from fibrotic constriction	Guides timing of surgery versus continued medical therapy
Invasive Hemodynamic Assessment	Equalized diastolic pressures, dip-and-plateau pattern, respiratory dissociation of intracardiac pressures, ventricular discordance, systolic area index ≥ 1.1	Reserved for equivocal or discordant noninvasive findings	Confirms constriction when diagnosis remains uncertain
Differentiation from Alternative Diagnoses (RCM and Severe TR)	RCM: low mitral annular e' velocities, biatrial enlargement, pulmonary hypertension, ventricular concordance. Severe TR: right ventricular and annular dilation, systolic hepatic vein flow reversal, prominent c-v waves	Distinguishes myocardial or valvular causes of systemic congestion from pericardial constriction	Identifies patients unlikely to benefit from pericardiectomy and avoids inappropriate surgery

Table 1 Preoperative evaluation of constrictive pericarditis using clinical assessment, multimodality imaging, and invasive hemodynamics to confirm constrictive physiology, guide surgical timing, and distinguish alternative causes of systemic congestion. RCM: restrictive cardiomyopathy; TR: tricuspid regurgitation; CP: constrictive pericarditis; LGE: late gadolinium enhancement

organ dysfunction, and myocardial involvement rather than the operation itself.^{1,2} Certain clinical phenotypes are associated with limited benefit from pericardiectomy and warrant individualized evaluation at centers of excellence.

ETIOLOGY BASED RISK

Etiology is a dominant determinant of operative risk and long-term outcomes following pericardiectomy. Idiopathic constrictive pericarditis consistently carries the most favorable prognosis, whereas post-cardiac surgery disease confers intermediate risk due to distorted anatomy and dense adhesions. Tuberculous constrictive pericarditis is associated with high early mortality and adverse outcomes, particularly in TB-endemic and immunosuppressed populations.^{20,37} Overall mortality for tuberculous pericarditis reaches 17% to 40% at 6 months after diagnosis, with operative risk strongly influenced by disease chronicity and extent of visceral pericardial involvement, underscoring the importance of early recognition and referral.^{17,38} Radiation-associated constrictive pericarditis is associated with the highest perioperative mortality and poorest long-term survival, reflecting frequent concomitant myocardial, valvular, coronary, and mediastinal involvement.^{7,39,40}

In a large observational cohort of 601 patients, in-hospital mortality varied markedly by etiology at 1.1% in idiopathic disease, 9.7% in post-surgical constriction, and 27% in radiation-associated disease, with corresponding declines in long-term survival.⁷ Contemporary series of radiation-associated constriction continue to demonstrate early operative mortality near 10% and poor long-term survival despite modern surgical techniques, often in the setting of incomplete resection and need for concomitant procedures.^{16,40} These findings underscore the importance of early referral and management at high-volume centers with expertise in pericardial disease, where operative outcomes have improved over time.⁹

RISK SCORES AND END-ORGAN DYSFUNCTION

No pericardiectomy-specific risk score exists; however, liver-based scoring systems provide robust prognostic information in constrictive pericarditis given the predominance of right-sided heart failure and congestive hepatopathy. Higher preoperative MELD and MELD-XI scores are independently associated with increased postoperative mortality and morbidity, with MELD-XI tertiles demonstrating a stepwise rise in 90-day mortality of approximately 3%, 8%, and 16% as well as higher rates of renal failure, transfusion, and chest tube output.⁴¹ MELD-XI performed as well as or better than MELD and offers a practical INR-independent tool for risk stratification.

The Child-Pugh score similarly predicts long-term outcomes. Patients with Child-Pugh class B or C have

substantially worse 5-year survival compared with class A patients, approximately 38% versus 81%, and a score ≥ 7 remains an independent predictor of mortality alongside mediastinal radiation, age, and renal dysfunction.⁴² These findings support incorporation of objective hepatic assessment into preoperative decision-making for complex cases, including selective hepatology consultation and targeted evaluation of portal hypertension (eg, transjugular liver biopsy and hepatic venous pressure gradient measurement), with endoscopic screening and management of esophageal or gastric varices when portal hypertension is present to mitigate perioperative bleeding risk in consultation with the hepatology team.⁴³

CARDIAC HEMODYNAMICS AND STRUCTURAL ABNORMALITIES

Preoperative cardiac hemodynamics provide important prognostic information regarding perioperative risk and likelihood of symptomatic improvement. Severely reduced cardiac output, particularly a cardiac index below approximately 1.2 L/m²/min, identifies patients at risk for postoperative low-output syndrome and prolonged inotropic or mechanical support.^{7,17} Right ventricular dilation and dysfunction are associated with worse outcomes and reflect advanced disease chronicity and impaired adaptation following release of pericardial restraint.^{36,44} Left ventricular systolic dysfunction raises concern for concomitant myocardial disease and may limit postoperative recovery.

As discussed previously, tricuspid regurgitation may coexist with constrictive pericarditis and complicate both diagnosis and management. Mild tricuspid regurgitation is present in approximately one-third of patients, while moderate or severe regurgitation occurs in about 10% and is independently associated with increased postoperative mortality.³⁵ Approximately half of patients experience postoperative progression of tricuspid regurgitation, which has been associated with a downward trend in survival.⁴⁵ Careful preoperative assessment is therefore essential, and concomitant tricuspid valve repair or replacement should be considered in patients with moderate or severe regurgitation.

INDICATIONS FOR SURGERY AND PREOPERATIVE OPTIMIZATION

Pericardiectomy is primarily indicated in patients with chronic constrictive pericarditis and persistent symptoms with objective evidence of constrictive physiology, as definitive reversal of pericardial noncompliance is unlikely with medical therapy alone.^{7,17} In this setting, diuretics

may provide symptomatic relief but are palliative and should not delay definitive surgical intervention once fibrosis or calcification predominates. In contrast, patients with inflammatory or transient constrictive pericarditis should be treated medically before considering surgery, using anti-inflammatory therapy guided by symptoms, inflammatory markers, and imaging evidence of active pericardial inflammation. This typically includes colchicine with or without nonsteroidal anti-inflammatory medication when not contraindicated, and/or corticosteroids with consideration of IL-1 inhibitors in selected cases, for a duration of 3 to 6 months, followed by reassessment with echocardiography at 8 to 12 weeks and serial C reactive protein measurements until normalization.^{3,37} However, failure of constrictive physiology to improve despite appropriate medical therapy should prompt early surgical referral without further delays.^{3,23} Preoperatively, one should focus on two objectives related directly to pericardial pathology: (1) lowering RA pressure via diuresis as tolerated, and (2) controlling pericardial inflammation if present to lower risk of complications intraoperatively and postoperatively (eg, injury to coronary arteries, bleeding, etc). In addition, optimization of renal function, liver decongestion, and improving nutritional and physical status are important.

Formal randomized data and contemporary American guidelines defining surgical timing are lacking. The 2025 European Society of Cardiology Guidelines for the management of myocarditis and pericarditis recommend pericardiectomy as the definitive treatment for chronic constrictive pericarditis or disease refractory to anti-inflammatory therapy, with preference for complete pericardial resection performed at experienced high-volume centers.³⁷ Similarly, the 2025 American College of Cardiology Expert Consensus Statement on the Diagnosis and Management of Pericarditis emphasizes early referral for radical pericardiectomy in patients with chronic constriction and heart failure, noting that medical therapy may alleviate congestion but does not alter disease progression.³ ECP requires careful reassessment following pericardial drainage because constrictive physiology often resolves, and only persistent symptomatic constriction reflecting visceral pericardial involvement necessitates pericardiectomy.^{46,47} Given the complexity of patient selection and the importance of timing, referral to centers of excellence with expertise in both medical and surgical management of pericardial disease is strongly recommended. The integrated diagnostic and therapeutic pathway, including medical therapy, reassessment, and indications for surgical referral, is summarized in [Figure 3](#).

OPERATIVE APPROACHES AND TECHNIQUES

SURGICAL ACCESS AND EXTENT OF RESECTION

The historical standard operation for constrictive pericarditis has been anterior pericardiectomy from phrenic nerve to phrenic nerve. However, increasing recognition of recurrent constriction following partial resection, along with outcome data demonstrating superior survival and functional recovery after more extensive surgery, has shifted contemporary practice toward complete or radical pericardiectomy in experienced centers.^{48,49} Partial resection leaves posterior and diaphragmatic pericardium intact and may result in residual or recurrent constriction due to persistent fibrotic or calcific bands.^{48,50}

Radical pericardiectomy aims to remove all constraining pericardial tissue and typically includes resection of the anterior pericardium from phrenic to phrenic nerve, extension inferiorly over the diaphragm, and superiorly to the great vessels, as well as removal of the diaphragmatic pericardium and posterior pericardium behind the left atrium between the pulmonary veins and along the inferior vena cava.⁵¹ A fundamental principle is avoidance of residual confluent bands or rings of pericardium, which may perpetuate constriction despite technically successful surgery.

Management of the phrenic nerve pedicles is critical to achieving complete resection while preventing diaphragmatic dysfunction. This may be accomplished by preserving a narrow pericardial strip along the nerve or mobilizing the phrenic nerve on a fat pedicle with en bloc pericardial excision.⁵¹ In selected patients, the epicardium itself may be thickened and fibrotic and contribute to constriction; when safely feasible, epicardial decortication is required to achieve adequate hemodynamic relief.⁹

CARDIOPULMONARY BYPASS AND EXPOSURE STRATEGIES

Although early guidelines discouraged routine cardiopulmonary bypass (CPB) because of bleeding concerns, contemporary experience suggests that selective or liberal use of CPB can facilitate safer and more complete pericardiectomy without independently worsening outcomes.^{7,52} CPB provides hemodynamic stability during extensive dissection, reduces the risk of myocardial or coronary injury, allows controlled manipulation of the lateral left ventricular and diaphragmatic surfaces, and enables concomitant procedures when required.

Importantly, CPB is best viewed as a tool to improve exposure and completeness of resection, and its use often reflects anatomic complexity rather than procedural risk.

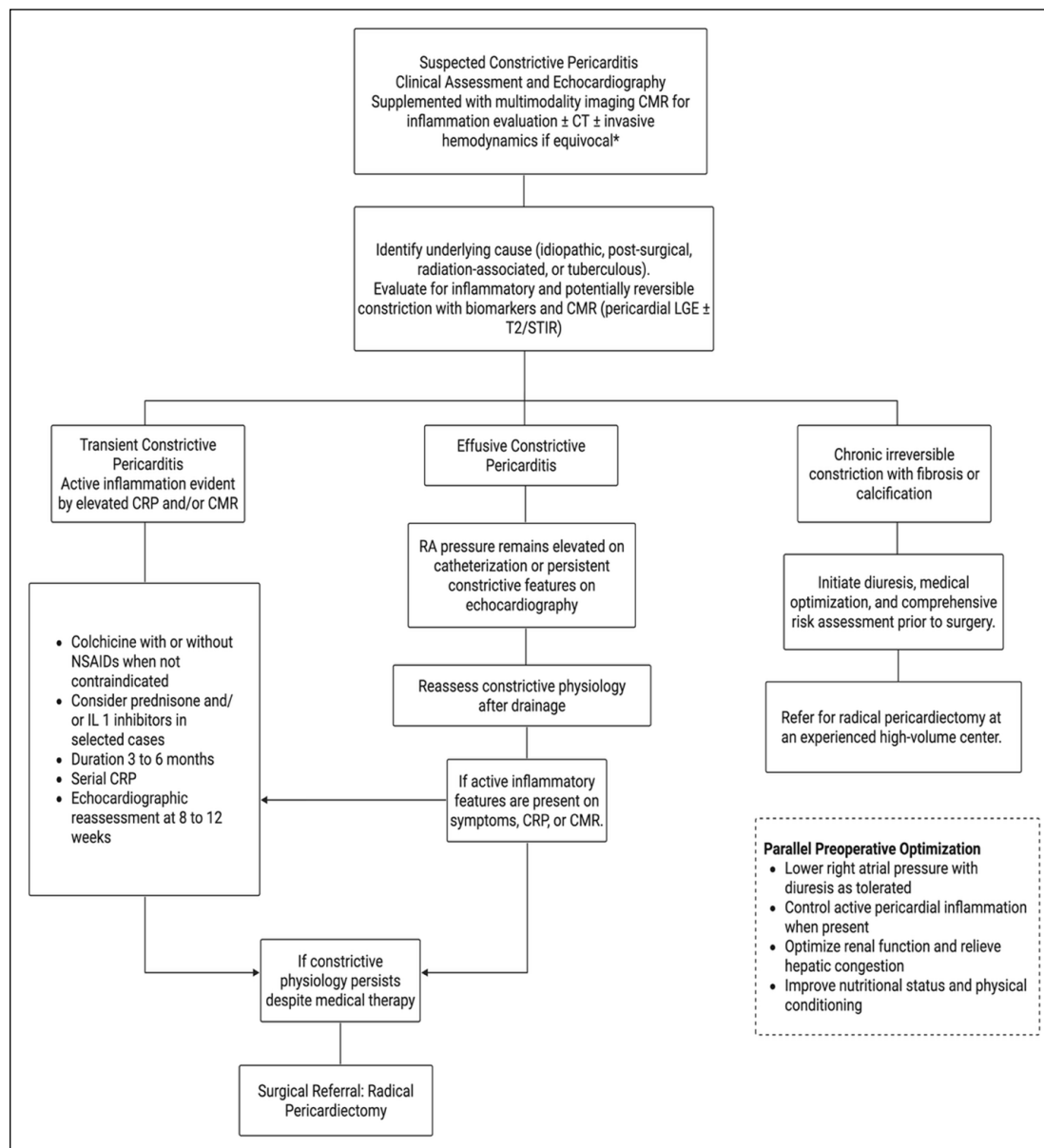


Figure 3 Diagnostic and surgical decision algorithm for constrictive pericarditis. (Created in BioRender with permission; Harake L, 2026; <https://BioRender.com/bl0r8ba>). CMR: cardiac magnetic resonance imaging; CT: computed tomography; CRP: C reactive protein; NSAIDs: nonsteroidal anti-inflammatory drugs; RA: right atrial

When CPB is avoided, off-pump apical suction devices may assist exposure and minimize hemodynamic compromise, particularly during dissection of the diaphragmatic and lateral ventricular surfaces.⁵² Importantly, experience from high-volume centers suggests that CPB use should be interpreted as a marker of advanced disease and operative complexity rather than an independent predictor of adverse outcomes.⁷

EFFUSIVE-CONSTRICTIVE PERICARDITIS

ECP often presents with a thickened parietal pericardium and an inflammatory epicardial rind, posing unique technical challenges.⁴ Complete removal of the epicardial

component may not be feasible in all cases. In such situations, a checkerboard technique, dividing the rind into multiple segments, may permit myocardial expansion. Residual epicardial inflammation frequently improves following pericardiectomy, and observational experience suggests reduced epicardial involvement in regions where parietal pericardium has been previously removed.⁵³

SPECIAL SURGICAL CONSIDERATIONS

Prior Cardiac Surgery

Patients with prior cardiac surgery, particularly those with patent coronary bypass grafts, represent a technically high-risk subgroup. Preoperative cardiac CT angiography is

essential to delineate graft location, especially left internal thoracic artery grafts. In regions where the phrenic nerve, grafts, and cardiac structures are closely approximated, leaving a small remnant of pericardium may be preferable to risking injury provided no constrictive bands remain.⁷

Mediastinal Radiation

Prior mediastinal radiation, commonly for Hodgkin disease, is associated with dense adhesions, hypervascular and fibrotic mediastinal tissues, and frequent concomitant myocardial involvement.^{40,54} Mixed constrictive and restrictive physiology is common and may limit symptomatic improvement even after technically successful pericardiectomy, underscoring the importance of careful preoperative assessment and patient selection.

Myocardial and Pericardial Calcification

Calcified pericardium may extend into the myocardium, and complete removal of intramyocardial calcium is often not feasible. However, care must be taken to disrupt confluent calcific bands that may act as residual constrictive elements. Rongeurs or oscillating saws may be required for dense calcification.^{7,55}

Valve Assessment and Concomitant Procedures

Relief of pericardial restraint frequently results in right ventricular dilation, which may exacerbate tricuspid regurgitation. Worsening tricuspid regurgitation occurs in approximately half of patients following pericardiectomy and is associated with reduced survival.⁴⁵ Careful preoperative valve assessment is therefore essential, and concomitant tricuspid valve repair or replacement should be considered in patients with moderate or severe regurgitation.

Phrenic Nerve Protection

Early identification of the phrenic nerves is critical to prevent diaphragmatic dysfunction. Opening both pleural spaces early in the operation facilitates visualization. Nerves may be mobilized using meticulous dissection with minimal electrocautery or preserved on a small pericardial island, which must be fully detached from surrounding reflections to avoid residual constriction.^{7,34}

POSTOPERATIVE MANAGEMENT AND PROGNOSIS

Early postoperative care after pericardiectomy focuses on hemodynamic stabilization and anticipation of complications related to abrupt release of pericardial constraint. Common early complications include atrial

arrhythmias requiring rate and/or rhythm control, vasoplegia particularly in patients with advanced hepatic disease, and relative adrenal insufficiency in those previously exposed to long-term corticosteroids. Vasoplegia may be profound and prolonged in cirrhotic patients, often necessitating several days of vasopressor support despite improving cardiac output.^{2,44,56}

Hemodynamic management prioritizes prevention of acute right ventricular distension. Strategies include cautious volume administration, fluid restriction, low-dose inotropic support, and atrial pacing when needed. The immediate goal is not normalization of cardiac output but modest improvement relative to preoperative values provided that filling pressures and pulmonary artery pressures improve. Persistent low cardiac output both before and after surgery is associated with worse outcomes and suggests a greater contribution from restrictive myocardial disease, particularly in post-radiation patients. A rare but important complication occurs when severe constriction is abruptly relieved, leading to acute ventricular volume overload and myocardial stunning. This is most often observed when the right ventricle is decompressed before the left in off-pump procedures and may result in cardiogenic shock requiring urgent extracorporeal mechanical support.⁵⁷

Reported perioperative mortality after pericardiectomy varies across contemporary series and is primarily driven by patient substrate and disease etiology rather than procedural risk alone.^{7,22,34,52} In a single-center experience spanning 1995 to 2010 and including 98 patients, in-hospital mortality was approximately 7%, with early risk related to the need for cardiopulmonary bypass and long-term outcomes strongly influenced by disease etiology.²² In the largest contemporary cohort of isolated pericardiectomy comprising 513 patients followed over 2 decades, operative mortality was low at 2.3%, with early death associated with ventricular dysfunction and renal disease rather than surgical factors.⁵² Similarly, in a large Cleveland Clinic series of 601 patients, Unai and Johnston reported an overall in-hospital mortality of 6%, varying markedly by etiology at 1.1% for idiopathic disease, 9.7% for postoperative constriction, and 27% for radiation-associated disease.⁷

Outcomes are excellent in patients undergoing pericardiectomy for refractory recurrent pericarditis, with near-zero operative mortality, underscoring the safety of the operation itself in appropriately selected patients.⁵¹ Advances in surgical technique and perioperative care have reduced early mortality over time, with large institutional series demonstrating a decline in 30-day mortality from approximately 13% in earlier eras to nearly 5% in contemporary practice.⁵⁸ However, long-term survival remains strongly determined by etiology, with

idiopathic disease carrying the most favorable prognosis and post-radiation or post-surgical constriction associated with substantially worse outcomes. These observations highlight the importance of early referral, careful patient selection, and management at experienced centers using a multidisciplinary approach.

FUTURE DIRECTIONS AND SYSTEMS OF CARE

Despite advances in imaging, perioperative management, and surgical technique, outcomes in constrictive pericarditis remain limited by delayed diagnosis, advanced disease at presentation, and variability in expertise across institutions. Earlier recognition of inflammatory and potentially reversible phenotypes, structured referral pathways, and standardized preoperative risk assessment may improve patient selection and timing of intervention. Given the complexity of constrictive pericarditis, optimal care requires dedicated centers of excellence with multidisciplinary expertise encompassing pericardiology, advanced cardiac imaging, cardiac surgery, anesthesiology, hepatology, and critical care. Development of specialized pericardial programs with longitudinal follow-up may reduce unnecessary surgery, improve operative safety in high-risk patients, and optimize long-term outcomes. Further prospective studies are needed to refine risk stratification, define optimal timing of intervention, and evaluate whether contemporary surgical strategies translate into durable survival benefit.

CONCLUSION

Constrictive pericarditis is a mechanically driven disease with heterogeneous etiologies, variable reversibility, and outcomes that depend primarily on disease chronicity, myocardial involvement, and end-organ dysfunction rather than the technical aspects of surgery alone. Pericardiectomy remains the definitive therapy for chronic constriction or refractory subacute CP and can be performed safely with meaningful symptomatic improvement when patients are carefully selected and referred before advanced systemic involvement develops. Multimodality imaging, risk stratification, and recognition of inflammatory or mixed phenotypes are essential to guide management and avoid both premature surgery and harmful delays. When performed at high-volume experienced centers with comprehensive perioperative support, radical pericardiectomy offers the best opportunity for durable hemodynamic and clinical recovery in appropriately selected patients.

KEY POINTS

- Constrictive pericarditis is a mechanically driven disease with heterogeneous etiologies and variable reversibility, requiring accurate differentiation between inflammatory, chronic fibrotic, and mixed phenotypes (concomitant constriction and restrictive cardiomyopathy such as in radiation heart disease patients) to guide management.
- Pericardiectomy is the definitive treatment for chronic constrictive pericarditis and refractory subacute pericarditis. Outcomes are determined primarily by disease substrate, myocardial involvement, and timing of intervention rather than the technical act of surgery alone.
- Radical pericardiectomy on cardiopulmonary bypass provides superior hemodynamic and functional outcomes compared with partial resection and should be pursued when safely feasible, particularly in chronic and recurrent disease.
- Preoperative multimodality imaging and hemodynamic assessment are essential to define disease extent, identify mixed constrictive restrictive physiology, and plan the operative strategy, including the need for cardiopulmonary bypass or concomitant valve intervention.
- Early referral to experienced centers of excellence with multidisciplinary expertise improves operative safety, facilitates aggressive yet controlled resection, and optimizes both short- and long-term outcomes.

COMPETING INTERESTS

Dr. Al-Kazaz has received research grants from Kiniksa Pharmaceuticals, Ventyx BioSciences, and Cardiol Therapeutics, is on the Speakers Bureau for Kiniksa Pharmaceuticals, and is a consultant for Edwards Lifesciences. Dr. Cremer is a consultant for Kiniksa Pharmaceuticals, CardiolRx, Ventyx Biosciences, Monte Rosa Therapeutics, Pfizer, Boston Scientific, and General Electric. Dr. Johnston is a consultant for Edwards Lifesciences, Medtronic, Terumo Aortic, and Artivion.

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