



CONSTRUCTIVE PERICARDITIS: A STRUCTURES REVIEW OF THE MEDICAL LITERATURE DIAGNOSIS, MULTIMODAL IMAGING AND CONTEMPORARY MANAGEMENT

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ABSTRACT

Constrictive pericarditis (CP) is an uncommon but potentially curable cause of heart failure with preserved ejection fraction, in which a fibrotic, inelastic, and often calcified pericardium restricts diastolic filling of the ventricles. The disease remains a diagnostic challenge because its clinical picture overlaps with restrictive cardiomyopathy, severe tricuspid regurgitation, and non-cardiac causes of systemic venous congestion. This review synthesizes the contemporary medical literature on CP, covering its epidemiology and evolving etiologic spectrum, the two cardinal pathophysiologic mechanisms (dissociation of intrathoracic and intracardiac pressures and exaggerated ventricular interdependence), the validated echocardiographic diagnostic criteria, the complementary roles of cardiac magnetic resonance imaging, cardiac computed tomography, and invasive hemodynamics, and current management strategies. Particular emphasis is placed on the identification of transient (inflammatory) constriction, which may resolve with anti-inflammatory therapy, and on the outcomes and risk stratification of radical pericardiectomy, the definitive treatment for chronic disease.

Keywords: Constrictive pericarditis; Pericardiectomy; Echocardiography; Cardiac magnetic resonance; Heart failure with preserved ejection fraction; Restrictive cardiomyopathy

1 INTRODUCTION AND EPIDEMIOLOGY

Constrictive pericarditis is the end stage of an inflammatory or fibrosing process of the pericardium, in which thickening, scarring, and variable calcification convert the normally compliant pericardial sac into a rigid shell. The result is impaired diastolic filling with preserved systolic function in most patients a surgically reversible form of heart failure. Because its presentation mimics far more common conditions, a high index of clinical suspicion is repeatedly emphasized across the literature as the cornerstone of timely diagnosis. CP is rare, and its prevalence and incidence are not well established. Approximately 1.8% of patients with acute pericarditis progress to chronic constrictive physiology, and the risk is strongly etiology-dependent: a prospective analysis estimated an incidence of 0.76 cases per 1,000 person-years after idiopathic or viral pericarditis, compared with 31.7 per 1,000 person-years after tuberculous

pericarditis and 52.7 per 1,000 person-years after purulent pericarditis.^[1] After cardiac surgery, chronic constriction develops in only about 0.2-2.4% of patients.^[2] The condition is more common in men, with a reported male-to-female predominance of approximately 3:1, and no racial predilection has been identified.^[3] The largest systematic review of the global epidemiology of CP, including 130 studies and 11,325 patients, documented two important trends: the age at diagnosis has increased markedly since 1990, and the etiologic distribution differs sharply by region. Tuberculosis remains the dominant cause in Africa and Asia, whereas previous chest surgery has become the leading cause in North America and Europe. HIV coinfection affects roughly 29% of African patients with CP, a feature not observed on other continents.^[4] Early in-hospital mortality after diagnosis has improved over time in this dataset.

2 PATHOPHYSIOLOGY

Chronic CP develops when healing of fibrinous or serofibrinous pericarditis or resorption of a chronic pericardial effusion obliterates the pericardial cavity with granulation tissue that progressively contracts, fibroses, and may calcify. The rigid pericardium has a much lower elastic limit than the normal pericardium, so early diastolic filling is unimpeded until the pericardial constraint is abruptly reached, after which filling ceases. This produces the characteristic rapid early filling with an abrupt mid-diastolic halt (the hemodynamic “dip-and-plateau” or “square-root” sign), in contrast to cardiac tamponade, in which filling is impeded throughout diastole.^[3,5] The consequence is reduced end-diastolic volume, reduced stroke volume, and chronic elevation of systemic venous pressures

Two interrelated mechanisms account for nearly all diagnostic findings in CP:

- **Dissociation between intrathoracic and intracardiac pressures.** The noncompliant pericardium insulates the cardiac chambers from respiratory changes in intrathoracic pressure. During inspiration, intrathoracic pressure falls and this change is transmitted to the extrapericardial pulmonary veins but not to the intrapericardial left atrium; the reduced pressure gradient impairs left-sided filling during inspiration.
- **Exaggerated ventricular interdependence.** Because total cardiac volume is fixed by the inelastic pericardium, inspiratory augmentation of right ventricular filling occurs at the expense of left ventricular filling, and vice versa during expiration. This reciprocal, respirophasic filling is visible as the inspiratory septal shift toward the left ventricle and underlies the discordant respiratory changes in left and right ventricular systolic pressures used in invasive diagnosis.^[6]

Kussmaul's sign—a paradoxical rise or failure of fall of jugular venous pressure during inspiration—reflects the inability of the constricted right heart to accommodate increased venous return.^[3] Contemporary frameworks view CP not as a static entity but as a continuum from an inflammatory, potentially reversible phase (edema and fibrin deposition, as in transient and effusive-constrictive forms) to established fibroelastic and calcific remodeling, which has major therapeutic implications.^[6]

3 ETIOLOGY

Virtually any cause of pericardial injury can culminate in constriction, and in many patients no antecedent event is ever identified; such idiopathic cases are presumed to follow unrecognized viral pericarditis. Only about 10% of patients report a documented episode of acute pericarditis.^[7]

- **Worldwide:** tuberculosis is the leading cause of CP; constriction develops in up to half of patients with tuberculous pericardial effusion despite antituberculous therapy.^[3]
- **High-income countries:** the dominant etiologies are idiopathic/presumed viral disease (roughly 40-60% of cases), previous cardiothoracic surgery, and mediastinal radiation therapy.^[3,7]
- **Radiation-induced CP** follows microvascular endothelial injury and typically manifests 5-10 years after treatment, with reported incidence ranging from 2% to 30% of irradiated patients; it frequently coexists with myocardial fibrosis, valvular disease, and coronary disease, which worsens surgical outcomes.^[7]
- **Less common causes** include connective tissue diseases (rheumatoid arthritis, systemic lupus erythematosus, scleroderma), malignancy (metastatic breast and lung carcinoma, lymphoma, mesothelioma), uremia, bacterial and fungal infection, trauma, post-myocardial infarction syndromes, and drugs (e.g., procainamide, hydralazine, methysergide).^[7]

Notably, the etiologic mix continues to evolve: historical series were dominated by tuberculosis, whereas contemporary Western cohorts are dominated by idiopathic and post-surgical disease, with iatrogenic and procedure-related cases increasingly reported.^[4,6]

4 CLINICAL PRESENTATION

CP typically presents insidiously as a chronic heart failure syndrome dominated by right-sided congestion: progressive exertional dyspnea and fatigue, elevated jugular venous pressure (often with Kussmaul's sign and prominent y descent), hepatomegaly, ascites that is frequently disproportionate to peripheral edema, and pleural effusions. The pericardial knock an early diastolic high-pitched sound corresponding to the abrupt cessation of ventricular filling is characteristic but not universal. Atrial arrhythmias, particularly atrial fibrillation, are common in advanced disease, and long-standing cases may develop cardiac cachexia, protein-losing enteropathy, and congestive cirrhosis. Because the syndrome overlaps with restrictive cardiomyopathy, severe tricuspid regurgitation, cirrhosis, and other causes of systemic venous hypertension, diagnostic delay is common; several reviews emphasize that an adequate history and careful physical examination, supported by a high index of suspicion, remain essential.^[8] CP should be actively considered in any patient with unexplained right heart failure and preserved ejection fraction, especially with a history of cardiac surgery, mediastinal irradiation, tuberculosis, or prior pericarditis.

5 DIAGNOSIS

No single test is diagnostic; current practice, endorsed by the 2015 ESC guidelines and the 2025 ACC expert consensus, integrates echocardiography as the first-line modality with cardiac magnetic resonance (CMR), cardiac computed tomography (CCT), and when noninvasive data are incongruent invasive hemodynamics.^[9,10]

5.1 Echocardiography

Transthoracic echocardiography is recommended as the initial test in all suspected cases. The key abnormalities directly reflect the two pathophysiologic mechanisms: respiration-related ventricular septal shift (“septal bounce”), respiratory variation of transvalvular inflow (classically >25% fall in mitral E velocity with inspiration and >40% increase in tricuspid E velocity), preserved or increased medial mitral annular tissue Doppler e' velocity, a reversed medial-to-lateral e' relationship (“annulus reversus”), and prominent expiratory diastolic flow reversals in the hepatic veins. Inferior vena cava plethora is present in nearly all patients and may be regarded as a prerequisite for the diagnosis.^[11] The most influential modern validation is the Mayo Clinic study by Welch et al. (2014), a blinded comparison of 130 patients with surgically confirmed CP against 36 patients with restrictive myocardial disease or severe tricuspid regurgitation. Three variables were independently associated with CP: respiration-related ventricular septal shift (odds ratio 7.71), medial mitral e' velocity (OR 5.11 per 5 cm/s), and hepatic vein expiratory diastolic reversal ratio (OR 3.02 per 0.5). The combination of septal shift with either medial e' ≥ 9 cm/s or a hepatic vein reversal ratio ≥ 0.79 yielded 87% sensitivity and 91% specificity; requiring all three criteria raised specificity to 97% at the cost of sensitivity (64%). Importantly, these criteria retained their discriminatory power in patients with atrial fibrillation or flutter (with the exception of mitral inflow variation).^[11]

Table 1: Key echocardiographic criteria for constrictive pericarditis (based on the Mayo Clinic criteria, Welch et al., 2014)

Echocardiographic criterion	Typical finding in CP	Diagnostic role
Respiration-related ventricular septal shift	Inspiratory shift of septum toward LV	Highly sensitive starting point; strongest independent predictor
Mitral inflow respiratory variation	>25% decrease in E velocity with inspiration	Supportive; not independent in Mayo analysis; unreliable in atrial fibrillation
Medial mitral annular e'	Preserved or increased (≥ 9 cm/s)	Independent predictor; separates CP from restriction
Annulus reversus (medial e' > lateral e')	Present	Supportive of constriction
Hepatic vein Doppler	Expiratory diastolic flow reversal (ratio ≥ 0.79)	Independent predictor; increases specificity
Inferior vena cava	Dilated (≥ 21 mm), inspiratory collapse <50%	Near-universal; consider prerequisite

5.2 Cardiac Magnetic Resonance Imaging

CMR provides both anatomic and physiologic information. Free-breathing real-time cine imaging demonstrates the early inspiratory septal shift indicative of constrictive physiology, while pericardial thickness is readily measured although a normal thickness does not exclude CP, being observed in approximately 18% of confirmed cases.^[10] The decisive contribution of CMR is tissue characterization: T2 short-tau inversion recovery (T2-STIR) hyperintensity reflects pericardial edema, and late gadolinium enhancement (LGE) reflects neovascularization and active inflammation. The 2025 ACC consensus recommends CMR to identify pericardial inflammation as the driver of constriction, and considers it reasonable when echocardiography is inconclusive.^[10] The landmark study by Feng et al. (2011) linked CMR findings to treatment response: among 29 patients with definite CP treated with anti-inflammatory therapy, constriction resolved in 14 (48%). The reversible subgroup had greater pericardial LGE thickness (about 4 vs 2 mm), more frequent moderate-to-severe LGE (93% vs 33%), and higher baseline CRP and ESR; an LGE thickness of 3 mm predicted reversibility with 86% sensitivity and 80% specificity, and a CRP cutoff of 21 mg/L predicted it with 70% sensitivity and 72% specificity.^[12] These data underpin the modern strategy of identifying the inflammatory, potentially reversible phase of constriction before committing patients to surgery.

5.3 Cardiac Computed Tomography

CCT is the most sensitive modality for detecting pericardial calcification a finding that supports, but is not required for, the diagnosis and is recommended by the 2025 ACC consensus as supplementary evidence for constriction and for preoperative planning, in particular to delineate the relationship of cardiovascular structures to the sternum and to map adhesions before pericardiectomy.^[10] Ancillary signs such as tubular ventricular deformation, atrial and caval dilation, and hepatosplenomegaly with ascites add supportive evidence.

5.4 Invasive Hemodynamic Assessment

When clinical and imaging data are incongruent, simultaneous left and right heart catheterization remains the reference standard. Classic findings include elevation and near-equalization of diastolic pressures (within ~5 mmHg), the dip-and-plateau waveform, and a prominent y descent in the atrial pressure tracing. The most robust modern criterion exploits ventricular interdependence: discordant respiratory changes in left and right ventricular systolic pressures, quantified by the systolic area index (ratio of RV to LV systolic pressure-time area in inspiration versus expiration), which in the Mayo Clinic catheterization series achieved very high diagnostic accuracy.^[13] The 2025 ACC consensus recommends invasive hemodynamic evaluation whenever noninvasive testing is nondiagnostic or equivocal.^[10]

5.5 Differentiation from Restrictive Cardiomyopathy

Distinguishing CP from restrictive cardiomyopathy (RCM) is the central differential challenge, and it matters because CP is often surgically curable whereas RCM requires medical management of an underlying myocardial disease (e.g., amyloidosis, hemochromatosis, sarcoidosis) or transplantation. Historically, hemodynamic criteria (RVEDP-LVEDP difference, RV systolic pressure, RVEDP/RV systolic ratio) classified about 75% of patients correctly but left one quarter unclassified.^[14] Doppler echocardiography transformed this distinction: respiratory changes in transvalvular flows are marked in CP and absent in RCM.^[15]

Table 2: Distinguishing constrictive pericarditis from restrictive cardiomyopathy.

Feature	Constrictive pericarditis	Restrictive cardiomyopathy
Primary pathology	Pericardial constraint (fibrosis/calcification)	Intrinsic myocardial stiffness or infiltration
Septal motion	Respirophasic septal shift; septal bounce	No exaggerated respiratory septal motion
Tissue Doppler e'	Preserved or increased medial e'; annulus reversus	Reduced medial and lateral e'
Respiratory inflow variation	Marked (mitral >25%, tricuspid >40%)	Minimal
Ventricular interdependence	Exaggerated (discordant RV/LV pressures)	Absent
BNP/NT-proBNP	Normal or modestly elevated	Usually markedly elevated
CMR / CT	Pericardial thickening, LGE, calcification	Myocardial tissue characterization (e.g., amyloid LGE pattern)

Definitive therapy	Pericardiectomy (often curative)	Treatment of underlying disease; transplantation in selected patients
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5.6 Special Variants: Transient and Effusive-Constrictive Disease

Transient constriction is a reversible, inflammation-dominant form in which pericardial edema and fibrin rather than dense fibrosis produce constrictive physiology; it typically follows viral or idiopathic pericarditis or cardiac surgery and resolves with effective anti-inflammatory therapy.^[6,16] In the series of Haley et al., a substantial minority of constrictive presentations resolved spontaneously or with medical therapy, and the Feng et al. CMR study later demonstrated that the reversible phenotype can be identified prospectively by the burden of pericardial LGE and systemic inflammation.^[12,16] Effusive-constrictive pericarditis (ECP) combines a hemodynamically significant effusion with constriction of the visceral pericardium; its hallmark is persistent elevation of right atrial pressure after pericardiocentesis has normalized intrapericardial pressure.^[17] ECP complicates about 1% of pericarditis cases overall but up to half of tuberculous cases. Post-drainage echocardiography typically unmasks respirophasic septal shift, mitral inflow variation, persistent hepatic vein diastolic flow reversals, and inferior vena cava dilation, and a 2024 international consensus recommends a comprehensive echocardiographic protocol for its evaluation.^[2]

6 MANAGEMENT

6.1 Anti-inflammatory Therapy

For hemodynamically stable patients with newly diagnosed or transient constriction and evidence of pericardial inflammation (elevated inflammatory markers or CMR T2-STIR/LGE abnormalities), a trial of anti-inflammatory therapy is recommended as first-line treatment before considering surgery.^[9,10] Regimens follow the general pericarditis paradigm: NSAIDs or high-dose aspirin plus colchicine, with corticosteroids reserved for refractory or specific etiologies and tapering should begin only after symptoms resolve and C-reactive protein normalizes. In tuberculous constriction, a 6-month course of standard multidrug antituberculous therapy is recommended and may itself resolve constrictive physiology; corticosteroids may augment clinical improvement, and pericardiectomy is advised if patients fail to improve or deteriorate after 4-8 weeks of antituberculous treatment.^[9,18]

6.2 Supportive and Diuretic Therapy

Diuretics relieve volume overload and are considered reasonable for symptomatic control, but they do not alter the natural history of chronic constriction and excessive diuresis can precipitate hypotension by reducing preload below the constrained filling limit. Evidence for goal-directed heart failure pharmacotherapy is limited because ventricular systolic function is usually preserved.^[10]

6.3 Surgical Pericardiectomy

Radical pericardiectomy is the definitive treatment for permanent (non-inflammatory) CP and for inflammatory CP that fails a 3-6-month trial of anti-inflammatory therapy.^[9,10] The operation should resect the pericardium as completely as possible anteriorly between the phrenic nerves and including the diaphragmatic and, where feasible, posterior surfaces usually via median sternotomy, with cardiopulmonary bypass available; partial pericardiectomy is explicitly not recommended because incomplete decortication is associated with persistent hemodynamic abnormalities and early mortality.^[10] Both the ACC and ESC documents stress referral to experienced tertiary pericardial centers.^[9,10] Outcomes have improved but remain strongly etiology dependent. In the landmark Mayo Clinic series of 135 patients (1985-1995), perioperative mortality fell from 14% in a historical cohort to 6%, 10-year survival was 52%, and 83% of late survivors were free of symptoms; independent predictors of late outcome were age, NYHA class, and most powerfully a post-radiation etiology.^[19] A contemporary 98-patient series (1995-2010) reported in-hospital survival of 92.9% and 1-, 5-, and 10-year survival of 82.5%, 64.3%, and 49.2%; five-year survival differed dramatically by cause (idiopathic 79.8%, postoperative 55.9%, post-radiation 11.0%), while cardiopulmonary bypass requirement predicted 30-day mortality (HR 21.2) and hypoalbuminemia and post-radiation etiology predicted late mortality.^[20] A 47-patient series dominated by tuberculous disease reported 2.1% in-hospital mortality, 10-year survival of 81%, and improvement of at least one NYHA class in 80% of patients, underscoring that outcomes in low-risk, non-radiation populations can be excellent.^[21]

Table 3: Representative outcomes after pericardiectomy for constrictive pericarditis.

Study	n	Era / setting	Perioperative mortality	5-year survival
Ling et al., Mayo Clinic, 1999	135	1985-1995, USA	6% (vs 14% historic)	71% (post-radiation worst)
Contemporary series, 2013	98	1995-2010, USA	7.1% (in-hospital)	64% overall; 80% idiopathic vs 11% post-radiation
Biçer et al., 2015	47	1992-2014, TB-predominant cohort	2.1%	85%

6.4 Prognosis

Untreated chronic CP is relentlessly progressive, with worsening refractory heart failure, hepatic dysfunction, and cachexia; medical management alone does not change its course. After successful radical pericardiectomy, most survivors achieve marked functional improvement typically at least one NYHA class but long-term survival remains inferior to that of an age- and sex-matched general population.^[19] The dominant adverse prognostic factor is a post-radiation etiology, because irradiation frequently damages the myocardium, valves, and coronary arteries alongside the pericardium, limiting both the completeness and the benefit of decortication. Additional predictors of poor early or late outcome across series include advanced age, higher NYHA class, renal dysfunction, hypoalbuminemia, hyperbilirubinemia, pulmonary disease, concomitant coronary artery disease, and the need for cardiopulmonary bypass.^[19,20] Patients with inflammatory (transient) constriction identified early and treated with anti-inflammatory therapy can avoid surgery altogether, which is the strongest argument for early recognition and CMR-based phenotyping.^[12]

7 CONCLUSIONS AND KEY CLINICAL POINTS

- CP is a rare, potentially curable cause of heart failure with preserved ejection fraction; suspect it in unexplained right heart failure, especially after cardiac surgery, chest irradiation, or tuberculosis.
- Its diagnosis rests on two mechanisms intrathoracic-intracardiac pressure dissociation and exaggerated ventricular interdependence captured by echocardiography (Mayo criteria: septal shift plus medial $e' \geq 9$ cm/s or hepatic vein reversal ratio ≥ 0.79).^[11]
- CMR identifies pericardial inflammation (T2-STIR edema, LGE) and predicts reversibility, enabling a medical-first strategy in inflammatory constriction; CT best demonstrates calcification and guides surgical planning; invasive hemodynamics with the systolic area index resolves equivocal cases.^[10,12,13]
- Differentiation from restrictive cardiomyopathy is essential and now largely achievable noninvasively: preserved tissue Doppler velocities, annulus reversus, and respirophasic changes favor constriction.^[15]
- Treatment is stage-dependent: anti-inflammatory therapy first when inflammation is present; radical pericardiectomy at experienced centers for permanent disease, with partial resection explicitly discouraged.^[9,10]
- Surgical outcomes are good in idiopathic and post-surgical disease but poor after chest irradiation; risk stratification by etiology, functional class, and nutritional/hepatic status should guide shared decision-making.^[19,20]

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