Constrictive Pericarditis: Modern Causes of an Old Disease

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Abstract

This review examines the evolving landscape of constrictive pericarditis (CP) through analysis of literature spanning 1932 to 2025, sourced from PubMed, Scopus, and Google Scholar using key terms related to the etiology, clinical features, diagnosis, and treatment of CP. Eligible literature included original research articles, systematic and narrative reviews, case reports, expert consensus statements, and guidelines from professional societies. CP is a chronic pericardial disease resulting in impaired ventricular filling and heart failure symptoms. While previously dominated by tuberculosis and idiopathic cases, its modern etiological profile has shifted significantly. This review explores the evolving causes, clinical features, diagnostic advances, and treatment strategies of CP in the current medical practice. Modern causes of CP include prior cardiac surgery/percutaneous intervention, radiation therapy, autoimmune diseases, viral pericarditis, and uremia associated with end-stage renal disease. Modern imaging modalities—particularly echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging—have improved diagnostic accuracy and helped differentiate CP from other causes of heart failure. Features such as pericardial late gadolinium enhancement and elevated inflammatory markers help identifying reversible subset of the disease, which may respond to anti-inflammatory therapy. In chronic, fibrotic cases, surgical pericardiectomy remains the definitive therapy, with outcomes improved by early diagnosis and appropriate timing of surgery. CP is no longer a relic of the past, but a dynamic condition shaped by modern medicine. Recognizing its evolving etiologies is critical for timely diagnosis and individualized treatment. With advances in imaging and inflammation-targeted therapies, opportunities exist to improve outcomes and reduce reliance on surgery in selected cases.

Keywords

- constriction
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- pathophysiology
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المقالة باللغة العربية

التهاب التامور المضيق: أسباب حديثة لمرض قديم

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تستعرض هذه المقالة التطورات في فهم التهاب التامور المضيق من خلال تحليل الأبحاث المنشورة بين عامي 1932 و2025، والتي تم الحصول عليها من قواعد بيانات PubMed و Scopus وGoogle Scholar باستخدام كلمات مفتاحية متعلقة بالأسباب، المظاهر السريرية، التشخيص، وعلاج التهاب التامور. اشتملت الأبحاث المختارة مقالات بحثية أصلية، مراجعات منهجية ووصفية، تقارير حالات، بيانات إجماع الخبراء، وإرشادات الجمعيات المتخصصة.

يعد التهاب التامور المضيق مرضاً مزمناً يصيب التامور ويؤدي إلى ضعف ملء البطينين وأعراض قصور القلب. بينما كان يُعزى سابقاً بشكل رئيسي إلى السل وحالات مجهولة السبب، فقد تغيرت خصائصه السببية الحديثة بشكل كبير. تستكشف هذه المراجعة الأسباب المتغيرة، المظاهر السريرية، التطورات التشخيصية، واستراتيجيات العلاج لـ التهاب التامور المضيق في الممارسة الطبية الحديثة.

تشمل الأسباب الحديثة لـ التهاب التامور المضيق جراحات القلب السابقة/التدخلات عبر الجلد، العلاج الإشعاعي، أمراض المناعة الذاتية، التهاب التامور الفيروسي، واليوريميا المرتبطة بمرض الكلى في المرحلة النهائية. وقد حسنت تقنيات التصوير الحديثة - خاصة تخطيط صدى القلب، التصوير المقطعي للقلب، والرنين المغناطيسي القلبي - دقة التشخيص وساعدت في تمييز التهاب التامور المضيق عن أسباب أخرى لقصور القلب. كما تساعد سمات مثل التباين المتأخر للجادولينيوم في التامور وارتفاع علامات الالتهاب في تحديد الحالات القابلة للانعكاس التي قد تستجيب للعلاج المضاد للالتهاب. في الحالات المزمنة الليفية، يظل استئصال التامور الجراحي العلاج النهائي، مع تحسن النتائج عند التشخيص المبكر والتوقيت المناسب للجراحة.

لم يعد التهاب التامور المضيق مرضاً من الماضي، بل أصبح حالة ديناميكية تشكلها الطب الحديث. إن التعرف على أسبابه المتغيرة أمر بالغ الأهمية للتشخيص في الوقت المناسب والعلاج الفردي. ومع تطور التصوير والعلاجات المضادة للالتهاب، توجد فرص لتحسين النتائج وتقليل الاعتماد على الجراحة في حالات مختارة.

الكلمات المفتاحية: تضيق، التهاب التامور المضيق، الفيزيولوجيا المرضية، أمراض التامور، التهاب التامور.

Introduction

The pericardium is a relatively avascular fibrous sac surrounding the heart, composed of two layers: the visceral and parietal pericardium. The visceral layer consists of a single layer of mesothelial cells adherent to the epicardium, while the parietal layer is a fibrous structure less than 2 mm thick, composed primarily of collagen with some elastin. Between these layers lies a potential space normally containing 15 to 35 mL of serous fluid. Functionally, the pericardium stabilizes the heart within the mediastinum, prevents excessive cardiac dilation during sudden volume shifts, and serves as a barrier to limit the spread of infection from adjacent structures.[1,2]

Constrictive pericarditis (CP) is a chronic condition marked by fibrotic thickening and often calcification of the pericardium, leading to impaired diastolic filling, reduced cardiac output, and causes systemic venous congestion. Once primarily attributed to infections such as tuberculosis—especially in the preantibiotic era[3,4]—its epidemiology has shifted significantly in recent decades. While tuberculosis remains a leading cause of CP in endemic regions, more contemporary etiologies in developed countries include prior cardiac surgery, interventional cardiac procedures, thoracic radiation therapy, viral pericarditis, and autoimmune disorders. These causes have been increasingly reported in recent literature and now account for a significant proportion of cases.[5–12]

This evolving clinical landscape necessitates a reappraisal of CP, not only regarding its etiology but also its diagnostic and therapeutic approach.

This review aims to explore the contemporary understanding of CP by bridging historical context with modern practice, highlighting advances in diagnosis, shifting etiologies, and current management strategies to improve patient outcomes.

Historical Perspective on CP

CP has been recognized for centuries.[13] In 1669, Richard Lower offered the first clinical insight into pericardial constriction by describing cardiac tamponade and pulsus paradoxus in patients with dyspnea, laying the foundation for the hemodynamic understanding of pericardial diseases.[14]

During the 18th century, Giovanni Battista Morgagni and Giovanni Maria Lancisi provided key pathological descriptions linking pericardial effusion and thickening to circulatory compromise and specific clinical signs, such as distended neck veins and abdominal swelling.[15,16]

The 19th century brought further clarity: Norman Chevers differentiated CP from other cardiac pathologies,[17] while Adolph Kussmaul and Friedrich Pick described hallmark signs of pericardial constriction including paradoxical pulse and hepatic congestion, findings that would later carry diagnostic importance.[18,19]

A major therapeutic milestone occurred in 1929, when Churchill performed the first successful pericardiectomy, later reinforced by Paul Dudley White's clinical series showing favorable outcomes with surgical intervention.[20,21] The physiologic understanding of the disease advanced significantly in the mid-20th century with Bloomfield et al's documentation of equalization of diastolic pressures and the characteristic dip-and-plateau waveform, hallmarks of constrictive physiology.[22]

By the latter half of the 20th century, the condition's iatrogenic nature came into focus, with Kendall et al and others, highlighting postoperative cardiac surgery as a significant cause.[23–29] These historical insights have paved the way for today's understanding of CP, informing both diagnostic approaches and therapeutic strategies.

Pathophysiology

CP results from scarring, thickening, and often calcification of the pericardium, forming a rigid, noncompliant shell that restricts diastolic filling and limits total cardiac volume. The hallmark hemodynamic feature is the equalization of end-diastolic pressures across all four cardiac chambers—reflecting external constraint by the pericardium rather than intrinsic myocardial dysfunction.[1,30]

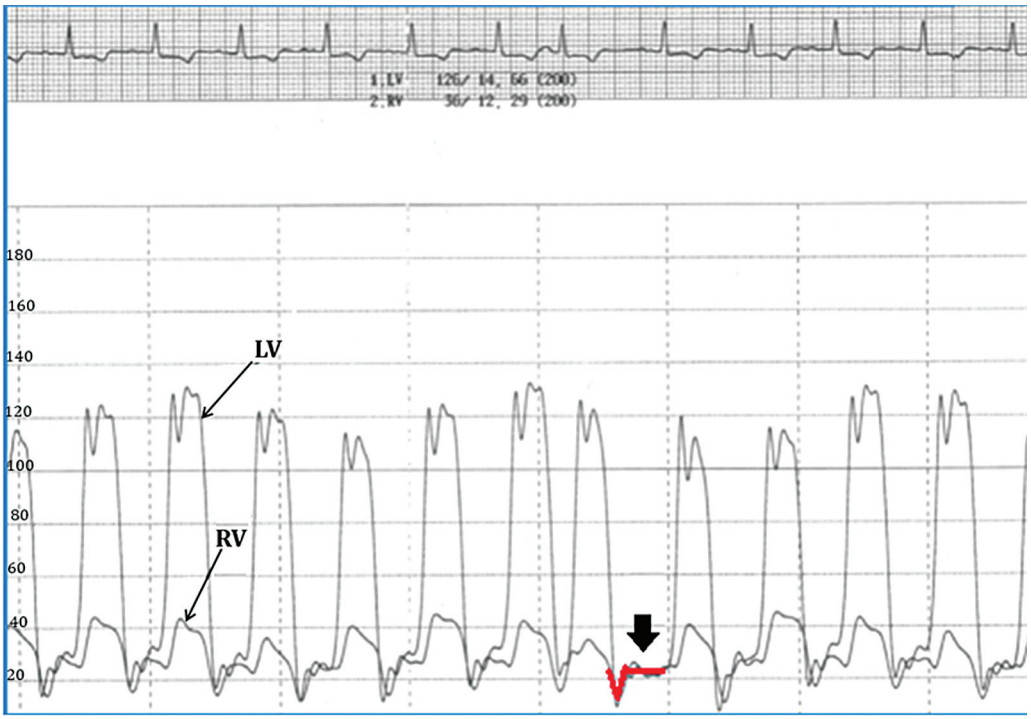

Fig. 1 Left ventricular (LV) and right ventricular (RV) pressures recorded simultaneously, showing that both pressures are equal throughout diastole. A long diastole (thick arrow) following a premature beat shows equalization of the LV and RV diastolic pressures and a "dip and plateau" pattern ("square root" sign).

In early diastole, rapid ventricular filling occurs due to pressure gradients between the atria and ventricles. However, once the fixed pericardial reserve volume is reached, filling is abruptly halted. This results in the characteristic "dip and plateau" or "square root" sign on ventricular pressure tracings, a key diagnostic clue[1,31–33] (►**Fig. 1**).

In CP, the thickened and noncompliant pericardium restricts the normal transmission of intrathoracic pressure changes to the cardiac chambers. As a result, right heart filling becomes impaired during inspiration, contributing to Kussmaul's sign—a paradoxical rise or lack of decline in jugular venous pressure (JVP) with inspiration. This impaired pressure transmission also leads to paradoxical interventricular septal motion during the respiratory cycle, a phenomenon known as septal bounce.[31,32] Septal bounce is characterized by the interventricular septum shifting toward the left ventricle during inspiration and toward the right ventricle during expiration, reflecting exaggerated ventricular interdependence caused by the encasing, rigid pericardium (►**Fig. 2**).

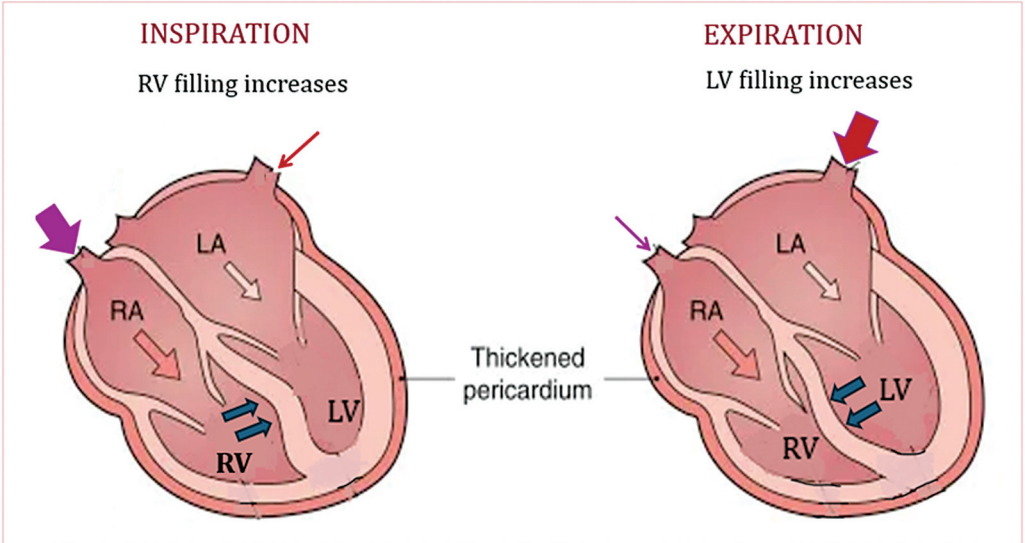

Fig. 2 Septal bounce (ventricular interdependence) during respiratory phases in constrictive pericarditis—due to noncompliant pericardium. RV, right ventricle; LV, left ventricle.

Clinical Presentation

CP often presents insidiously, with nonspecific symptoms such as malaise, lack of energy, and easy fatigability, which can delay diagnosis. The clinical picture is typically dominated by signs and symptoms of right-sided heart failure. Patients commonly report progressive peripheral edema, abdominal bloating with increasing abdominal girth, and right upper quadrant discomfort or pain due to hepatic congestion.[1,34,35]

A hallmark clinical sign is elevated JVP, often accompanied by a positive Kussmaul's sign, a paradoxical rise or failure of the JVP to fall during inspiration. While highly suggestive of CP, Kussmaul's sign is not specific and may also be observed in restrictive cardiomyopathy, pulmonary embolism, right ventricular infarction, and advanced right heart failure.[1,35]

On cardiac auscultation, heart sounds may be muffled due to the dense, fibrotic pericardium. A classic auscultatory finding is the pericardial knock—a high-pitched, early diastolic sound—reflecting abrupt cessation of ventricular filling. Although distinctive, this finding is not always present. Pulsus paradoxus is uncommon in classic CP but, if present, may suggest an effusive-constrictive variant.[1,2,35]

Additional physical findings include hepatosplenomegaly, ascites, and right upper quadrant tenderness from hepatic congestion. A characteristic clue is disproportionate peripheral edema in the absence of significant pulmonary rales, reflecting predominant systemic venous congestion.[1,35,36]

Etiology

Globally, tuberculosis remains the most common cause of CP, especially in low- and middle-income countries where the disease remains endemic.[4,5] In contrast, the etiology of CP in developed nations has shifted markedly in recent decades.

In high-income settings, the majority of cases are now attributed to idiopathic or viral pericarditis, accounting for approximately 42 to 49% of cases. This is followed by postcardiac surgery (11–37%) and chest radiation (9–31%) as leading contributors. Other notable etiologies include autoimmune diseases, malignancy, and postinfectious causes unrelated to tuberculosis.[33–37]

A retrospective review of 135 cases of surgically confirmed CP at the Mayo Clinic highlighted this shift in causation. The three most common identifiable etiologies were previous cardiac surgery (18%), idiopathic or viral pericarditis (16%), and mediastinal irradiation (13%). The study also revealed a male predominance, with 76% of cases occurring in men.[33,34]

Given this changing landscape, an understanding of contemporary risk factors is essential for early recognition. The next sections of this review will focus on the modern causes of CP in greater detail, including postcardiac interventions, postradiation, autoimmune, and iatrogenic contributors.

The Modern Landscape of CP

Postcardiac Interventions

(1) *Cardiac surgeries*, including coronary artery bypass grafting (CABG), valve replacement, and other open-heart procedures, are well-recognized contributors to the development of CP. The primary mechanism involves trauma and subsequent scarring of the pericardium incurred during or following surgical intervention.[23–29,33,37]

During cardiac surgery, the pericardium is typically opened to access the heart. This manipulation can result in direct injury to the pericardial layers, triggering acute inflammation. The disruption of the pericardial structure initiates a cascade of inflammatory responses that may persist beyond the perioperative period. Notably, postpericardiotomy syndrome (PPS)—a known postoperative complication—is characterized by pericardial inflammation that, if severe or unresolved, can evolve into chronic fibrosis and pericardial thickening.[5,29,30]

Intraoperative or postoperative bleeding into the pericardial space, whether due to incomplete hemostasis or coagulopathy, exacerbates the inflammatory process. Blood accumulation in the pericardial sac stimulates a fibrinous inflammatory response that may ultimately result in fibrotic scarring. Animal models have shown that pericardial adhesions form when spilled blood contacts injured serosal surfaces.[27,28] The extent of inflammation and fibrosis depends not only on the volume of blood but also on the severity of pericardial trauma.

Risk factors for developing postsurgical CP include longer operative times and extensive manipulation of the heart and pericardium. The timing of disease onset can vary widely—from as early as 3 weeks to decades after surgery—making high index of suspicion essential.[25,26]

Early-onset constriction is often driven by acute inflammation and unresolved surgical complications, while late-onset disease tends to reflect chronic scarring, fibrosis, and calcification. This temporal variability requires clinicians to maintain a high index of suspicion, especially in patients with compatible symptoms and a history of cardiac surgery.[22,25,26]

The true incidence of postsurgical CP is likely underestimated. Mild or subclinical cases may resemble low cardiac output syndrome—particularly in patients following valve surgery or CABG with underlying left ventricular dysfunction. These patients may respond to standard heart failure therapies, such as diuretics, and thus evade further diagnostic evaluation.[29,30]

Although PPS has been implicated as a risk factor, occurring in up to 30% of patients after cardiac surgery, its role remains uncertain. The presence of PPS does not reliably predict progression to constriction, and conversely, CP can occur in patients without any signs of PPS.[23,25,29]

(2) *Percutaneous coronary interventions* (PCIs), while a common and vital procedure, can occasionally result in CP, though this is a rare complication. A notable case reported in 2006 described a patient who developed CP 6 months following PCI. The patient presented with exertional dyspnea, excessive fatigue, progressive lower extremity edema, and increasing abdominal girth due to ascites. Transthoracic echocardiography and hemodynamic assessment via cardiac catheterization confirmed

the diagnosis of pericardial constriction, underscoring the potential role of PCI in triggering this condition.[6]

While the exact pathophysiological mechanisms remain somewhat unclear, coronary artery perforation during catheter or wire manipulation has been identified as a potential cause of CP following PCI.[9,10] However, this is generally not directly attributed to coronary perforation during the procedure itself. Rather, CP following PCI is typically a consequence of inflammation and subsequent fibrosis of the pericardium. This inflammatory response is often triggered by mechanical irritation, the use of contrast agents, or other procedural factors.

During PCI, catheters and guidewires are maneuvered within the coronary arteries and heart chambers. This manipulation can lead to minor trauma to the epicardial surface or pericardium, which in turn causes localized inflammation. Additionally, the use of iodinated contrast media during PCI can provoke an inflammatory reaction, particularly in susceptible individuals. In more severe cases, coronary artery perforation, although rare, can occur as a complication of PCI. If blood and contrast material leak into the pericardial space due to perforation, it can trigger acute pericarditis.[28] Chronic inflammation and the healing response may then lead to fibrosis and thickening of the pericardium, ultimately resulting in CP.[9,10]

(3) *Catheter-based ablation procedures*, particularly those used to treat atrial fibrillation (AF), have revolutionized the management of cardiac arrhythmias. However, while these procedures are generally effective, they can occasionally lead to complications involving the pericardium, including acute pericarditis and, in rare cases, CP.[38–41]

During catheter-based ablation, energy sources such as radiofrequency (RF) or cryoablation are used to create lesions in the myocardium to disrupt abnormal electrical circuits. While the primary target is myocardial tissue, the energy can inadvertently affect adjacent structures, including the epicardium and pericardium. Excessive heat from RF ablation or extreme cold from cryoablation can cause thermal injury to the pericardial layers.[39] This injury triggers an inflammatory response in the pericardium, which can lead to acute pericarditis. In rare instances, this inflammation may evolve into CP.

CP following catheter-based ablation for AF is primarily caused by thermal injury to the pericardium, microscopic perforations, or an exaggerated immune response. These insults induce acute inflammation, which, if unresolved, can progress to chronic fibrosis and calcification of the pericardium. The resulting rigidity impairs cardiac filling, leading to the clinical syndrome of CP.[9,39,41]

Radiation Therapy

Radiation-induced CP is a late complication of thoracic radiation therapy, often manifesting years to decades after exposure, particularly in patients treated for malignancies involving the chest, such as Hodgkin's lymphoma, breast cancer, and other mediastinal tumors.[11,12] Heart disease occurring after irradiation of the mediastinum was first recognized in the 1960s, when long-term survival after radiotherapy for Hodgkin's disease became frequent.[12] In recent years, with improvements in the techniques of radiotherapy and with greater use of chemotherapy, cases of heart disease after radiation have been fewer and less severe. However, since pericarditis may develop or become clinically manifest many years after the radiotherapy, new cases do continue to appear.[42]

Radiation therapy damages the pericardium through a combination of acute inflammatory responses and chronic fibrotic processes. Radiation causes direct injury to the pericardial cells, leading to an acute inflammatory response. This phase is characterized by pericardial effusion, which may be asymptomatic or present as acute pericarditis. Inflammation leads to increased vascular permeability and fibrin deposition in the pericardial space. Persistent inflammation triggers fibroblast activation and collagen deposition, resulting in thickening of the pericardium.[42–44]

Over time, chronic inflammation and oxidative stress lead to progressive fibrosis and calcification of the pericardium. The normally elastic pericardial tissue becomes rigid, impairing diastolic filling of the heart.

Contributing factors include higher doses of radiation (> 30 Gy) and larger treatment fields, which increase the risk of pericardial injury.[45,46] Preexisting cardiovascular disease, autoimmune conditions, or genetic predispositions may exacerbate the effects of radiation. Patients who undergo cardiac surgery after radiation therapy are at higher risk due to the cumulative trauma to the pericardium along with the myocardium.[45,47]

The latency period between radiation exposure and the development of CP can range from months to decades, with most cases occurring 5 to 20 years after treatment. Calcification is a hallmark of advanced disease and contributes to the restrictive physiology seen in CP.[12]

Autoimmune Diseases

Several autoimmune diseases have been associated with CP, though this manifestation is relatively rare. The progression from pericardial inflammation to constriction can occur if the initial inflammation is recurrent or inadequately treated.[48–51]

Systemic lupus erythematosus (SLE): SLE is one of the most common autoimmune diseases associated with pericardial involvement. Pericarditis is a frequent cardiac manifestation in SLE patients, and it can present as the first manifestation of SLE.[50] While CP is less common, it can develop as a complication of recurrent or chronic pericardial inflammation.[50]

Rheumatoid arthritis (RA): RA can involve the pericardium, leading to pericarditis. Although CP is rare in RA, it has been reported, particularly in cases with prolonged disease duration or inadequate control of systemic inflammation.

Pericardial involvement is a common occurrence in RA, affecting about one-third of patients with a prevalence from 30 to 50% and is frequently asymptomatic.[48,49,51]

Systemic sclerosis (scleroderma): Systemic sclerosis is known to affect the heart, including the pericardium.

Pericardial involvement can lead to effusions and, in rare cases, progress to CP due to fibrosis and calcification of the pericardial layers.[51]

Sjögren's syndrome: While pericardial involvement is uncommon in primary Sjögren's syndrome, cases of pericarditis and even CP have been documented. The pathogenesis is thought to involve immune-mediated inflammation leading to fibrosis.[52] In all these conditions, early recognition and treatment of pericardial inflammation are crucial to prevent progression to CP. Management typically involves immunosuppressive therapy tailored to the underlying autoimmune disease.[51]

Viral Pericarditis

Viral pericarditis is the most common cause of acute pericarditis in developed countries. Common causative agents include coxsackievirus B, echovirus, adenovirus,[30,36,53,54] and more recently COVID-19.[55–57] The typical course of viral pericarditis is self-limited; however, in rare cases, persistent or recurrent inflammation can lead to chronic fibrotic changes of the pericardium, ultimately resulting in CP.[30,36]

The mechanism is believed to involve an exaggerated or unresolved immune response following viral infection, leading to pericardial thickening, fibrosis, calcification, and loss of pericardial elasticity. These structural changes impair diastolic filling of the heart, causing the classic hemodynamic findings of CP. Clinical signs may appear weeks to months after the initial viral illness, and the diagnosis is often supported by imaging findings (e.g., pericardial thickening on computed tomography [CT] or magnetic resonance imaging [MRI]) and history of preceding viral symptoms.[30,36,53,54]

End-Stage Renal Disease Pericarditis

Pericarditis in end-stage renal disease (ESRD) can occur either before dialysis initiation (*uremic pericarditis*) or during maintenance dialysis (*dialysis-associated pericarditis*).[58,59] Uremic pericarditis has been arbitrarily defined as pericarditis that develops before or within 8 weeks of initiation of dialysis, while dialysis-associated pericarditis is used to define pericarditis in patients on dialysis for more than 8 weeks. Both forms result from the accumulation of uremic toxins, chronic inflammation, and fluid overload. Inadequate dialysis, persistent systemic inflammation, or secondary infections may exacerbate the condition.[58]

Patients with ESRD are particularly prone to developing pericardial calcifications, a hallmark of advanced constrictive physiology.[49,60] Risk factors for progression to CP include delayed or suboptimal dialysis, chronic fluid overload, and recurrent pericardial effusions.[58–61]

Clinical suspicion should be raised when dialysis patients present with signs of right heart failure (e.g., peripheral edema, ascites, jugular venous distention) that are not explained by volume overload alone. Diagnosis is confirmed through echocardiography, CT, or cardiac MRI, and management often requires both aggressive dialysis and, in severe cases, pericardiectomy.[58–61]

Diagnosis

The diagnosis of CP is often challenging and must be individualized based on clinical context. In some patients, a diagnosis may be suggested by the medical history, physical examination, and basic imaging such as chest radiography. In others, more advanced investigations, including echocardiography, cross-sectional imaging, and invasive hemodynamic studies, may be required.[35,37]

Timely diagnosis requires a high index of suspicion, informed by clinical presentation, imaging findings, and hemodynamic data. Fortunately, advancements in multimodality imaging, including echocardiography, cardiac CT, and cardiac MRI, have improved early recognition and accurate classification. Importantly, CP is a potentially curable condition; some patients respond to anti-inflammatory therapy, while others benefit from surgical pericardiectomy.[35]

A high index of suspicion remains essential, particularly in patients presenting with signs of right-sided heart failure that seem disproportionate to pulmonary congestion or left-sided cardiac disease. A comprehensive understanding of the disease's pathophysiology, along with judicious use of diagnostic tools, greatly enhances diagnostic accuracy, especially in cases where pericardial and myocardial involvement coexist.[33–37] Differentiating CP from restrictive cardiomyopathy is of critical clinical importance, as CP is a potentially reversible condition with surgical treatment, whereas restrictive cardiomyopathy typically carries a more progressive course with limited curative options.

Laboratory Evaluation

Inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein may be elevated in transient or subacute constriction and can help identify patients who might benefit from anti-inflammatory therapy.[35]

Liver function tests may show elevated enzymes and hyperbilirubinemia due to hepatic congestion from chronic right heart failure. Hypoalbuminemia can occur secondary to protein-losing enteropathy or proteinuria has been reported, which completely resolved following pericardiectomy.[62]

Natriuretic peptides (B-type natriuretic peptide [BNP], N-terminal-pro-BNP) are typically only mildly elevated, which can aid in differentiating CP from restrictive cardiomyopathy.[63]

Imaging

Chest X-ray may reveal pericardial calcification, which is pathognomonic when present in the appropriate clinical setting. Echocardiography is the first-line imaging modality for evaluating suspected CP. It is essential to consider CP in patients with prior pericarditis, pericardial effusion, cardiac surgery, interventional procedures, or chest radiation.

Two-dimensional and Doppler echocardiography can demonstrate the hallmark physiologic features of CP, such as: respiration-related ventricular septal shift (septal bounce)—the most sensitive marker (93%); preserved or increased medial mitral annular e' velocity along with reduced lateral mitral annular e' velocity, the so-called

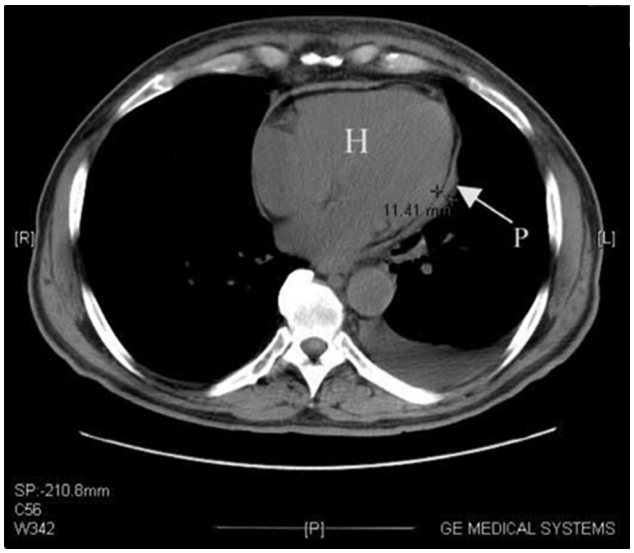

Fig. 3 Computed tomography (CT) of the heart (H) showing a thick pericardium (*P*; *arrow*).

"annular reversus"; and prominent expiratory diastolic flow reversal in hepatic veins.[36,64,65] These findings reflect the core pathophysiologic mechanisms of CP: dissociation of intrathoracic and intracardiac pressures and interventricular dependence within a fixed volume.

Cardiac MRI: Accurately assesses pericardial thickening (> 4 mm) with sensitivity of 93%.[66] Cardiac MRI pericardial late gadolinium enhancement (LGE) can identify active pericardial inflammation, guide anti-inflammatory therapy, and could predict the reversibility of CP after anti-inflammatory therapy. It can be performed serially in CP patients to follow disease progression and resolution with medical management, thus avoiding unwanted referrals to surgery.[67,68] It also helps differentiate CP from restrictive cardiomyopathy with high sensitivity and specificity, particularly by detecting myocardial delayed enhancement (present in restrictive cardiomyopathy but typically absent in isolated CP). Assesses myocardial pathology when cardiomyopathy is suspected.[66]

Cardiac CT: Provides excellent visualization of pericardial calcification, fibrosis, and thickening (►**Fig. 3**). Not influenced by body habitus and useful when echocardiography is suboptimal. Clearly demonstrates respirophasic septal motion and alternative causes of dyspnea (e.g., lung disease, diaphragmatic paralysis, etc.).[66,69,70]

It must be noted that pericardial constriction could happen in patients with normal pericardial thickness. Talreja et al[71] reported up to 18% of surgically confirmed CP cases may show normal pericardial thickness, underscoring that normal imaging does not exclude the diagnosis. Pericardial thickness was not increased in 18% of patients with surgically proven CP, although the histopathological appearance was focally abnormal in all cases. When clinical, echocardiographic, or invasive hemodynamic features indicate constriction in patients with heart failure, pericardiectomy should not be denied on the basis of normal thickness as demonstrated by noninvasive imaging.[71]

Invasive Hemodynamic Assessment

Cardiac catheterization remains the gold standard when noninvasive tests are inconclusive. Typical findings include equalization of diastolic pressures across all four chambers, dip-and-plateau (square root) sign in ventricular pressure tracings, and discordance between intrathoracic and intracardiac pressures, confirming the pathophysiology of constriction[36,64,65,70] (►**Fig. 1**).

The *Mayo Clinic diagnostic criteria* integrate echocardiographic and catheterization data, emphasizing ventricular interdependence and pressure dissociation unique to CP[72] (►**Table 1**).

Distinguishing CP from restrictive cardiomyopathy is critical, as pericardiectomy can cure CP, while treatment for restrictive cardiomyopathy is supportive.[69,70]

Treatment

The management of CP depends on the activity of the inflammatory process, the chronicity of the disease, the

Table 1 Mayo Clinic diagnostic criteria for constrictive pericarditis

Category	Findings/Criteria
Clinical findings	- Elevated jugular venous pressure (JVP) with Kussmaul's sign - Peripheral edema, ascites
Echocardiographic features	- Septal bounce (paradoxical septal motion) - Respiratory variation in mitral/tricuspid inflow (> 25%) - Annulus reversus (medial E′ > lateral E′) - Annulus paradoxus (E′ preserved despite elevated filling pressures)
Cardiac catheterization	- Equalization of end-diastolic pressures in all chambers - Dip-and-plateau (square root) sign - Ventricular discordance with respiration
Imaging (CT or MRI)	- Pericardial thickening (> 2 mm) - Pericardial calcification - Abnormal interventricular dependence
Additional findings	- Improvement with anti-inflammatory therapy (in subacute/inflammatory cases) - Biopsy/histology may show chronic inflammation or fibrosis

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging.

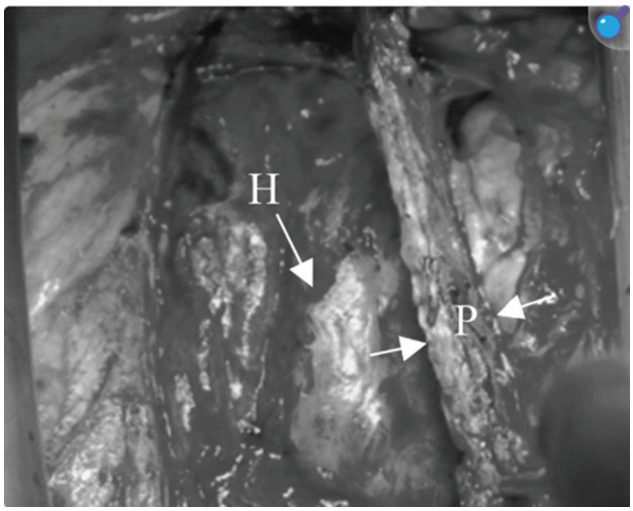

Fig. 4 Intraoperative photograph taken during surgical stripping of the pericardium (P), illustrating the markedly thickened and adherent pericardial tissue. H, heart.

severity of symptoms, and the underlying etiology. An individualized approach is essential, with treatment goals centered on alleviating symptoms, reducing inflammation when present, and, when necessary, performing surgical intervention to relieve pericardial constriction.[30,34,37]

In patients with active pericardial inflammation, a subset of them experiences reversibility of pericardial inflammation, a condition referred to as "transient constriction" may respond to anti-inflammatory therapy without surgical intervention.[34,73] Predictors of favorable response include chest pain, elevated inflammatory markers, and moderate to severe LGE of the pericardium on cardiac MRI. These features suggest ongoing inflammation that may still be reversible.[34,67,68] Thus, detection of pericardial inflammation may be useful, as it may identify patients with transient CP who are good candidates for anti-inflammatory therapy.

When CP becomes chronic and the pericardium progresses to fibrosis and calcification, medical therapy alone is insufficient. In these cases, the definitive treatment is radical pericardiectomy, which involves surgical excision of the thickened, fibrotic pericardium (►**Fig. 4**). This procedure restores normal cardiac filling by removing the physical constraint around the heart. Early surgical intervention, before the development of significant myocardial dysfunction, is associated with better hemodynamic recovery and long-term survival. However, pericardiectomy carries substantial perioperative risk, especially in patients with advanced disease, comorbidities such as chronic kidney disease, or a history of prior cardiac surgery.[33,65,74]

Conclusion

CP is a complex condition with shifting etiologies—from tuberculosis to modern causes like cardiac interventions and radiation therapy. Advances in imaging (MRI, CT) and hemodynamic assessment have refined diagnosis, while treatment hinges on distinguishing reversible inflammation (managed medically) from chronic fibrosis (requiring pericardiectomy).

Perspective

Looking ahead, the evolving landscape of CP demands heightened vigilance, particularly among oncologists, cardiologists, and rheumatologists managing at-risk patients. Future research should explore biomarkers for early detection and refine pericardiectomy techniques in high-risk groups. Bridging historical insights with contemporary practice will be key to improving outcomes in this potentially curable disease.

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Conflict of Interest
None declared.

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