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Chapter

Constrictive Pericarditis

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Abstract

Constrictive pericarditis (CP) is a challenging clinical scenario in which the heart muscle is entrapped by thick, fibrous, and frequently calcified pericardial layers. Whereas infectious diseases (mostly bacterial) had been observed as the main etiology in the last decades, nowadays, post-surgical or radiotherapy iatrogenic inflammation is becoming highly prevalent with the exception of developing countries and patients with immunodeficiency in which tuberculosis is still frequently observed. Clinically, progressive dyspnea and peripheral edema are present and frequently considered of unknown origin because of the diagnostic challenge that CP poses. As a matter of fact, a specific knowledge of echocardiography and right heart catheterization is essential to recognize constriction features. Moreover, a valuable support is provided by dedicated imaging modalities (mostly magnetic resonance). Complete surgical removal of the pericardium (pericardiectomy), when feasible and performed early, is associated with excellent symptomatic improvement. Unfortunately, in specific scenarios (radiation therapy) or when surgery is performed after severe constriction development, surgical outcomes are poor, and CP assumes the profile of an end-stage disease. This reinforces the unmet need of early detection of CP and the development of novel therapeutic strategies.

Keywords: constrictive pericarditis, right heart catheterization, pericardiectomy, heart failure, restrictive cardiomyopathy

1. Introduction

Constrictive pericarditis (CP), firstly described by Richard Lower in “Tractatus de Corde” (1669), is the end-stage evolution of chronic inflammation and fibrosis of pericardium. Many of the causes of acute pericarditis listed in previous chapters have been associated with the development of pericardial constriction weeks to months after the acute episode. Nonetheless, the appearance of progressive dyspnea and peripheral edema are not promptly addressed to CP and are frequently considered of unknown origin because of the diagnostic challenge that CP poses [1]. As a matter of fact, diagnosis of CP can be challenging because of the low prevalence (<1% of acute pericarditis, 0.2–0.4% of patients submitted to cardiac surgery) and subsequent detrraining of echocardiography specialists. Moreover, constrictive hemodynamics can be difficult to distinguish from restrictive cardiomyopathy (RCM) physiology, a primary myocardial disease, and finally, pericardium could present a normal thickness in almost 20% of CP cases with calcifications present in less than half of patients [2].
2. Etiology

Traditionally, CP had been described months to years after acute bacterial pericarditis, whereas in the last decades, chronic inflammation associated with previous thoracic surgery or radiotherapy has become the most frequent cause of CP. A recent meta-analysis of patients submitted to pericardiectomy for symptomatic CP [2] has collected data about 2114 patients admitted between 1991 and 2019. Idiopathic etiology was present in approximately half of patients (50.2%) followed by post-cardiac surgery (26.2%) and mediastinal radiotherapy (8.9%). Interestingly, studies published after 2000 have reported a dramatic decrease of cases secondary to cardiac surgery with respect to previous reports (15% vs. 33%, p < .001). This could reflect the evolution of cardiac surgery techniques with progressively reduced operative times and close echocardiographic evaluation after surgery.

Moreover, end-stage renal disease, connective tissue disorders (i.e., lupus erythematosus, rheumatoid arthritis, systemic sclerosis, etc.), and pulmonary diseases, including pulmonary asbestosis and mesothelioma infiltrating the pericardium, are less frequent but important causes of CP. More exceptional is CP secondary to transmural myocardial infarction (Dressler’s syndrome), given the spread of primary angioplasty and, consequently, the reduction of infarct size, in developed countries. Finally, CP secondary to tuberculosis infection in developed countries has been reported to be a rare condition (3%) with an increasing trend in the last decades due to imported cases and the spread of HIV infections. Nonetheless, taking into account socioeconomic background, tuberculosis infections and, along with them, late complications like CP are significantly increasing in developing countries. Consequently, tuberculosis has become the first cause of CP in countries of sub-Saharan Africa and few countries of Asia, including India where tuberculosis was associated with more than half of cases of CP (51.6%) in a retrospective single-center analysis, including patients submitted to pericardiectomy between 2009 and 2020 [3].

3. Pathophysiology

Chronic pericardial inflammation usually drives a structural change of pericardial layers, resulting in progressive fibrosis and calcifications and leading to partial adhesions between the layers. Consequently, difficulties in diastolic ventricle filling can be observed. As a matter of fact, during diastole, ventricles experience an active (ATP-consuming) relaxation with a rapid decrease of chamber pressures leading to mitral valve opening and early inflow with a velocity as higher as the pressure gradient between atrial and ventricular chambers. This phase is usually followed by an atrial contraction leading to further ventricle filling with no significant increase of end-diastolic ventricular pressures unless pathologic conditions, like CP, occur. In case of CP, given the reduced compliance of pericardium and its reduced stretching, diastolic pressures of the ventricles rapidly increase, and consequently, ventricular filling abruptly ceases during early to mid-diastole, when cardiac volume reaches the limit set by non-compliant pericardium. Thus, atrial emptying will be incomplete, leading to the increase of atrial and pulmonary/systemic venous pressure [4]. Systemic venous congestion results in hepatic congestion, peripheral edema, and ascites and, if long-standing, in cardiac cirrhosis and symptoms secondary to low cardiac output (Figure 1). As a matter of fact, although left ventricle (LV) ejection fraction is usually normal, the absolute reduction of diastolic filling, due to pericardial
Constrictive Pericarditis
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reduced compliance, leads to reduced cardiac output and, consequently, to fatigue and reduced functional class. Finally, physiologic reduction of intrathoracic pressure during inspiration acts on lungs and pulmonary veins, as usual, but will not be transmitted to the heart (heart-lungs decoupling) because of the limited heart diastolic compliance (myocardial relaxation, after reaching maximal diastolic volume limited by pericardium incompliance, fails to increase linearly with respect to negative intrathoracic pressure that usually drives a suction phenomenon). Consequently, whereas in normal hearts, trans-mitral flow increases during inspiration, as a consequence of this suction phenomenon, in patients with CP, it is reduced because pulmonary veins have a negative pressure with respect to the left atrium, resulting in a reduction of forward blood flow, and, finally, to LV diastolic filling [5]. This phenomenon clearly does not apply to right ventricle (RV) because inspiratory negative pressure is not applied to systemic veins (originating outside of thorax), and consequently, venous pressure is not lower than right atrial pressure so that diastolic filling of RV is less reduced than LV during inspiration (Figure 2). This right-left mismatch and ventricle inter-dependence secondary to thickened non-compliant pericardium (expansion of one ventricle occurs at expenses of the other one because both are into a rigid pericardial envelope) explain the reason why, as explained before, during inspiration, LV filling decreases whereas RV filling increases with secondary leftward interventricular septal shift (septal bounce). Conversely, during expiration, pulmonary vein pressure increases, driving forward blood flow and, therefore, increasing trans-mitral flow.

Figure 1.
Constrictive pericarditis physiology. During early to mid-diastole, ventricular filling (red arrows) is limited by pericardial thickness and incompliance (A) that resist to myocardial relaxation (white arrows) and, therefore, limiting ventricular preload to the early diastolic phase as showed by trans-mitral Doppler inflow pattern characterized by elevated and short “e”-wave and a minor “a”-wave (B) and sharp “y” descending wave at jugular vein pulse (C). Limitation to ventricular filling is, finally, associated to peripheral vein congestion as highlighted by dilation of inferior vena cava (D).
In this case, right ventricle filling, expressed as trans-tricuspid flow, decreases, and leftward interventricular septal deviation disappears [6].

4. Clinical “red flags”

When approaching a patient with CP, jugular venous pressure (JVP) increases, with specifically an abnormal increase during inspiration explicated by the incompilance of pericardium that impedes the venous blood, that usually increases because of suction inspiratory forces, to enter into the RV. This phenomenon (Kussmaul’s sign) is opposite to normal condition when, during inspiration, usually JVP falls because more blood enters into RV. Moreover, JVP increase is associated to peculiar invasive pressure patterns (sharp descending “y” wave associated to rapid ventricle diastolic inflow and, if sinus rhythm is present, sharp descending “x” wave associated with late systolic inflow produced by atrial contraction) [7]. Consequently, the presence of peripheral edema, hepatomegaly, and, eventually, ascites are the cornerstone of clinical assessment. Finally, although the presence of restricted RV diastolic filling, most of all during inspiration when the higher venous return is not followed by an increase of ventricular filling, tricuspid flow increases with respect to mitral flow, explaining the presence, frequently, of a peripheral pulse markedly diminished, or even abolished, during ordinary or quiet inspiration (paradoxical pulse) secondary to the decrease of LV filling and, consequently, the drop of systolic pressure (>10 mmHg) during inspiration.

Finally, an essential clinical feature of CP is a pericardial knock, a high-pitched sound that occurs in early diastole and is best heard at the left sternal border and/or the cardiac apex [8].

5. Non-invasive imaging “red flags”

The essential technique for non-invasive assessment of CP is echocardiography that, moreover, is widely available and cost-saving. Therefore, it should be considered a first-line exam in patients with suspicion of CP. This is because, beside echocardiography,
Constrictive Pericarditis
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Diagnosis of CP can be challenging with other techniques given the fact that there are no specific findings pathognomonic of pericardial constriction. As a matter of fact, atrial fibrillation at EKG can be a frequent finding in this subset of patients but, given its high prevalence in general population, lacks adequate specificity. On chest X-rays, pericardial calcification is not always seen and, when encountered, is not necessarily an expression of constrictive physiology [9]. Moreover, another useful but not specific marker of CP is pulmonary vascular congestion and redistribution on chest X-rays, secondary to the increase of LV filling pressures. Similarly, a CT scan permits to detect even small spots of pericardial calcification and minor increases of pericardial thickness (>2 mm) [10]. This applies also to MRI, without the need for iodinated contrast or ionizing radiations but with reduced accuracy than CT in detecting small calcifications and measuring thickness ([Figure 3](#)). Nonetheless, although pericardial morphology can be described precisely, physiologic repercussions of CP on ventricular diastole cannot be estimated directly and can be only presumed by hepatic venous congestion, ascites, and pleural effusions. A step-forward is obtained by Cine acquisition in which ventricular-wall-motion abnormalities and ventricular-contour distortion secondary to localized adhesions to pericardium (corresponding to areas of major pericardial calcifications) can be visualized, and moreover, ventricular inter-dependence can be derived by leftward interventricular septal shift during early diastole ([septal bounce](#)) [11].

Consequently, given the limitations of the aforementioned non-invasive techniques, echocardiography, when feasible (optimal ultra-sonographic window is not always available), is the exam of choice in this subset of patients. Importantly, a respirometer is mandatory in order to detect respirophasic changes of ventricular diastolic filling and septal movements [12].

Similar to Cine CT or MRI, first step of assessment of CP by echocardiography is the observation of thickened pericardium [13], with or without areas of tethering on myocardium, usually at the level of the right free wall, appreciated on sub-costal and apical 4-chambers views. In addition, septal bounce phenomenon during inspiration, a constant finding of CP, can be highlighted as a septal notch in an M-mode long-axis parasternal view [14].

Moreover, dilation of supra-hepatic and inferior vena cava is observed in a sub-costal view in almost all patients with CP.

Central role in echocardiographic assessment of CP is played by Doppler hemo-dynamic evaluation. Most of the times, Doppler findings can confirm constrictive physiology without the need for invasive confirmation. Mitral and tricuspid inflows are characterized by high early diastolic velocities (E wave) with short deceleration time and significant respiratory variations in 2/3rd of patients [15]. Mitral E wave variation
Pericarditis - Diagnosis and Management Challenges

>25% (minimum at the end of inspiration) and tricuspid E wave variation >40% (maximum at the end on inspiration) are considered pathognomonic of CP, although absence of respiratory variation does not exclude the diagnosis (Figure 3). As a matter of fact, the presence of respiratory variations of mitral- and tricuspid-inflow-Doppler patterns alone can be present also without CP, like in patients with severe COPD due to higher respiratory variations of intra-thoracic pressures. Nonetheless, patients with COPD present a marked increase of inferior vena cava and supra-hepatic vein systolic forward flow velocity, whereas in patients with CP, this increase is blunted [16]. It is important to remember that in patients with CP, during inspiration, tricuspid flow is relatively increased with respect to mitral flow, but absolute flow is limited by pericardial constriction and, therefore, cannot increase significantly. A higher positive predictive value (96%) is offered by supra-hepatic-vein-Doppler pattern characterized by a decrease of expiratory diastolic forward velocities with large expiratory diastolic reversals.

Another useful parameter to detect CP is obtained by mitral annular tissue Doppler assessment of early diastolic velocity (e’wave), with evidence of “annulus reversus”, consistent with similar or slower lateral-wall relaxation with respect to septal wall (e’septal/e’lateral ratio > 0.91) when in normal conditions the opposite occurs, that has 95% of positive predictive value of CP [17]. Interestingly, absolute values of medial e’waves could be normal (septal e’wave > 9 cm/sec) or even increased (annulus paradoxus) despite the evidence of increased LV diastolic pressures (ratio E/e’ > 13) [18]. This phenomenon is explicated by the predominance of longitudinal cardiac relaxation of septum during LV diastolic filling because lateral wall is frequently entrapped and tethered by thick and calcified pericardium. This marker, also, has 95% of positive predictive value for CP diagnosis (Figure 4). Unfortunately, both annulus reversus and annulus paradoxus are affected by a significant proportion of false negative results (50% and 57% of negative predictive values, respectively). Consequently,

Figure 4.
Tissue Doppler characteristics of constrictive pericarditis. Contrary to normal physiology, in constrictive pericarditis lateral wall relaxation velocity (white arrow) during early diastole (e’wave) is usually is frequently reduced in comparison with septal e’wave (annulus reversus). This occurs because lateral wall is frequently entrapped and tethered by thick and calcified pericardium (white line). Moreover, although diastolic filling restriction is present, septal e’wave can be normal (> 8 cm/second) and can be used as a reliable marker of constriction in order to differentiate the latter with myocardial restriction (annulus paradoxus).
Constrictive Pericarditis
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A multi-parametric approach is warranted to assess a patient with suspected CP, and in doubtful cases or when quality of echocardiography is sub-optimal (challenging acoustic window, respirometer not available), a multi-modal assessment with CT and/or MRI can be helpful in identifying calcifications, ventricular wall distortions, septal bounce during inspiration, and systemic vein congestion [19]. Finally, if doubts still persist, right- and left-heart catheterization is essential for invasive pressure measurement and, therefore, demonstration of equalization of RV and LV-end-diastolic pressures secondary to the non-compliance of pericardium, a common constraint for both ventricles.

6. Invasive hemodynamic assessment

Simultaneous right- and left-heart catheterization is currently the gold standard for the diagnosis of CP. Evidence of equalization of end-diastolic pressures (≤ 5 mmHg difference between right- and left-ventricle-end-diastolic pressures secondary to fixed pericardial volume and consequent ventricular interdependence) and the visualization

![Figure 5](https://example.com/figure5.png)

*Invasive pressure measurement characteristics of constrictive pericarditis. Constrictive physiology is characterized by sharp ventricular pressure increase after early diastolic inflow that, in comparison with normal hearts, is highlighted by a peculiar diastolic pressure curve morphology known as “square root” or “dip and plateau” (panel A). Moreover, ventricular interdependence (panel B) is another crucial marker of constriction and is demonstrated by equalization of end-diastolic pressures (≤ 5 mmHg difference between RV and LV end-diastolic pressures) secondary to fixed pericardial volume in which both ventricles are moving (*). The same mechanism, added to the dissociation between intra-cardiac and intrathoracic pressures, explains the evidence of RV and LV opposite respiratory variations (panel B). This is also a useful marker to distinguish pericardial constriction from myocardial restriction (in this case, LV pressure varies in the same manner whereas RV pressure remains stable as its preload is not influenced by intrathoracic pressure and both ventricles are independent from each other).*
of “square root” or “dip and plateau” sign (secondary to sharp ventricular-pressure increase when pericardial constraining volume is reached immediately after early diastolic inflow) are considered the most important features for the diagnosis [20]. Another important marker of constriction is the presence of significant respiratory variations of LV and RV systolic and diastolic pressures as a consequence of dissociation between intracardiac and intrathoracic pressures (Figure 5). This has been quantified using the systolic area index (ratio of RV to LV systolic pressures x time area during inspiration). If >1.1 (RV pressure increasing while LV pressure decreasing during inspiration), it is highly suggestive of CP. Moreover, Kussmaul’s sign, quantified as <5 mm Hg decrease in right atrial pressure during inspiration, is often encountered. It is worth mentioning that hypovolemia secondary to previous aggressive diuretic therapy can mask hemodynamic features described above. An important tip in these cases is to perform a fluid challenge with rapid infusion of saline (500–1000 ml over 5–10 min) before assessment [21]. Finally, not specific but important findings at invasive assessment are also the reduction of stroke volume (as per Frank-Starling effect secondary to reduced diastolic filling) and the maintenance of pulmonary artery pressures within or mildly above upper normal limit, explaining the higher prevalence of right instead of left heart-failure symptoms.

7. Constrictive versus restrictive physiology: differences and similarities

Clinical spectra of CP and RCM frequently overlap given the defect of diastolic ventricle filling that is common to both diseases. Anyway, as a specific treatment for each of them is present, a correct differential diagnosis is mandatory. RCM is characterized by increased myocardial stiffness and, therefore, increased ventricular filling pressures in both the systemic and pulmonary circulations with the increase of both mitral and tricuspid inflows during inspiration. Differently, CP is characterized by discordant respiratory flow variations in RV and LV (ventricular interdependence) (Figure 5), frequently accompanied by the paradoxical pulse sign and septal bounce pattern and, predominantly, by systemic venous congestion. That makes the presence of symptoms and signs of pulmonary edema and congestion more frequent in RCM than in CP. This phenomenon is also reflected by the presence of severe post-capillary pulmonary hypertension in RCM, whereas it is almost absent in patients with CP. Similarly, although both present a “square root” morphology of ventricular diastolic pressure, tele-diastolic pressures of LV and RV are usually equal in CP, whereas LV has usually a higher pressure (4–5 mmHg more) than RV in RCM [22]. Finally, from a clinical perspective, the presence of pericardial knock suggests CP diagnosis.

Moreover, non-invasive imaging modalities, like echocardiography, CT scan, and cardiac MRI, are helpful in the diagnostic process as the presence of pericardial calcifications and/or increased pericardial thickness suggest CP, whereas ventricular hypertrophy (with or without delayed gadolinium enhancement at MRI) and marked atrial enlargement suggest RCM. Finally, myocardial tethering by adhered pericardium is present in CP (absent in RCM) and is accompanied by LV shape deformations and/or reduced circumferential restoration and speckle-tracking examination with normal longitudinal restoration. On the contrary, in RCM, circumferential restoration is normal, whereas longitudinal restoration is reduced. Similarly, e’lateral is equal or slower than e’medial in CP (annulus reversus), whereas it is the opposite in patients with RCM (Figure 4). Medial e’wave is usually normal in patients with CP (reflecting
Constrictive Pericarditis
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the absence of myocardial disease), whereas it is reduced in patients with RCM (annulus paradoxus) [23]. Finally, in doubtful cases, endomyocardial biopsy could confirm or exclude RCM [24].

8. Treatment

Surgical pericardiectomy is the treatment of choice of CP with acceptable outcomes in the long-term [25–28]. Nonetheless, diuretic therapy is the first-line treatment of CP, often started even before proper diagnosis is obtained, and permits initially to control mild symptoms and reduce venous congestion. Unfortunately, as CP progresses, patients become refractory to diuretics and maintain an adequate cardiac output with compensatory sinus tachycardia. This is the reason why beta-adrenergic blockers, verapamil, and diltiazem should be avoided. In case of high-rate supraventricular arrhythmias, digoxin is the negative chronotropic agent of choice. In the specific subset of patients developing CP early (<3 months) after cardiac surgery, treatment with nonsteroidal anti-inflammatory agents, colchicine, and steroids for at least 3 months has been proposed. Predictors of success of this strategy are increased biomarkers of systemic inflammation (hsCRP) and evidence of significant pericardial inflammation visualized as intense delayed enhancement on MRI [29, 30]. Nonetheless, surgical pericardiectomy should not be delayed in case of failure of initial anti-inflammatory strategy as earlier surgery is associated with improved outcomes. In patients without ongoing acute pericardial inflammation or with long-standing symptoms, surgical pericardiectomy is the first-line treatment. Different surgical approaches have been described (on- versus off-pump, median sternotomy versus mini-invasive thoracotomy) without a clear benefit of one of them. Complete pericardiectomy, defined as extensive excision of pericardium up to superficial epicardium, if involved, anteriorly between the 2 phrenic nerves and from the great arteries superiorly to the diaphragm inferiorly, posteriorly between the left phrenic nerve to the left pulmonary veins,

Figure 6.
Surgical approach to constrictive pericarditis. Complete pericardiectomy with extensive excision of pericardium anteriorly between the 2 phrenic nerves and from the great arteries superiorly to the diaphragm inferiorly (left panel) and posteriorly between the left phrenic nerve to the left pulmonary veins is considered the gold standard for treatment of CP. When extensive calcification is encountered, a less invasive approach with multiple transverse and longitudinal incisions up to the epicardial layer ("waffle" procedure) with the help of a dedicated ultrasonic scalpel (*) is considered a valid alternative (right panel).
including the diaphragmatic wall of left ventricle, is highly recommended. In case of severe calcification, it can be associated with ultrasound or laser debridement. Moreover, a less invasive approach with multiple transverse and longitudinal incisions on the epicardial layer (“waffle” procedure) has been proposed in patients with extensive calcific involvement of visceral pericardium and epicardium (Figure 6) [31, 32]. Despite long-standing experience in this procedure, pericardiectomy has a relatively high perioperative mortality rate (2–20%) associated with frequently reported low cardiac output. Predictors of poor perioperative outcomes are post-radiation CP, comorbidities (COPD, renal insufficiency, coronary artery disease, etc.), prior cardiac surgery, significant cardiac involvement (reduced LV systolic function, myocardial fibrosis/atrophy, severe tricuspid regurgitation), cardiopulmonary bypass, and poor functional status (New York Heart Association (NYHA) stage IV symptoms) [26]. Therefore, safety concerns about post-operative complications explain current indication to manage conservatively elderly healthy subjects, most of all in the presence of mild constriction, with pericardiectomy as a second-line therapy in case of progression. Similarly, patients at higher operative risk like elderly patients with severe symptoms and comorbidities are considered at prohibitively high risk, whereas radiation-induced CP is considered a relative contraindication to surgery.

9. Prognosis

Pericardiectomy, if performed early after diagnosis, is usually associated with acceptable quality of life. Symptomatic relief (associated with diastolic function recovery in up to 50% of cases) usually occurs immediately after surgery or, only in a small proportion of patients, after few months [27]. Long-term survival rates, unfortunately, remain moderately acceptable, despite surgical advances in the last decades, as reported in a meta-analysis of patients submitted to pericardiectomy [2] in which pooled all-cause 1-year and 5-year mortality rates after pericardiectomy were 17.4% and 32.7%, respectively. Interestingly, patients enrolled after 2000 had higher 1-year and 5-year all-cause mortality rates compared with before 2000 (19.8% vs. 10%, p = 0.01, and 49.4% versus 20%, p < 0.001, respectively). This possibly reflects the shift that occurred in the last decades toward more complex and recurrent etiologies of CP like cardiac surgery or mediastinal radiotherapy. As a matter of fact, patients with CP secondary to cardiac surgery have significantly higher risk of all-cause mortality after pericardiectomy when compared with patients with idiopathic etiology (HR: 2.15; 95% CI: 1.21 to 3.61, p = 0.01), with even worse outcomes when CP secondary to radiotherapy is compared with idiopathic etiology (HR: 3.21; 95% CI: 1.56 to 6.50, p < 0.01) [2]. Finally, pericardiectomy performed in patients with CP secondary to tuberculosis, the most common etiology observed in developing countries, has been recently reported to have similar outcomes with respect to other etiologies, although with more technical complexity in terms of increased operative time, more blood loss, and prolonged ICU and hospital stay [3].

10. Conclusion

Diagnosis of CP in the context of patients with signs and symptoms of heart failure can be challenging, and frequently, distinguishing it from RCM can be difficult. Firstly, thinking about CP when evaluating patients with diastolic dysfunction, most
of all after cardiac surgery or mediastinal radiotherapy, is crucial to recall in our minds all the characteristics of CP and make it possible to address the correct diagnosis. CP should also be suspected when ventricular filling restrictions are observed few months after a tuberculosis infection, taking into account that tuberculosis is the first cause of CP in countries of sub-Saharan Africa and few countries of Asia.

Secondly, the use of multimodality imaging is the cornerstone for the diagnosis of CP and the evaluation of the extension of the disease and, finally, to guiding surgical treatment. In doubtful cases, we should not hesitate to ask for invasive pressure assessment, safe and diagnostic in the majority of the cases. Finally, long-terms results of surgery in patients with chronic end-stage disease are poor, most of all when CP is secondary to previous cardiac surgery or radiation therapy, also with less invasive surgical strategies like “waffle” procedure. Consequently, new therapeutic strategies are strongly warranted. Meanwhile, an early diagnosis could make the difference in the natural history of the disease and, therefore, should be actively promoted.

**Acronyms and abbreviations**

- CP: constrictive pericarditis
- RCM: restrictive cardiomyopathy
- LV: left ventricle
- RV: right ventricle
- JVP: jugular venous pressure
- CT: computed tomography
- MRI: magnetic resonance imaging
- hsCRP: high sensitive C reactive protein
- NYHA: New York Heart Association

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