Chapter 3
Constrictive Pericarditis

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Abstract The diagnosis of constrictive pericarditis requires a high degree of clinical suspicion, for the signs and symptoms of this disease can be falsely attributed to other causes. Herein, we present A 67 year old woman, known case of breast cancer from 17 years ago, whose symptoms of right heart failure were initially attributed to cardio-toxicity due to chemotherapy.

Case Presentation

Herein, we present a 67-year-old woman, known case of breast cancer from 17 years ago, treated by surgery in 1381/8 and followed by 7 courses of chemotherapy and 27 courses of radiotherapy until 1382/2, after that she was followed by her oncologist.

She demonstrated dyspnea on exertion FCII from 2 years ago and orthopnea and abdominal distention from 4 months ago and was admitted to another center with impression of decompensating heart failure with a left ventricular ejection fraction of 30%.

She was discharged. One month later he presented with worsening breathlessness and ascites. Echocardiography, in our center revealed the diagnosis of constrictive pericarditis (CP) and CMR confirmed that. She underwent near-complete pericardiectomy and has made a good recovery. This case exemplifies the difficulty in diagnosing this condition, the investigation by echocardiography, and the benefit of surgery.

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Clinical and Echocardiographic Findings and Discussion

CP or Restrictive Cardiomyopathy (RCM) should be considered in any patient with the disproportional degree of right-sided HF with NL systolic and valve function. With a history of prior surgery, pericarditis, and radiation CP is more likely. Pericardial calcification seen on chest radiography is helpful but occurs in only 23% of patients and should raise the suspicion of TB pericarditis. Calcification per se is not diagnostic for CP.

The clinical and hemodynamic profiles of restriction (myocardial diastolic heart failure) and constriction (pericardial diastolic heart failure) are similar, although their pathophysiologic mechanisms are distinctly different.

Both are caused by limited or restricted diastolic filling, with relatively preserved global systolic function (Fig. 3.1).

Currently, the most common cause of constriction is previous cardiac surgery, followed by pericarditis, an episode of pericardial effusion, and radiotherapy. In the developed world the cause is most commonly idiopathic, postsurgical, or radiation injury.

In CP, diastolic dysfunction is related to a thickened or noncompliant pericardium, but Diastolic dysfunction in RCM or myocardial disease is the result of a stiff and noncompliant ventricular Myocardium, frequently RCM resulting from infiltrative cardiomyopathy is the easiest abnormality to diagnose typical 2D and biochemical features. A non-infiltrative type of RCM is more difficult to diagnose.

In CP Thickened, inflamed, adherent, or calcific pericardium forms a rigid shell that limits diastolic filling of the heart, in up to 20% of patients. The pericardial thickness is relatively normal but adherent to the epicardium pericardial thickness may be normal.

Atrial enlargement is less prominent in CP than in restrictive cardiomyopathy, but it can be as large.

To establish the diagnosis of CP, the following two hemodynamic characteristics need to be demonstrated either with 2D or Doppler echocardiography or with cardiac catheterization:

1. Disassociation between intrathoracic and intracardiac pressures and
2. Exaggerated ventricular interdependence

2D Echocardiographic Features of CP

- Thickened pericardium
- Abnormal ventricular septal motion

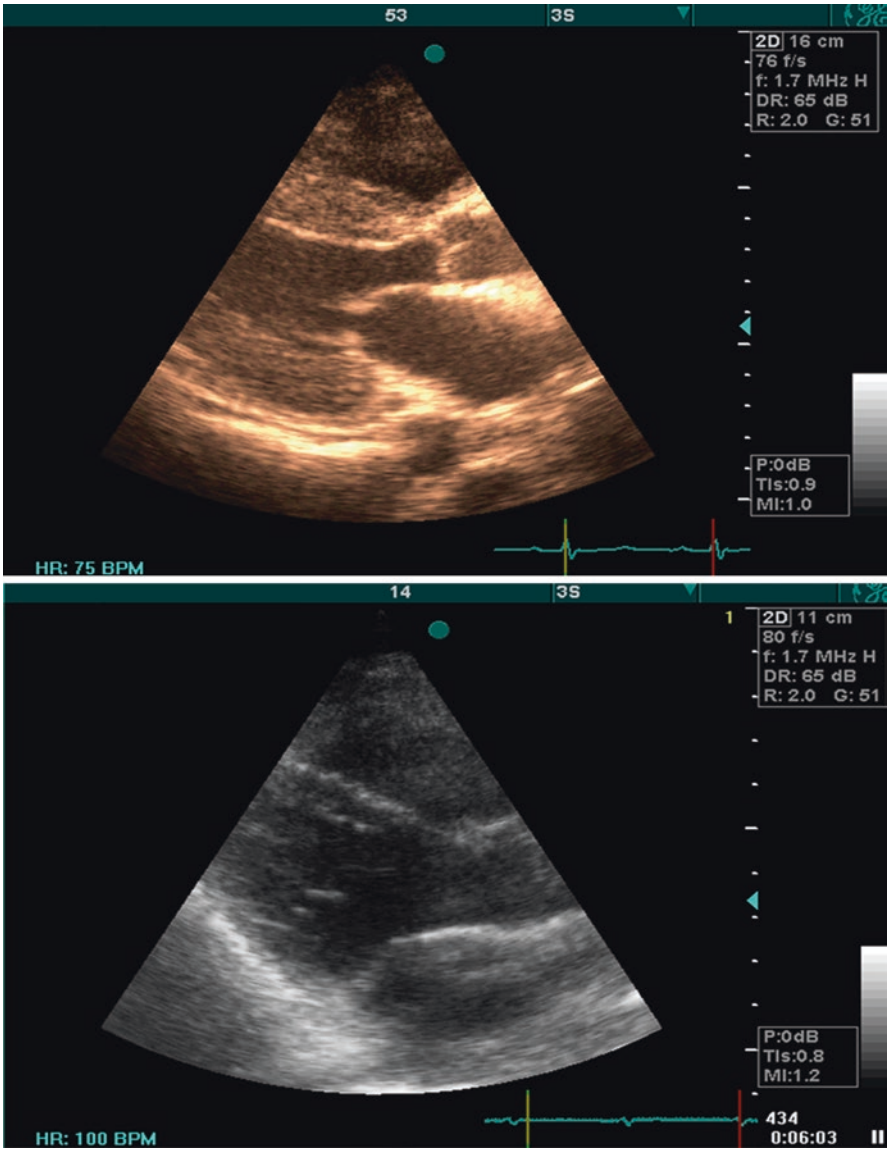

Fig. 3.1 Upper: Myocardial diastolic heart failure due to RCM. Lower: Pericardial diastolic heart failure due to CP and thickened pericardium

- Respiratory variation in ventricular size
- Dilated hepatic vein and inferior vena cava

But these findings are not sensitive or specific

M-Mode Echocardiographic Features of CP

- Flattening of the LV posterior wall during diastole and thickening (Fig. 3.2).
- Abrupt displacement of the IVS during early diastole (Septal bounce) Fig. 3.3, Upper.
- Septal motion reflects the competitive filling of the two ventricles Fig. 3.3, lower with constriction ventricles that may fill in an alternate fashion and produce a wavy pattern of diastolic septal motion.
- Premature pulmonic valve opening due to elevated RVEDP

Normally, with inspiration, intrathoracic pressure falls (normally 3–5 mm Hg) and the pressure in other intrathoracic structures (PVs, pulmonary capillaries) decreases to a similar degree. This inspiratory pressure change is not fully transmitted to the intrapericardial and intracardiac cavities.

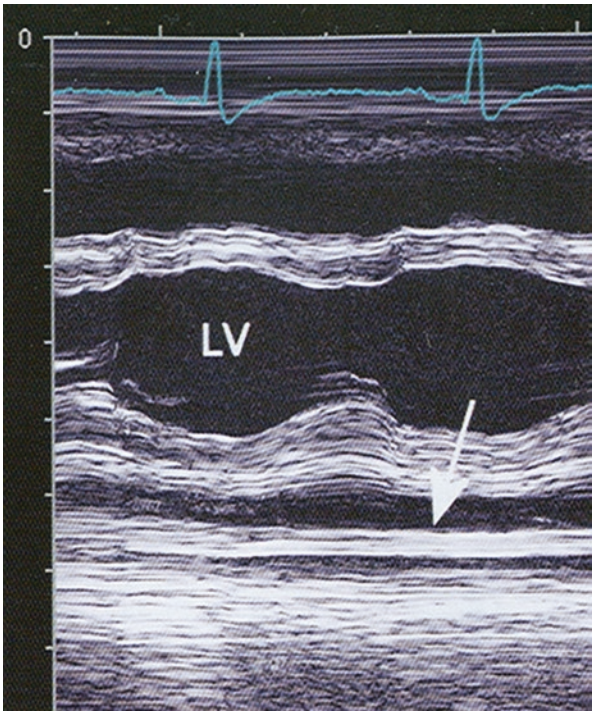

Fig. 3.2 Thickened pericardium in M-Mode Echocardiography (white arrow)

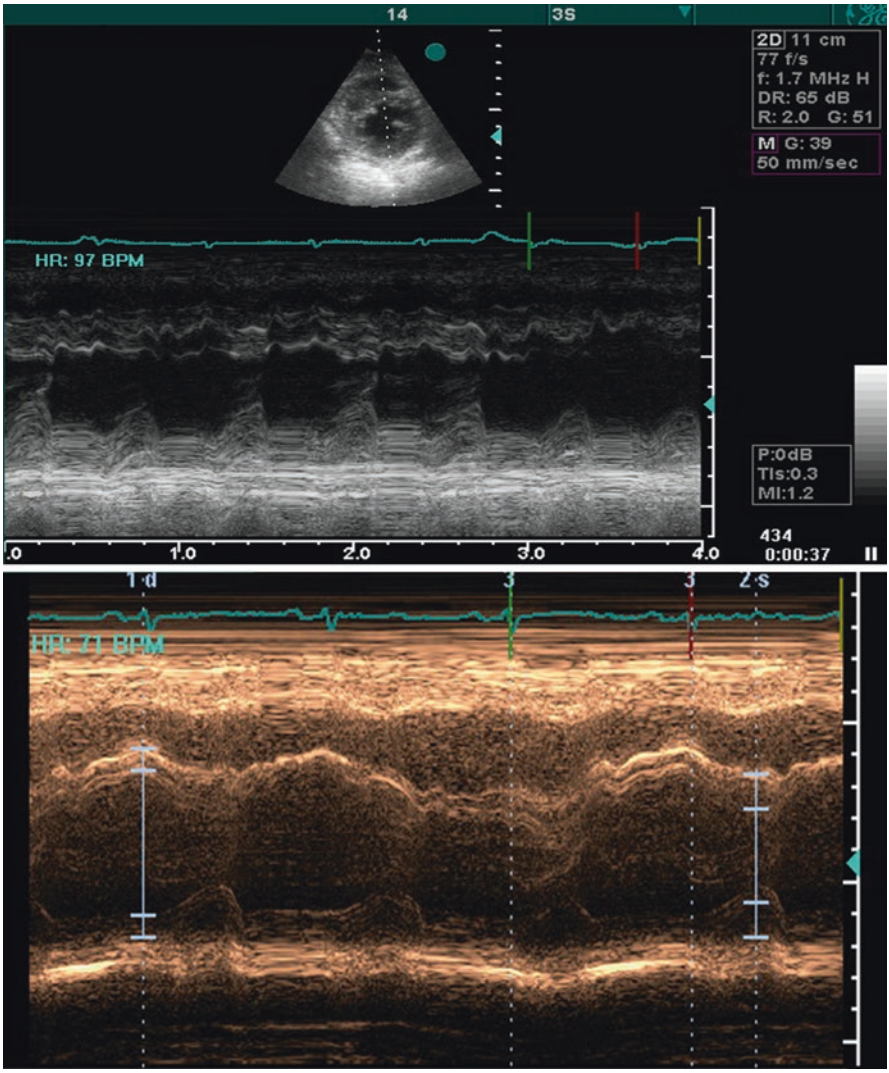

Fig. 3.3 Upper: Abrupt displacement of the IVS during early diastole (Septal bounce). Lower: Septal motion reflects a wavy pattern of diastolic septal motion

Consequently, the driving pressure gradient for LV filling decreases immediately after inspiration and increases with expiration.

In CP, a thickened or inflamed pericardium prevents full transmission of the intrathoracic pressure changes that occur with respiration to the pericardial and intracardiac cavities.

Diastolic filling (or distensibility) of the LV and RV relies on each other because the overall cardiac volume is relatively fixed within the thickened or noncompliant (adherent) pericardium, so there is exaggerated respiratory variation in heart chambers (Fig. 3.4).

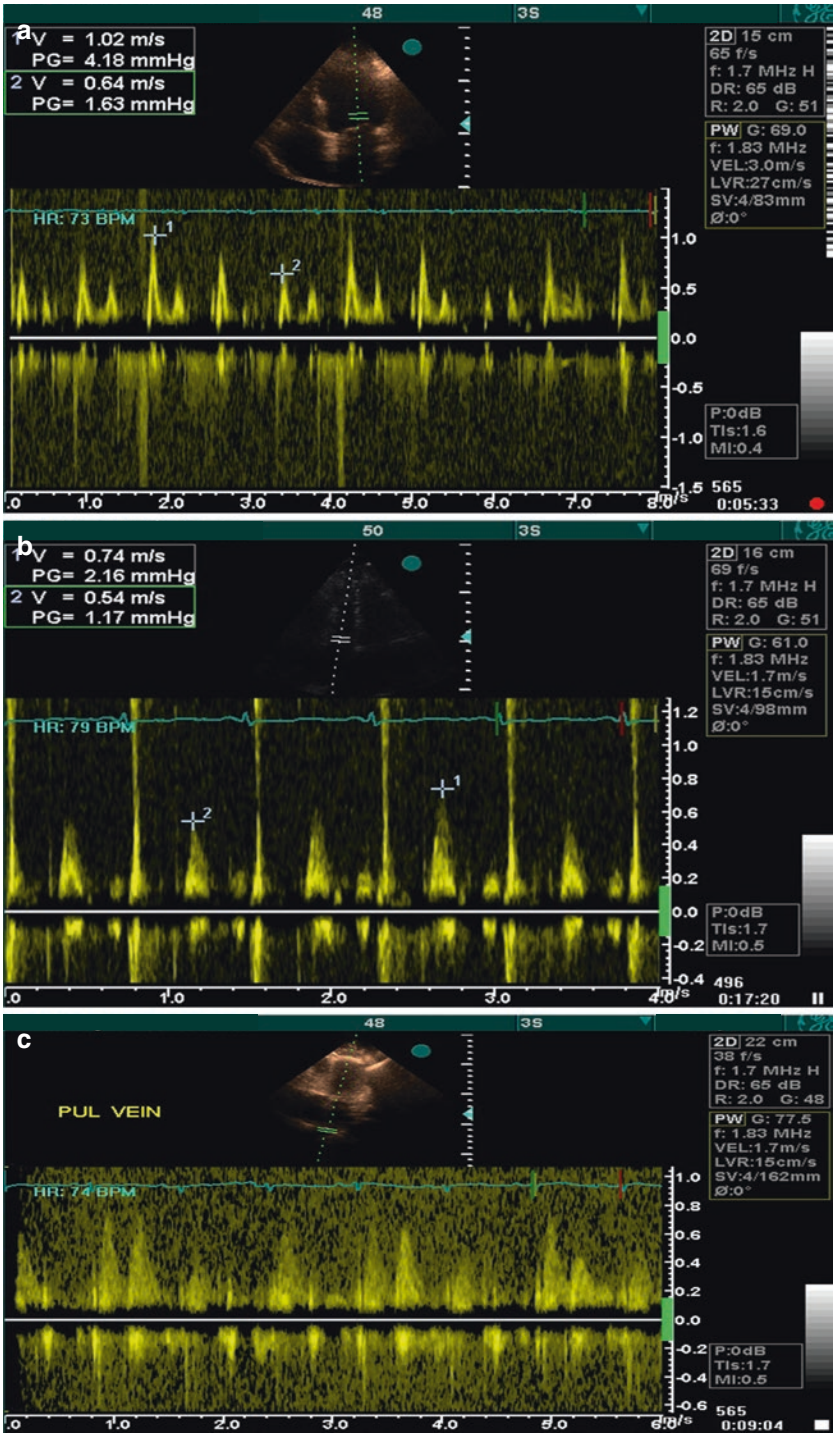

Fig. 3.4 Respiratory variation in (**a**) MV, (**b**) TV and in (**c**) pulmonary veins

Another important echocardiographic finding HV flow reversal after the onset of expiration, with decreased forward flow during diastole (D) (Fig. 3.5).

TDI is at least as sensitive as conventional echocardiography Doppler study. In myocardial disease, the mitral septal annulus velocity that reflects myocardial relaxation is decreased <7 cm/s because myocardial relaxation is abnormal; however, in constrictive pericarditis, the mitral annulus velocity, especially the septal annulus velocity, is relatively normal or even increased (Fig. 3.6).

Regional variations in deformation and strain include reduced LV circumferential strain, torsion, and early diastolic untwisting with preserved longitudinal strain and deformation.

In contrast, in restriction, circumferential strain and untwisting are preserved but these parameters are reduced in the longitudinal direction.

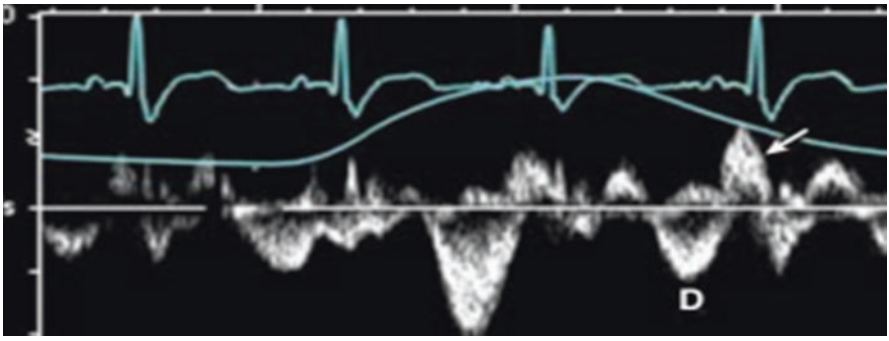

Fig. 3.5 Hepatic vein diastolic flow reversal after the onset of expiration

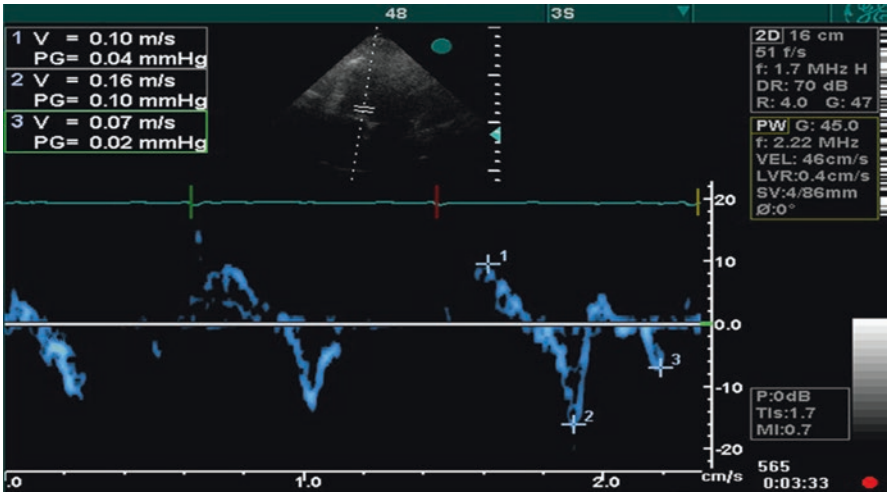

Fig. 3.6 Increase in MV septal annulus velocity in CP

When a comprehensive transthoracic echocardiographic study is diagnostic for constriction, no further diagnostic testing should be necessary. In equivocal cases, where noninvasive evaluation is inconclusive or discordant with clinical findings, hemodynamic assessment by cardiac catheterization should be performed [1–6].

Treatment

CP has a progressive but variable course. For most patients, surgical pericardiectomy is the definitive treatment.

Pericardiectomy for constriction has a relatively high perioperative mortality rate, ranging from 2% to nearly 20% in modern series.

Our case underwent near-complete pericardiectomy and has made a good recovery, and also echocardiography after surgery in the hospital and also 4 months later showed improvement in LV and RV function (Fig. 3.7) [4, 5].

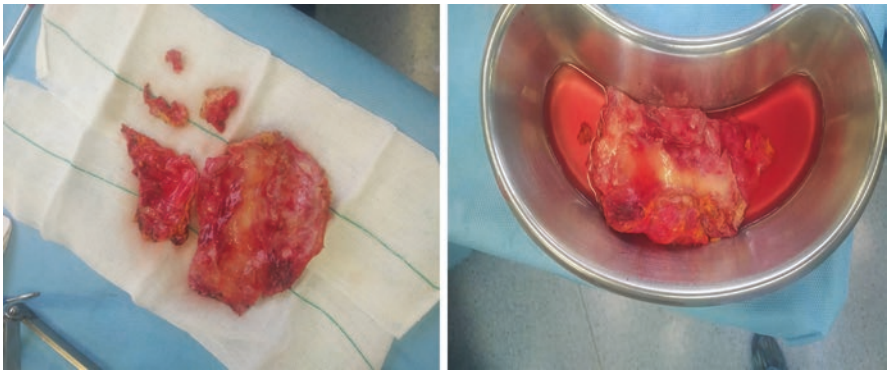

Fig. 3.7 Thickened pericardium after surgery

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