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Original Article

Systematic review of non-invasive cardiovascular imaging in the diagnosis of constrictive pericarditis

Sivakumar Ardhanari [a,1], Bharath Yarlagadda [a,1], Vishal Parikh [b], Kevin C. Dellsperger [c], Anand Chockalingam [a], Sudarshan Balla [a], Senthil Kumar [a,*]

[a] Department of Medicine, Division of Cardiology, University of Missouri School of Medicine, Columbia, MO, United States
[b] Department of Cardiology, University of South Florida, Tampa, FL, United States
[c] Department of Medicine, Augusta University Health, Augusta, GA, United States

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ABSTRACT

Background: Diagnosis of constrictive pericarditis (CP) can be challenging. It can be nearly impossible to distinguish CP from other causes of right heart failure. Although various imaging modalities help in the diagnosis, no test is definitive. Several reviews have addressed the role of various imaging techniques in the diagnosis of CP but a systematic review has not yet been published.
Objective: Our intention was to study the ability of various non-invasive imaging modalities to diagnose CP in patients with surgically confirmed disease and to apply our findings to develop a clinically useful diagnostic algorithm.
Methods: A PubMed (NLM) search was performed with MeSH term "constrictive pericarditis". Original articles that investigated the ability of various cardiovascular imaging modalities to noninvasively diagnose surgically confirmed CP were included in our review. Investigations that included any cases without surgical confirmation were excluded.
Results: The PubMed search yielded 3001 results with MeSH term "constrictive pericarditis" (January 8, 2016). We identified (40) studies on CP that matched our inclusion criteria. We summarized our results sorted by individual non-invasive CV imaging modalities – echocardiography, cardiac computed tomography (CT), and magnetic resonance imaging (MRI). Under each imaging modality, we grouped our discussion based on different parameters useful in CP diagnosis.
Conclusions: In conclusion, contemporary diagnosis of CP is based on clinical features and echocardiography. Cardiac MRI is recommended in patients where echocardiography is not diagnostic. Both cardiac MRI and CT can guide surgical planning but we prefer MRI as it provides both structural and functional information.

1. Introduction

Constrictive pericarditis (CP) is characterized by focal or global scarring and loss of elasticity of the pericardium with or without associated thickening. The abnormal pericardium impedes diastolic filling causing elevated systemic venous pressures. This causes right heart failure that classically manifests as lower extremity edema, ascites, and poor effort tolerance.[1] However, the clinical features are not unique making the diagnosis challenging. Restrictive cardiomyopathy (RCM) is a close clinical mimic as it also causes impaired ventricular filling resulting in similar clinical presentation. It is imperative to resolve this diagnostic dilemma because patients with CP can be effectively cured with pericardiectomy. The evaluation of CP includes detailed clinical history and examination, echocardiogram, cardiac catheterization, cardiac computerized tomography (CT), and magnetic resonance imaging (MRI).[2] Several recent reviews have addressed this topic.[1–8] However, a systematic review has not yet been published.

2. Methods

PubMed (NLM) search was performed with MeSH term "constrictive pericarditis". Original investigations that involved imaging diagnosis of CP were included in our review. The diagnosis

* Corresponding author at: University of Missouri School of Medicine, One Hospital Drive, CE306, Columbia, MO 65212, United States. Tel.: +1 573 882 2296; fax: +1 573 884 7743.
E-mail address: kumarse@health.missouri.edu (S. Kumar).
[1] These authors contributed equally to this work.

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of CP had to be confirmed based on surgical findings and pathology in all patients. Case reports, studies performed exclusively in children (age < 18 years), and publications in languages other than English were excluded. We excluded studies on effusive-constrictive pericarditis and constrictive epicarditis.

3. Results

The PubMed search yielded 3001 results with MeSH term "constrictive pericarditis" (January 8, 2016). We identified 40 original investigations published between 1978 and 2015 that studied a total of 1244 patients (76% males and age range 19 months to 87 years). An etiology was reported for 1073 patients; of these, CP was idiopathic in 297 patients (28%). When a cause was identified, the etiology of CP included surgery (232 patients, 22%), tuberculosis (231 patients, 21%), radiation (87 patients, 8%), viral (40 patients, 4%), and miscellaneous causes (186 patients, 17%) that included infection, inflammation, trauma, malignancy, collagen vascular disease, and myocardial infarction.

Surgical and pathological findings (pericardial thickening, fibrosis, adhesions, calcification, bulging of the heart out of the pericardial incision at pericardiectomy) were reported only in a few studies – 6 echocardiography studies,[9–14] 3 MRI studies,[15–17] and 4 CT studies.[9,11,18,19] We summarized our results sorted by individual non-invasive CV imaging modalities – echocardiography, cardiac computed tomography (CT), and magnetic resonance imaging (MRI) (Tables 1–3). Under each imaging modality, we grouped our discussion based on various structural and functional alterations induced by CP – pericardial thickness, motion of pericardium and myocardium, constrictive physiology, septal bounce, chamber geometry, and vascular dilatation.

3.1. Echocardiography

3.1.1. Pericardial thickness

Transthoracic echocardiography has limited accuracy to assess pericardial thickness[20] and was present in only 37% of CP patients[9]; transesophageal echocardiography is superior but is rarely performed for this indication alone.[10,11]

3.1.2. Motion of the pericardium and the myocardium

By echocardiography, pericardial adhesion may be evident as thickened, parallel, adherent pericardial layers that are pulled together during systole.[21] Pericardial tethering and restricted posterior wall motion are commonly reported in patients with CP.[12,22,23]

Tissue Doppler (TD) echocardiography measures low velocity Doppler signals from myocardial motion during early diastole and systole denoted as e' and S' respectively. Conventionally, the myocardial velocities are measured from samples placed at the mitral annulus at the septal or medial and lateral walls.

3.1.2.1. Early diastolic myocardial velocity (e′). In normal subjects, early diastolic myocardial velocities (e') sampled at the lateral wall tend to be higher than the velocities measured at the septal wall. Mitral "annulus reversus" is the reversal of the normal relationship of higher lateral to lower medial e' velocities reported in 74% of patients with CP (Figs. A and B).[53] This is related to the tethering of the lateral wall by pericardium unlike the septal wall. Mitral "annulus reversus" is unique to CP and is not present in RCM.

RCM being a myocardial disease tends to have lower TD velocities in contrast to CP and normal subjects. Butz et al. reported septal e' velocity of 13 cm/s in CP vs. 4 cm/s in RCM and lateral e' velocity of 11 cm/s in CP vs. 5 cm/s in RCM.[24] Ha et al. reported that a cutoff for $e' > 8$ cm/s can be used to differentiate CP from RCM (Fig. A).[25] Sengupta et al reported a lower cutoff of 5 cm/s for mean e' of the 4 LV walls to differentiate CP from RCM without overlap.[26] Mitral e' has also been shown useful, even in the absence of expected respiratory variation in early rapid filling (E) velocity.[27] Interestingly, an inverse correlation exists between E/e' and left ventricular (LV) filling pressures in patients with CP (annulus paradoxus) compared to direct correlation in primary myocardial disease such as RCM[28]; this was also confirmed by the same group of researchers in a subsequent study. However, a recent report could not reproduce the finding of annulus paradoxus in 49 patients with surgically confirmed CP.[29]

3.1.2.2. Systolic mitral annular velocity (S′). Butz et al. reported systolic mitral annular velocity (S') velocity of 7 cm/s in CP vs. 4 cm/s in RCM. A combination of average septal and lateral wall systolic (S') velocity of <8 cm/s and e' velocity of <8 cm/s had a 93% sensitivity and 88% specificity in excluding CP.[24]

Several other echocardiography techniques show promise for CP diagnosis. Myocardial velocity gradient quantifies spatial distribution of intramural velocities across the myocardium and another technique to express the differences in myocardial motion between CP and RCM. Myocardial velocity gradient was lower in RCM during ventricular ejection and rapid ventricular filling compared to CP and normal controls. Myocardial velocity gradient was positive in RCM and negative in CP and normal controls during isovolumic relaxation.[23]

Lu et al. showed that in normal subjects, the motion of the myocardium was greater than that of the pericardium, but the motion of the outer and inner-layers of the myocardium were almost exactly the same. However, in patients with CP, the outer-layer myocardium had far reduced motion similar to the pericardium, while the motion of the inner-layer myocardium was stronger than that of the outer-layer myocardium. This study was able to quantify this difference with the equation ([D3 − D2]/[D2 − D1]) through 2D echocardiography and quantitative tissue Doppler imaging (where D1 is the systolic peak displacement of pericardium; D2 displacement of outer myocardium; D3 displacement of inner myocardium).[30]

Strain imaging by echocardiography was reported to be useful in differentiating CP from RCM. CP is characterized by reduced circumferential strain, torsion and untwisting velocity but normal longitudinal strain. In RCM, there is reduced longitudinal strain but normal circumferential strain. This is due to the fact that subendocardial fibers (predominantly responsible for longitudinal shortening) are more affected in RCM and subepicardial fibers (predominantly responsible for circumferential shortening) in CP.[31]

3.1.3. Ventricular interdependence

Abnormal early diastolic filling is a prominent feature of both CP and RCM; the findings that favor CP include rapid early LV filling, shorter duration of rapid filling period, and reduced peak LV filling rate.[14,20] An earlier study reported increased E–F slope on M mode echocardiography (mitral valve early diastolic closing velocity) in patients with CP.[12] Doppler echocardiography can be used to differentiate CP from RCM based on respiratory changes in transvalvular flow velocities (Figs. C and D). Hatle et al. reported significant changes in left ventricular isovolumic relaxation time (IVRT) and in early mitral and tricuspid flow velocities at onset of inspiration and expiration in CP, but not in RCM or normal subjects. Respiratory variation in early mitral flow (E) velocity was >25% in CP (Fig. C) vs. <15% in RCM; the respiratory variation in these parameters normalized after pericardiectomy in patients with CP.[32–34]

"Occult CP" is an entity in patients with strong suspicion for CP without diagnostic features of constriction by imaging attributed to variations in loading conditions. Altering preload can help bring out constrictive physiology in these patients. Preload reduction can

Table 1
Original investigations for diagnosis of CP in surgically confirmed patients – Echocardiography. COPD = chronic obstructive pulmonary disease; CP = constrictive pericarditis; E = early mitral inflow velocity; e' = early diastolic mitral annular velocity; LV = left ventricle; NA = not available; NPV = negative predictive value; PPV = positive predictive value; RCM = restrictive cardiomyopathy; S' = systolic mitral annular velocity; SD = standard deviation; TB = tuberculosis. Data for pericardial thickness, e' and S' were rounded to nearest whole number.

Echocardiography								
Year; Author	n	Mean age ± SD (range)	Male/ female	Comparison groups with n	Etiology of constrictive pericarditis with n	Parameter/cutoff	Sensitivity	Specificity
2014; Welch[39]	130	62 ± 12	107/23	RCM or severe tricuspid regurgitation 36	Idiopathic/collagen vascular disease/prior pericarditis 77; Surgery 39; Radiation 14	1. Respiration related ventricular septal shift (PPV 92, NPV 74)	93	69
						2. Medial mitral $e' \geq 9$ (PPV 92, NPV 57)	83	81
						3. Hepatic vein expiratory diastolic reversal ratio ≥0.79 (PPV 96, NPV 49)	76	88
						1 + (2 or 3)	87	91
						All 3	64	97
						Annulus paradoxus, i.e. Lower E/e' ratios at the medial mitral annulus in patients with CP (5.8; CI 3.6 to 9.3) vs. patients without CP (16.1; CI 11.6–21.2, $p < 0.001$)		
2011; Veress[53]	99	58 ± 15	72/27	None	Idiopathic 33; Surgery 34; Radiation 13; Other 19	Annulus reversus (medial $e' >$ lateral e', reverse of normal) present in 74% of patients with CP		
2010; Butz[24]	34	58 ± 12	18/16	RCM 26	Surgery 13; Radiation 3; Unknown 18	RCM vs. CP: S' 4 vs. 7 cm/s; septal e' 4 vs. 13 cm/s; lateral e' 5 vs. 11 cm/s		
						$S' < 8$ cm/s and $e' < 8$ cm/s for RCM	93	88
2009; Lu[30]	20	33	11/9	Normal 20	TB 10; Surgery 2; Unknown 8	Quantitative tissue Doppler (R = D3 − D2/D2 − D1. D1 systolic peak displacement of pericardium; D2 outer myocardium; D3 inner myocardium); $R > 1.2$	90	85
						$R = 5 \pm 4.7$ in CP; 0.6 ± 0.7 in normal; $p < 0.05$		
2008; Sengupta[31]	26	56	16/10	RCM 19; Normal 21	Surgery 5; Radiation 8; Viral 5; Idiopathic 8	Significantly reduced circumferential strain, torsion and early diastolic untwisting velocities (E_r) in CP; significantly reduced longitudinal displacement (E_m) in RCM		
						Torsion <10°	83	84
						$E_r > -50°$/s	57	95
						$E_m > 5$ cm/s	92	90
2008; Sengupta[26]	16	62	13/3	RCM 15	Surgery 7; Radiation 2; Idiopathic 7	e' averaged from all 4 walls (>6.6 cm/s)	93	93
						e' averaged from all 4 walls >5 cm/s correctly distinguished CP from RCM		
2005; Sengupta[13]	40	24 ± 12	24/16	Normal 35; Abnormal septal motion due to other causes 20	TB 26; Pyogenic 2; Radiation 2; Unknown 10	Higher septal e' velocity (>7 cm/s) and early diastolic biphasic motion of interventricular septum in CP	83	93
2004; Ha[25]	23	59 (27–87)	21/2	Amyloid 38; Primary RCM 14	Surgery 8; Unknown 15	$e' \geq 8$ cm/s in CP	95	96
2004; Sengupta[41]	45	24 ± 12	24/21	Normal 35; RCM 11; Right heart failure 20; Chronic pericardial effusion 11	TB 26; Pyogenic 2; Radiation 2; Idiopathic 15	$e' > 8$ cm/s in 40/45 with CP, 8/20 with right heart failure, all with Chronic pericardial effusion. $e' < 8$ cm/s in 8/11 with RCM		
						Using combined e', E, M mode and 2D echo	89	95
2003; Talreja[9]	143	(12–82)	108/35	None	Surgery 40; Radiation 21; MI 12; Trauma 2; Collagen vascular disease 11; Viral 17; Other infection 7; Renal failure 1; Other 13; Idiopathic 39 (some had more than one etiology)	Echocardiography diagnostic of CP (PPV 53)		
						Abnormal septal motion (PPV 49)		
						Atrial enlargement (PPV 61)		
						Thickened pericardium (PPV 37)		
2002; Ha[27]	19	57 ± 13	17/2	None	Surgery 6; Unknown 13	Normal mitral annular velocity (mean 12 ± 4) even in patients without respiratory variation in mitral inflow velocity (9 of 19 patients)	100	
2002; Izumi[10]	7	57 ± 5	6/1	None	Pericarditis 3; TB 1; Surgery 2; Idiopathic 1	Thickened pericardium over right atrium in 6/7 patients but none over LV in esophageal views but in 7/7 patients over LV in transgastric view		

Table 1 (*Continued*)

Echocardiography								
Year; Author	*n*	Mean age ± SD (range)	Male/ female	Comparison groups with *n*	Etiology of constrictive pericarditis with *n*	Parameter/cutoff	Sensitivity	Specificity
2001; Ha[28]	10	64 (54–72)	8/2	None	Surgery 4; Idiopathic 6	Inverse correlation between E/e' and LV filling pressures in patients with CP; Mean e' was 11 ± 4 cm/s (range, 7–21 cm/s). Pulmonary capillary wedge pressure and LV end diastolic pressure were 25 ± 6 and 27 ± 6 mmHg		
2000; Palka[23]	10	57 ± 14	7/3	RCM 15; Normal 30	Idiopathic 4; Surgery 4; Radiation 1; Malignancy 1	Doppler myocardial velocity gradient measured from left ventricular posterior wall was lower in RCM during ventricular ejection (RCM 2.8 ± 1.2 vs. CP 4.4 ± 1.0 vs. Normal controls 4.7 ± 0.8 s^{-1}; $p < 0.01$) and during rapid ventricular filling (RCM 1.9 ± 0.8 vs. CP 8.7 ± 1.7 vs. normal controls 3.7 ± 1.4 s^{-1}). Doppler myocardial velocity gradient was positive in RCM and negative in CP and normal controls during isovolumic relaxation (+0.7 ± 0.4 vs. −1.0 ± 0.6 vs. −0.4 ± 0.3 s^{-1}; $p < 0.01$)		
1998; Boonyaratevej[33]	20	58 ± 12	19/1	COPD 20	Idiopathic 9; Viral 5; Surgery 4; Trauma 1; Rheumatoid arthritis 1	Respiratory variation in mitral E velocity was 41% (compared to COPD 46%) Respiratory variation in SVC systolic flow velocity was 4 ± 3 cm/s (compared to COPD 40 ± 19 cm/s); $p < 0.0001$		
1997; Ling[11]	11	53 ± 15	11/0	Normal 21	Irradiation 2; Idiopathic 4; Post-CABG 4; Myelodysplastic syndrome 1	Pericardial thickness ≥3 mm (PPV 88, NPV 94)	95	86
1997; Oh[35]	12	60 (47–73)	10/2	None	NA	Respiratory variation in mitral E velocity after decreasing preload in patients with constriction who do not exhibit the typical respiratory change; The mean percent respiratory change in E velocity was 5 ± 7% at baseline and 32 ± 28% with preload reduction		
1996; Klodas[38]	5	68 (61–76)	5/0	Heart failure due to other causes 12	Surgery 1; Idiopathic 4	Tricuspid regurgitation peak velocity, duration and VTI increased with inspiration in CP but decreased in controls		
1994; Mantri[40]	33	27 ± 17 (2.5–62)	21/12	RCM 8; Normal 33	NA	Left atrial dilatation in CP and RCM		
1994; Oh[35]	28	55 ± 15	21/7	CP 25; RCM 1; Normal 2	Idiopathic 8; Surgery 6; Radiation 3; TB 1; Rheumatoid arthritis 1; Unknown 6	E velocity ≥25% increase with expiration. Hepatic vein flow – augmented diastolic flow reversals after onset of expiration ≥25% of forward diastolic velocity)	88	
1989; D'Cruz[22]	7	61 ± 3	7/0	Normal 23; HCM 13	NA	Angle formed by junction of LV and left atrial posterior walls in parasternal long axis view by 2D echocardiography <150° in 5/7 with CP vs. none in normal subjects and HCM		
1989; Hatle[32]	7	52 ± 11	NA	RCM 12; Normal 12	Unknown 3; Surgery 2; Radiation 2	Respiratory variation in left ventricular isovolumic relaxation time Early mitral flow (E) velocity >25% in CP vs. <15% in RCM		
1983; Janos[14]	4	(9–67)	NA	3 RCM; 39 Normal	TB 2; Surgery 2	Very rapid early filling in CP vs. prolonged mid diastolic filling in RCM		
1978; Schnittger[12]	37	NA	NA	None	NA	Abnormal septal and posterior wall motion; high E–F slope		

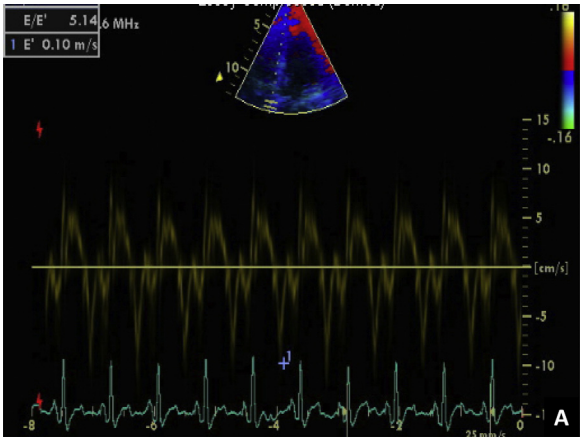


Fig. A. Tissue Doppler echocardiography showing 10 cm/s medial e' velocity.

demonstrate ventricular interdependence in those who do not have the typical respiratory change in mitral E velocity at baseline (presumed to be due to volume overload). The mean percent respiratory change in E velocity was $5 \pm 7\%$ at baseline and $32 \pm 28\%$ with preload reduction.[35] Conversely, in volume depleted patients, hemodynamic measurements may have to be repeated after a fluid load to establish the diagnosis of CP.[36] Patients with CP on mechanical ventilation showed reversal of the expected physiologic variations in mitral inflow and pulmonary vein flow parameters attributed to the changes in the intrathoracic pressures.[37]

Unlike patients with other causes of heart failure, those with CP show increased peak velocity and duration of tricuspid regurgitation during inspiration.[38] Respiratory variation in superior vena cava (SVC) flow was useful in differentiating CP vs. chronic obstructive pulmonary disease (COPD) (4 ± 3 cm/s in CP vs. 40 ± 19 cm/s in COPD).[33] Augmented late systolic as well as diastolic flow reversals after onset of expiration in hepatic vein flow have been shown to have a high specificity for CP (Fig. E) compared to RCM.[34,39] It has also been shown that in patients with CP, SVC systolic flow is decreased, absent, or reversed, but in diastole, forward flow is increased with increased late backflow.[20]

Septal bounce is a commonly used term to describe the abnormal beat to beat diastolic septal motion in patients with CP. Visually, it is appreciated as a shudder or oscillatory motion (leading to the term septal bounce). It is likely another manifestation of ventricular

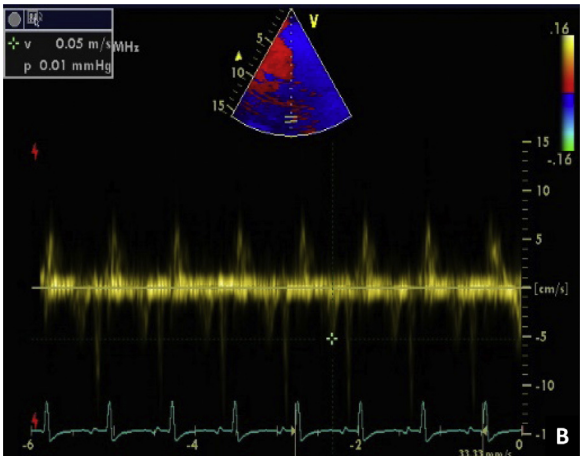


Fig. B. Tissue Doppler echocardiography showing lateral e' velocity of 5 cm/s (same patient as Fig. A). There is reversal of the normal relationship of higher lateral to lower medial e' velocities in this patient with surgically proven CP (annulus reversus).

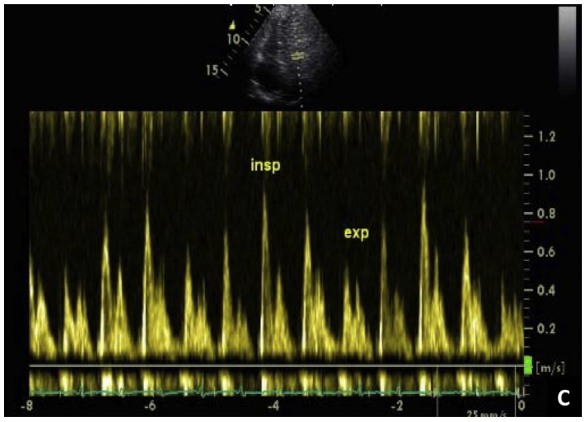


Fig. C. Pulse wave Doppler echocardiography showing respiratory variation in early mitral flow (E) velocity of >25% in CP confirmed by surgery.

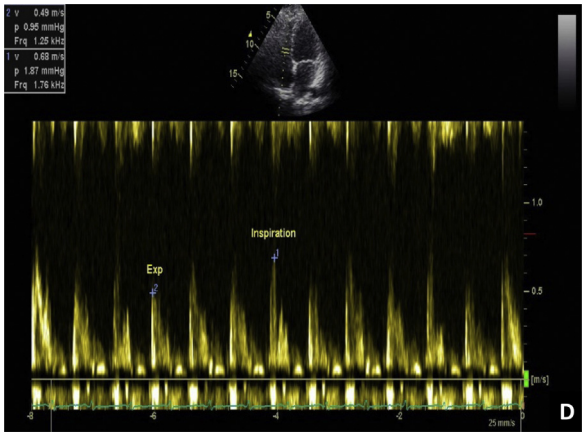


Fig. D. Pulse wave Doppler echocardiography showing respiratory variation in early tricuspid flow (E) velocity.

interdependence when the observation of septal motion is not limited to inspiration and also impacted by events of the cardiac cycle.[9,12,13,39] The presence of septal bounce had a sensitivity of 62% and specificity of 93% for diagnosis of CP.[21]

3.1.4. Chamber geometry and vascular dilatation

Atrial enlargement was reported in 61% patients with CP.[8,9,40] Dilated IVC and hepatic veins with blunted respiratory variation are commonly seen in patients with right heart failure including CP.[8]

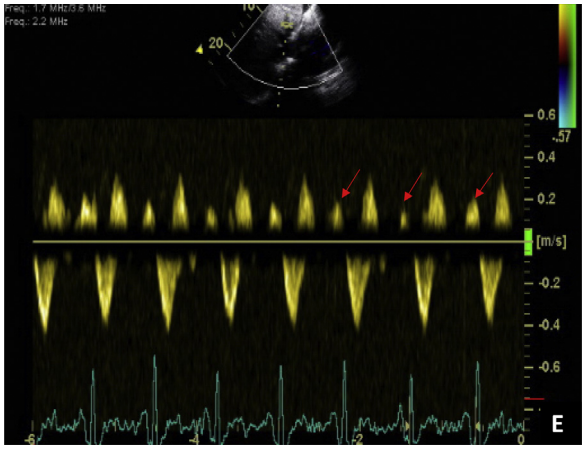


Fig. E. Pulse wave Doppler echocardiography showing arrows pointing toward expiratory diastolic flow reversals in the hepatic veins.

3.1.5. Combination of findings

In a study of 34 patients, Butz et al. reported that a combination of average septal and lateral wall systolic (S') velocity of <8 cm/s and e' velocity of <8 cm/s had a 93% sensitivity and 88% specificity in ruling out CP.[24] Combination of Doppler (E, e'), M-mode, and 2D echocardiographic parameters had 89% sensitivity and 95% specificity for CP diagnosis.[41]

3.2. Computerized tomography

3.2.1. Pericardial thickness

CT provides excellent visualization of the pericardium (Fig. F). Suchet et al. demonstrated increased pericardial thickness of ≥3 mm in all patients with CP.[18] In one study, 72% of patients with CP had thickened pericardium by CT; in addition, calcified pericardium was found in 25%.[9] Using cine CT, pericardial thickness was 10 ± 2 mm in CP, 2 ± 1 mm in RCM, and 1 ± 1 mm in normal controls ($p < 0.05$ for CP vs. no CP).[19] Overall, CT is recognized as an excellent tool to determine pericardial thickness and the most sensitive technique to identify pericardial calcification.[42]

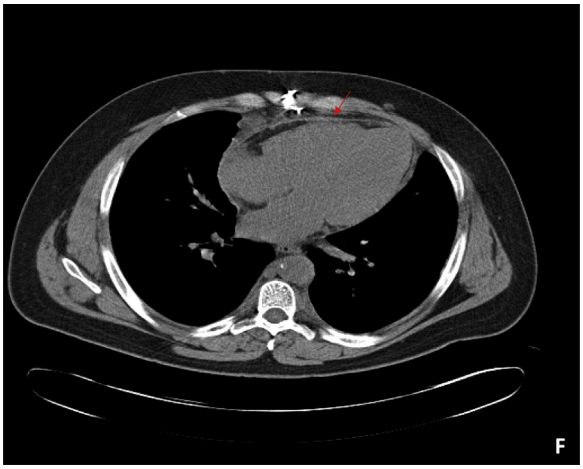


Fig. F. CT showing thickened pericardium (arrow) in surgically confirmed CP.

3.2.2. Ventricular interdependence

In an earlier study with cine CT, the rapidity of diastolic filling (assessed by calculating the percent filling fraction in early diastole) was increased for both LV and RV in patients with CP.[19] Kloeters et al. used electron beam CT demonstrating an abnormal rapid diastolic left and right ventricular filling and thickened pericardium in patients with CP compared to patients with either dilated cardiomyopathy or normal subjects.[43] The findings from the above studies need to be replicated using multi-slice CT scanners as electron beam CT is no longer used in clinical practice.

3.2.3. Chamber geometry and vascular dilatation

CT is not very sensitive in detection of abnormal ventricular morphology and interventricular septal deviation, which were found in 31% and 15% of patients with CP respectively; however, IVC dilation is almost universal in CP and was reported in 97% of patients.[9,18]

3.3. Magnetic resonance imaging

3.3.1. Pericardial thickness

In a study by Cheng et al., the maximal pericardial thickness in CP (Figs. G and H) was significantly greater than controls and RCM patients (4–12 mm in CP vs. 1–3 mm in controls and RCM; $p < 0.001$).[15] In another study, pericardial thickness >4 mm was present in 17 out of 22 patients with CP compared to none of the 20 normal controls.[44] A threshold of pericardial thickness >3–4 mm yielded a sensitivity and specificity of 83–91% and 100% to diagnose CP.[17,44]

Table 2
Original investigations for diagnosis of CP in surgically confirmed patients – Computerized tomography. CP = constrictive pericarditis; CT = computerized tomography; LV = left ventricle; NA= not available; NPV = negative predictive value; PPV = positive predictive value; RCM = restrictive cardiomyopathy; SD = standard deviation. Data for pericardial thickness were rounded to nearest whole number. *Age and sex reported for 238 patients that includes 26 patients excluded from study.

Year; Author	*n*	Age ± SD (range)	Male/female	Comparison groups with *n*	Etiology of constrictive pericarditis with *n*	Parameter/cutoff
2008; Kloeters[43]	5	51	5/0	Dilated cardiomyopathy with CorCap 10; Normal 10	Infection 2; Collagen vascular disease 2; Unknown 1	Significantly accelerated LV and right ventricular filling; Significantly increased pericardial thickness 5 ± 1 vs. 1 mm by electron beam CT
2003; Talreja[9]	143	(12–82)	108/35	None	Surgery 40; Radiation 21; Myocardial infarction 12; Trauma 2; Collagen vascular disease 11; Viral 17; Other infection 7; Uremia 1; Other 13; Idiopathic 39 (some had more than one etiology)	CT diagnostic of CP (PPV 68) Thickened pericardium (PPV 72) Abnormal ventricular morphology (PPV 31) Calcified pericardium (PPV 25)
1997; Ling[11]	11	53 ± 15	11/0	Normal 21	Radiation 2; Idiopathic 4; Surgery 4; Myelodysplastic syndrome 1	Pericardial thickness measured by electron beam CT correlated well with transesophageal echocardiography and pathology measurements
1993; Oren[19]	5	62 ± 7	NA	Cardiomyopathy with normal pericardium 7; Normal 7	Radiation 1; Surgery 1; Idiopathic 3	Using cine CT, pericardial thickness 10 ± 2 mm in CP vs. 2 ± 1 mm in cardiomyopathy with normal pericardium vs. 1 ± 1 mm in normal ($p < 0.05$ for CP vs. no CP) Left ventricular filling fraction was 83 ± 6% in CP vs. 62 ± 9% in cardiomyopathy vs. 44 ±v5% in normal Right ventricular filling fraction 93 ± 5% in CP vs. 62 ±v14% in cardiomyopathy vs. 35 ± 6% in normal ($p < 0.05$ CP vs. no CP
1992; Suchet[18]	186	(19 months–78 years)*	174/64*	None	TB 157; Radiation 2; Malignancy 2; Sarcoidosis 1; Surgery 2; Post pericardiectomy 1, idiopathic 21	Pericardial thickness ≥3 mm in all patients with CP; Inferior venacava dilation 97%; abnormal ventricular morphology 31%; deviation of interventricular septum 15%

Table 3
Original investigations for diagnosis of CP in surgically confirmed patients – Magnetic resonance imaging. CP = constrictive pericarditis; LV = left ventricle; NA = not available; NPV = negative predictive value; PPV = positive predictive value; RCM = restrictive cardiomyopathy; RV = right ventricle; SD = standard deviation; TB = tuberculosis. Data for pericardial thickness were rounded to nearest whole number. * Age and sex information includes 7 patients without CP or RCM.

Magnetic resonance imaging								
Year; Author	*n*	Age ± SD (range)	Male/ female	Comparison groups with *n*	Etiology of constrictive pericarditis with *n*	Parameter/cutoff	Sensitivity	Specificity
2015; Power[48]	16	NA	NA	2	NA	Absence of slippage between visceral and parietal pericardium on radiofrequency tissue tagging was diagnostic of CP. PPV, NPV 100%	100	100
2015; Bolen[17]	42	55 ± 16	39/3	21 patients without CP	Idiopathic 22; Surgery 10; Viral 3; Radiation 2; Others 5	Pericardial thickness 3.1 ± 2.5 mm	83	100
						Relative interventricular septal excursion 11.4 ± 8.7%	93	95
						Both parameters combined	100	90
						SVC and IVC size >2.6 cm	55	95
						Diastolic septal bounce	90	85
						Ventricular interdependence	88	100
						LV area change 17.7 ± 24.1%	86	100
						RV area change 26.4 ± 9%	57	86
2015; Angheloiu[52]	11	62 ± 14	7/4	11 normal volunteers	NA	Compression of RV in 4 chamber view (1 − RV surface area/Cardiac surface area) (0.88 ± 0.03 in CP vs. 0.85 ± 0.03, $p = 0.02$)	82	82
						Angle between tricuspid valve annulus plane and interventricular septum (81 ± 9 in CP vs. 91 ± 7, $p = 0.01$)	73	91
						Impact angle between tricuspid inflow vector and septum (8.6 ± 8.7 in CP vs. 0 ± 6.6, $p = 0.01$)	73	91
						Proportion of tricuspid inflow impacting septum (0.38 ± 0.19 in CP vs. 0.01 ± 0.03, $p < 0.0001$)	100	100
2013; Anavekar[51]	17	62 ± 16	NA	35 patients without CP	NA	Biventricular end diastolic area in inspiration/expiration = 1 in CP vs. 1.28 in those without CP		
2013; Kusunose[49]	52	59 ± 14	46/6	RCM 35; Normal 26	Radiation 2; TB 1; Surgery 10; Idiopathic 39	LV lateral wall strain/LV septal wall strain 0.8 in CP vs. 1.1 in RCM and 1 in Normal. Cutoff <0.96	86	96
						RV free wall strain/LV septal wall strain 0.8 in CP vs. 1.4 in RCM and 1.2 in Normal. Cutoff <0.97	76	85
2011; Cheng[15]	23	43 (15–77)	18/5	RCM 22; Normal 25	Unknown 10; Surgery 4; TB 7; Inflammatory/ infection 2	Relative atrial volume ratio >1.32 (left/right atrial volume)	83	86
						Diastolic septal bounce	96	100
						Pericardial thickness CP 4–12 mm; normal and RCM 1–3 mm; $p < 0.001$		
2012; Young[45]	52	59 ± 13	43/9	Chronic recurrent pericarditis 16; Other pericardial pathology 8	Surgery 13, Radiation 6; Idiopathic 18; Viral 10; Autoimmune 3; Trauma 2; Others 2 (includes 2 with overlapping chronic recurrent pericarditis and CP)	Mean IVC diameter 3.1 ± 0.4 cm		
						Pericardial thickness 9.2 ± 7.0 mm with calcification; 4.6 ± 2.1 mm without calcification in CP		
						Abnormal septal motion 86% in CP		
						Pericardial enhancement in CP 76% vs. Chronic recurrent pericarditis 94%		
2010; Bauner[44]	22	52 ± 12 (41–70)	18/4	Normal 20	Surgery 11; Radiation 3; Inflammatory 2; Unknown 6	Abnormal septal motion 21/22 in CP vs. 0/20 in Normal	96	100
						RV volume reduced in CP ≤ 133 ml	77	90
						Tricuspid early filling/atrial component reduced in CP ≤ 1.3	77	95
						Pericardial thickness ≥4 mm 17/22 in CP vs. 0/20 in Normal	91	100
						All 4 parameters	83	90
2006; Francone[16]	18	63	9/9	Normal 17; Inflammatory pericarditis 6; RCM 15	NA	Ventricular coupling (max. septal excursion with respiration 11.8%); Significantly increased max. pericardial thickness 8 ± 6 mm vs. 2 ± 1 mm normal vs. RCM 3 ± 2 mm vs. Inflammatory pericarditis 12 ± 4 mm		
2005; Francone[50]	6	47 ± 10	3/3	Normal 6; RCM 4; Chronic pulmonary embolism/Cor pulmonale 5; Pericardial effusion 6	NA	In all CP patients, onset of inspiration lead to a leftward inversion/flattening of the septum during early ventricular filling		

Table 3 (*Continued*)

Magnetic resonance imaging								
Year; Author	*n*	Age ± SD (range)	Male/ female	Comparison groups with *n*	Etiology of constrictive pericarditis with *n*	Parameter/cutoff	Sensitivity	Specificity
2003; Giorgi[46]	21	63 (21–79)	24/17*	RCM 13; Normal 12	NA	Abnormal diastolic septal motion (PPV 100, NPV 83) Pericardial thickening in 21/21 CP patients (mean thickness 7 mm) vs. 1/13 RCM patients	81	100

Pericardium tends to be thicker in patients with calcification as was reported in a recent study. Pericardial thickness was 9.2 ± 7.0 mm with calcification and 4.6 ± 2.1 mm without calcification.[45] Giorgi et al. found that abnormal focal or diffuse pericardial thickening was noted in 21 out of 21 patients with CP with a mean thickness of 7.1 mm compared to only 1 out of 13 patients with RCM.[46] In a study by Lachhab et al, the average thickness of pericardium was 8 mm in patients with CP and the thickening was circumferential in 64% and localized in 36%; more importantly, the assessment of pericardial thickness using MRI showed 100% concordance with surgical findings.[47]

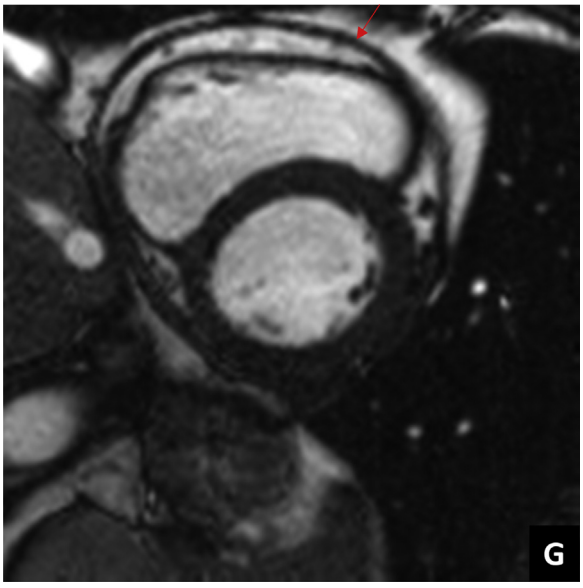


Fig. G. Cardiac MRI cine showing thickened pericardium (arrow). The pericardium is thickened (5 mm in maximum thickness) circumferentially that was correlated to surgical findings.

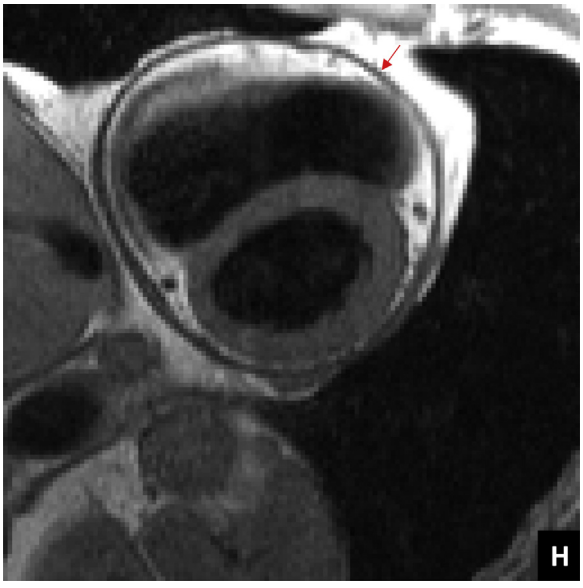


Fig. H. Cardiac MRI dark blood images showing thickened pericardium (arrow).

3.3.2. Motion of pericardium and myocardium

Pericardial adhesions can be visualized directly by cine MRI and myocardial tagging. Application of MRI tag lines in a grid-like pattern over a certain imaged slice allows for the study of the deformation of the grid over time. Absence of slippage between visceral and parietal pericardium on radiofrequency tissue tagging was diagnostic of CP with sensitivity and specificity of 100%.[48]

Kusunose et al. demonstrated abnormal myocardial mechanics in patients with CP by assessment of myocardial strain using MRI. They reported a depressed LV lateral wall and RV free wall strain with preserved LV septal wall strain in patients with CP. A ratio of LV lateral wall strain to septal wall strain of <0.96 had a sensitivity and specificity of 86% and 96% respectively for diagnosis of CP; similarly, a ratio of RV free wall strain to septal wall strain <0.97 had a sensitivity and specificity of 76% and 85% respectively.[49] These findings are consistent with prior echocardiography literature on the similar parameters.

3.3.3. Ventricular interdependence

Presence of ventricular interdependence (septal shift toward left during inspiration) using real-time cine MRI in the short-axis plane (Fig. I and Cine 2) had 81–88% sensitivity, 100% specificity, 90% accuracy, 100% positive predictive value (PPV), and 83% negative predictive value in the diagnosis of CP.[16,17] Also, a septal shift cutoff of 11.8% of the biventricular diameter was able to completely differentiate CP from RCM and normal subjects.[16] In a recent study, similar cutoff of 11.4 ± 8.7% had a sensitivity and specificity of 93% and 95% respectively.[17] This finding was best seen in the base of the ventricle and in the first heartbeat after inspiration. An earlier study also compared the utility of this technique in

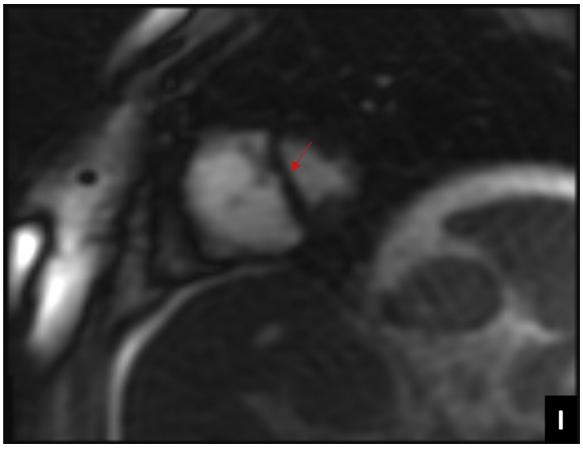


Fig. I. Cardiac MRI showing leftward shift of the interventricular septum (arrow) during inspiration, which is consistent with ventricular interdependence in a patient with ascites and leg edema that resolved after pericardiectomy.

Table 4
Distinguishing features between constrictive pericarditis and restrictive cardiomyopathy seen on imaging.

	Constrictive pericarditis	Restrictive cardiomyopathy
Pericardial thickening	Almost universal	Absent
Annulus reversus	Present	Absent
e', S', respiratory variation in E velocity	Higher	Lower
Ventricular interdependence and septal bounce	Present	Absent
Hepatic vein diastolic flow reversal in expiration	Present	Absent
Left atrial to right atrial volume ratio	Higher	Lower

differentiating CP from other entities with septal shift: (1) cor pulmonale – septal shift was present but respiration did not change the septal position; (2) pericardial effusion – septal shift was also present in 1 of 6 patients but pericardial effusion can be readily diagnosed; and (3) normal volunteers – septal shift was found in two of six normal volunteers but minimal compared to that in CP patients.[50] Ventricular interdependence was demonstrated by Anavekar et al. using the ratio of biventricular end diastolic area in inspiration to expiration; this ratio was 1 in CP compared to 1.28 in those without CP ($p < 0.0001$).[51] Similar to echocardiography, MRI can also demonstrate increased early ventricular filling and decreased or absent late filling using velocity-encoded phase contrast MRI or plotting ventricular volumes against time when visualized on a four-chamber or short-axis cine image field.[46]

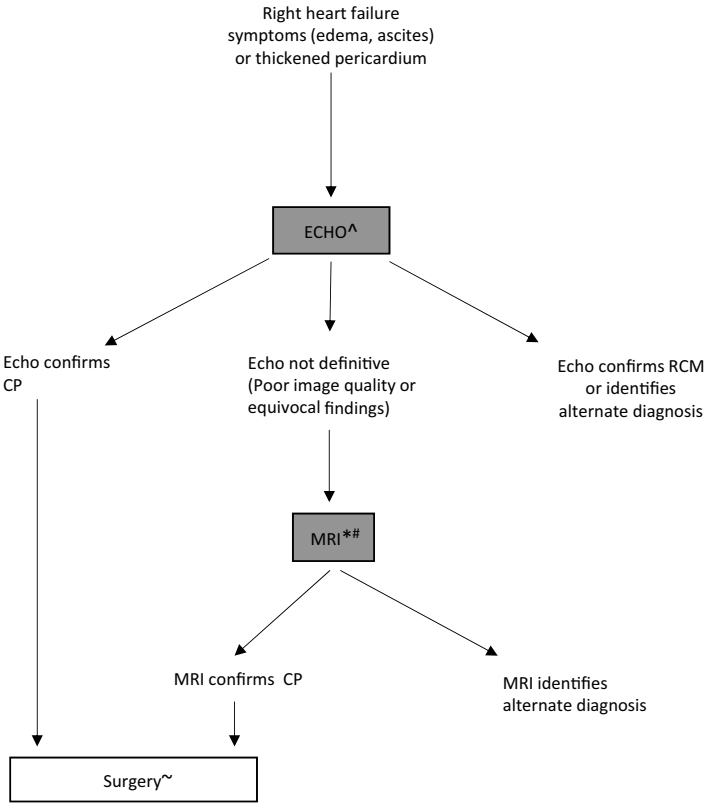
The presence of septal bounce has been reported almost universally by MRI in patients with CP with a sensitivity and specificity of 90–96% and 85–100% respectively.[15,17,44,45] By detailed analysis of interaction between cardiac blood flow and septal motion, 4 newer parameters for CP diagnosis were reported. Patients with CP compared to controls had significantly greater compression of RV, lesser angle between the tricuspid valve annulus plane and the interventricular septum, greater impact angle between the tricuspid inflow vector and septum and higher proportion of tricuspid inflow impacting the septum.[52]

3.3.4. Chamber geometry and vascular dilatation

Patients with CP had reduced RV volume; compared to normal controls, a RV volume of <133 ml had a sensitivity and specificity of 77% and 90% respectively for diagnosis of CP.[44] LV area change $17.7 \pm 24.1\%$ had a sensitivity and specificity of 86% and 100% respectively; RV area change $26.4 \pm 9\%$ had a sensitivity and specificity of 57% and 86% respectively.[17] Cheng et al. recently demonstrated that CP could be differentiated from RCM by precise quantification of biatrial enlargement. The relative atrial volume ratio (left atrium volume/right atrium volume) was significantly greater in CP patients versus those with RCM. This can be explained by the fact that the posterior wall of the left atrium is actually anatomically separated from the pericardial space and so it expands greater than the right atrium in patients with CP, whereas in patients with RCM, both atria expand an equal amount.[15] IVC dilatation is a common finding in CP patients with one study reporting IVC diameter of 3.1 ± 0.4 cm.[45] In a recent study, SVC and IVC size >2.6 mm had a sensitivity and specificity of 55% and 95% respectively.[17]

4. Discussion

CP is usually suspected either due to symptoms of right heart failure or pericardial thickening noted during chest imaging. The



^(Mayo criteria; Criteria for overall diagnosis)
1.Respiration related ventricular septal shift
2.Medial mitral e' > 9 cm/s (Figure 1, panel D)
3.Hepatic vein expiratory diastolic reversal ratio > 0.79 (Figure 1, panel C)
1 + (2 or 3) - Sensitivity 87, Specificity 91
All 3 - Sensitivity 64, Specificity 97
or
Cutoff of 5 cm/s for mean e' of the 4 LV walls correctly distinguished CP from RCM without overlap.

***Pericardial thickness ≥ 3 mm (Figure 1, panel G,H) + Relative interventricular septal excursion ≥ 12% (Figure 1, panel I)**
Sensitivity 100, Specificity 90
or
a) Abnormal septal motion + RV volume ≤ 133 ml + Tricuspid E:A ≤ 1.3 + Pericardial thickness ≥ 4 mm Sensitivity 83, Specificity 90
or
b) LV lateral wall strain/LV septal wall strain <0.96 Sensitivity 86, Specificity 96
or
c) Relative atrial volume ratio > 1.32 (LA volume/RA volume)
Sensitivity 83, Specificity 86

a, b and c are imaging parameters which require volume assessments/strain imaging and are not routinely used.

~cardiac catheterization for hemodynamics / CT or MRI to assess degree and extent of pericardial thickness and calcification if needed by surgeon;

#CT if MRI contraindicated.

Fig. J. Diagnostic algorithm for CP.

Table 5
Summary and comparison of findings by various imaging modalities in the assessment of constrictive pericarditis.

Findings	Echocardiography	CT	MRI
Pericardial thickness and calcification	TTE has limited accuracy, TEE superior	• Best modality to assess for pericardial calcification • Useful for the assessment of entire pericardium and surgical planning	Useful for the assessment of entire pericardium and surgical planning
Motion of the pericardium and the myocardium	• Higher e' and S' help differentiate CP from RCM • Annulus reversus – unique to CP	Limited ability to assess physiology	• Myocardial tagging technique - high diagnostic accuracy
Ventricular interdependence and septal bounce	• Higher respiratory variation in E velocity seen in CP vs. RCM • Augmented diastolic hepatic vein flow reversal highly specific for CP • Septal bounce present in CP but not RCM	Limited ability to assess pathophysiology	• Septal shift easier to demonstrate with MRI than Echo • Septal bounce reported almost universally • Velocity encoded phase contrast MRI to detect respiratory variation in E velocity – inferior in temporary resolution to Doppler echocardiography
Chamber geometry and venous dilation	Dilated atria, IVC and hepatic veins seen both in CP and RCM	Similar to Echo	• Chamber volume quantification superior to Echo • Left atrial to right atrial volume ratio higher in CP vs. RCM

available evidence suggests CT and MRI as the best methods to accurately measure pericardial thickness. Normal pericardial thickness is usually 1–2 mm based on gross pathology data. Pericardial thickness >3–4 mm by either CT or MRI will usually warrant further assessment for CP. The diagnosis of CP is strengthened greatly if the pericardium is diffusely rather than focally thickened. While pericardial thickness is a very useful parameter in diagnosis of CP, constriction with normal-thickness pericardium has been well recognized.[9] In one study, 18% of patients had constrictive physiology with a normal-thickness noncompliant pericardium. Since these patients will also benefit from pericardiectomy, lack of pericardial thickening should not be used to exclude CP.[9]

Based on our systematic review, we generated an algorithm incorporating echocardiography, cardiac MRI, and CT that can be useful for diagnosis of CP (Fig. J). Echocardiography is an essential first step for patients presenting with findings of CP such as peripheral edema and ascites. The combination of respiration related interventricular septal shift and either medial mitral e' velocity >9 cm/s or hepatic vein expiratory diastolic reversal ratio >0.79 had a sensitivity of 87% and specificity of 91% for diagnosis of CP. Using all 3 findings as diagnostic criteria increased the specificity to 97% but lowered sensitivity to 64%. Alternatively, a cutoff of 5 cm/s for mean e' of the 4 LV walls correctly distinguished CP from RCM without overlap.[26] Due to sensitivities >90%, absence of respiratory ventricular septal shift or reduced mitral annular e' (<9 cm/s) can be used to exclude CP.[25,39] Echocardiography is also very useful in identifying differential diagnoses such as restrictive cardiomyopathy, dilated cardiomyopathy, valve disease, or significant pulmonary hypertension. Table 4 summarizes the distinguishing features of constrictive pericarditis from restrictive cardiomyopathy.

If echocardiography is not definitive (poor image quality or equivocal findings), cardiac MRI would be the next logical step. Cardiac MRI provides structural and functional data and is preferred over cardiac CT. Pericardial thickness ≥3 mm and respiratory septal excursion ≥12% in combination have a sensitivity and specificity of 100% and 90% respectively.[17] Therefore, the absence of both these findings will definitively rule out CP. Novel parameters with high sensitivities and specificities have been described (items a, b, and c in Fig. J), which may need further validation. In future, with widespread adoption of volume criteria and strain imaging, we anticipate their routine use in clinical practice. Even if echocardiography is confirmatory for CP, cardiac MRI or CT can still be useful for surgical planning. Table 5 summarizes and compares the ability of the different imaging modalities in identifying the various diagnostic findings of constrictive pericarditis.

4.1. Limitations

Since CP is a relatively rare diagnosis, most of the available literature is based on small single center studies. Although the diagnostic utility of numerous techniques such as strain imaging (echocardiography) and atrial volumes (MRI) have been elegantly demonstrated, these are yet to be adopted in many imaging laboratories. Most studies stated that surgical and pathological findings were used to confirm CP diagnosis but the exact criteria were reported only in a few studies.

5. Conclusion

In most patients, contemporary diagnosis of CP is based on clinical features and echocardiography. Cardiac MRI is recommended in patients where echocardiography is not diagnostic. Both cardiac MRI and CT can guide surgical planning but we prefer MRI due to its ability to provide both structural and functional information.

Conflicts of interest

The authors have none to declare.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ihj.2016.06.004.

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