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Research Article

Assessment of Myocardial Mechanics in Patients Undergoing Pericardiectomy for Chronic Constrictive Pericarditis by Tissue Doppler Imaging and 2D Speckled Tracking Echocardiography: A Prospective Observational (Cohort) Study

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Abstract

Background: This study was designed to prospectively evaluate the changes in tissue Doppler imaging (TDI) at mitral and tricuspid annuli and two dimensional speckle tracking echocardiography in patients undergoing pericardiectomy for chronic constrictive pericarditis and identify the relationship if any of the tissue Doppler imaging and speckle echocardiographic derived variables with patient's symptomatic status following surgery.

Patients and Methods: Twelve patients undergoing pericardiectomy for constrictive pericarditis aged 7 years to 70 years (median 21; IQR: 19.75-26.5 years) were studied for 2-36 months (median 19 months). They underwent Doppler flow velocity, TDI, and 2D-speckle echocardiographic studies. Friedman' test was used to test the changes in TDI-derived mitral and tricuspid annular velocities and speckle derived parameters in postoperative period from baseline.

Results: Despite congestive heart failure, all patients had normal left ventricular ejection fraction and increased medial mitral and tricuspid early diastolic septal velocity (e′) with "annulus reversus". This pattern of annular velocity improved maximally in the immediate postoperative period. At closing interval, 2 (16.7%) patients continued to be in New York Heart Association class II and both of them continued to remain in atrial fibrillation. There was statistical significant improvement in the Global cirumferential strain than in global longitudinal and global radial strain after pericardiectomy.

Conclusions: We conclude that tissue Doppler imaging and speckle tracking echocardiography are useful investigative modalities for serial evaluation of patients undergoing pericardiectomy. It can be performed serially with a high degree of reproducibility.

Keywords: tissue doppler imaging; two-dimensional speckle echocardiography; chronic constrictive pericarditis; Pericardiectomy; echocardiography

Introduction

Pericardiectomy is usually the only accepted curative treatment for constrictive pericarditis and several studies including ourselves have shown its efficacy in improving symptoms with normalization of hemodynamics in the majority of cases. [1-7]

However, the outcome after pericardiectomy is variable for multifactorial reasons. [6,7] This could be because constrictive pericarditis is a

heterogeneous disease and some patients have concomitant myocarditis or myocardial fibrosis. Another reason could be due to incomplete pericardiectomy due to its severity and calcification. [6.31]

Non-invasive assessment of regional myocardial function by magnetic resonance imaging and computed tomography imaging are useful diagnostic alternatives. Echocardiography remains advantageous for widespread clinical use because of its portability, low risk, and comparatively high temporal resolution. [14,22,32]

Doppler myocardial imaging is an echocardiographic technique that has the potential to enhance diagnostic information available from Doppler blood-flow indices. [7-11] Specifically, tissue Doppler imaging (TDI) has allowed the determination of discrete amplitude cut- off points at the lateral mitral annulus to distinguish constrictive pericarditis from restrictive cardiomyopathy without overlap.[33-39,40-47]

Because the mechanoelastic properties of the myocardium are preserved in constrictive pericarditis, the longitudinal mitral annular velocities are normal. Tissue Doppler imaging can measure mitral or tricuspid annular motion which reflects ventricular systolic and diastolic motion in the long axis.[10,33-39] In constrictive pericarditis, early diastolic septal velocity (medial e) is preserved or even increased, [10,33-39] due to limitation of lateral expansion by the constricting pericardium, and early diastolic lateral mitral annular velocity (mitral lateral e) tends to be lower than medial e′ which is a reversal of their normal relationship. [10,33-39] This mitral annular velocity pattern is relatively specific for constrictive pericarditis in patients with heart failure since e′ velocity is usually reduced in patients with myocardial disease whether left ventricular ejection fraction (LVEF) is preserved or reduced.[48-60]

In our previous investigation, we had prospectively evaluated the changes in tissue Doppler imaging (TDI) at mitral and tricuspid annuli in patients undergoing pericardiectomy for chronic constrictive pericarditis (CCP) and identified the relationship of all the TDI-derived variables with the patients symptomatic status following pericardiectomy.[10] Our previous study evaluated the relationship of TDI-derived mitral and tricuspid annular velocities with the postoperative functional status and concluded that TDI is a useful investigative modality for diagnosis of constrictive pericarditis and are non-predictors of postoperative outcome following pericardiectomy.[10]

Heart performs complex rotational and translational movement inside the chest, thus distorting the measurements of myocardial velocities. In our previous study, we only recorded tissue Doppler imaging of longitudinal axis motion in the 4-chamber view. [10] Due to the local tethering effect, analysis of multiple annular regions could have provided additional helpful data.[40-47]

Myocardial regional mechanics assessed by echocardiographic approaches have been described by 4 principal types of strain or deformation: longitudinal, radial, circumferential, and rotational. Although myocardial fibre orientation results in these strain vectors occurring three dimensionally in an integrated manner, most investigative works have been done using individual strain assessments. The term strain applied to echocardiography in a simplistic sense is to describe lengthening, shortening, or thickening, also known as regional deformation.[40-47]

Speckle-tracking-derived deformation analysis can provide not only strain (and strain rate) but displacement and rotation of the myocardium.[40-47] In addition to the short-axis rotation, more recently, speckle-tracing echocardiography could assess longitudinal septal-to-lateral rotation displacement (SLRD), which can quantify the rocking or swinging motion of the whole heart. [40-47]

Although several studies have evaluated left ventricular mechanics of patients with constrictive pericarditis quantitatively, there are limited data on the assessment of change before and after pericardiectomy. There are no studies either on the comparison of myocardial mechanics following pericardiectomy performed via median sternotomy or modified anterolateral thoracotomy. There are no data either on the degree and timing of reduction of the speckle-tracking derived myocardial mechanics and their relationship following surgery. After total and radical pericardiectomy, the heart loses its support from the pericardium which limits undue cardiac displacement and starts to swing vigorously. [40-47]

Since the degree of restriction in myocardial motion in CCP is never uniform, and there may be underlying myocardial fibrosis, restricting the analysis only to the annular myocardial segment will miss the complete picture of the disease. Therefore, the inclusion of multilevel myocardial segment will provide a global picture rather than regional.

In the immediate postoperative period, the myocardial function is impaired by numerous factors like myocardial oedema, use of inotropic agents and arrhythmia. Therefore, it's prudent to study the parameters at multiple stages i.e. preoperative (baseline), immediate postoperative, day 4 postoperative, at discharge, at 3 and 6 months.

This prospective non-randomised study aims to: i) serially evaluate the immediate and late effects of total and radical pericardiectomy on the clinical outcome and left ventricular size and function, ii) serially assess the effect of total and radical pericardiectomy on the speckle tracking derived myocardial mechanics, namely, longitudinal displacement (LD), longitudinal strain (LS) and septal-to-lateral rotational displacement (SLRD), iii) analyze the relationship of the speckle tracking derived parameters with the patients symptomatic status in the pre- and postoperative period, and iv) compare the speckle tracking derived parameters after total and radical pericardiectomy via median sternotomy and modified left lateral thoracotomy and objectively assess the adequacy of pericardiectomy.

Patients and Methods

Criterions of decision-making and selection of patients

This study included diagnosed patients with CCP with raised central venous pressure (CVP)/ right atrial pressure (RAP) more than 12mmHg, with or without hepatic dysfunction, renal dysfunction, pleural effusion and massive ascites. This also included patients with constrictive pericarditis with hemodynamic decompensation requiring inotropes, and ventilator support in the preoperative period, and patients with focal/patchy calcific pericarditis. Echocardiographically, pericardial thickness of $\geq$ 3 mm was considered significant.

Patients with i) annular constrictive pericarditis, ii) calcific pericardial patch compressing predominantly the right atrium and right ventricular outflow tract, iii) circumferential “cocoon” calcification encompassing all cardiac chambers, iv) calcific spurs penetrating the ventricular chambers, v) recurrent pericarditis following previous partial pericardiectomy, vi) constrictive pericarditis following mediastinal irradiation, vii) extracardiac intrapericardial mass, vii) previous open heart surgery and those with a gradient between superior and inferior venacava and right atrium $\geq$2mmHg were preferably considered for median sternotomy approach. Median sternotomy was preferred in this subset of patients for improved surgical exposure and easy institution of cardiopulmonary bypass if required, for inadvertent cardiac injury and bleeding.

A modified left anterolateral thoracotomy was the preferred approach in the remaining patients of CCP. In general, it is our institutional protocol to select the patients for a left anterolateral thoracotomy in cases of purulent pericarditis and CCP. Thoracotomy was the preferred option in these patients because of the presence of concomitant pyothorax and the concerns of sternal infection. We could achieve total pericardiectomy in

these patients because of loculations and poorly formed adhesions which could be easily peeled off. [3,4,8,10,11]

Exclusion criteria: Patients with concomitant congenital or acquired heart disease were excluded.

Study design: Prospective observational (cohort) study

This study conforms to the principles outlined in the declaration of Helsinki and was approved by the Institutional Ethics Committee. Patients were enrolled in the study protocol after obtaining informed written consent from patients/parents/guardians.

Between January 2018 and August 2020, 12 consecutive patients (9 males) undergoing total pericardiectomy via median sternotomy (n=7), and modified left anterolateral thoracotomy (n=5) without cardiopulmonary bypass for CCP operated by a single surgeon were included in this study. Patients' age at operation ranged from 7 to 70 years (median: 21; IQR:19.75-26.5 years).

Preoperatively, 9 (75%) patients were males. Preoperatively 9 (75%) and 2 (16.7%) patients were in New York Heart Association Class III and IV respectively with congestive heart failure (CHF) as the presenting symptom.

Six (50%) patients had ascites and atrial fibrillation was found in 5 (41.7%) patients. Two (16.7%) patients exhibited evidence of grade II mitral and tricuspid regurgitation. Demographic details are summarized in table 1.

Eight out of 12 (66.7%) patients had history of pulmonary tuberculosis and all patients received multidrug therapy (isoniazid, rifampicin, ethambutol, pyrazinamide) for 3 months followed by triple drug therapy for 9 months after operation. Preoperatively, all patients were administered digitalis and diuretics.

The etiology was considered tubercular if the histopathology of the excised pericardium showed granulomas, caseation, giant cells (n=8, 66.7%), or if the debris removed at surgery was positive for acid fast bacilli (n=8, 66.7%). A history of pulmonary and lymph node tuberculosis was present in all (n=12, 100%) patients.

Chest roentgenogram revealed pleural effusion (n=7, 58.3%), and pulmonary infiltrates (n=9, 75%). A lateral chest roentgenogram and computed tomogram demonstrated islands of focal/oblique patchy calcification over the anterior and diaphragmatic surfaces of the heart in 5 (41.7%) patients.

None had mitral annular calcification. Five of 12 (41.7%) patients with atrial fibrillation were in New York Heart Association class IV (**Table 1**). The clinical profile, Doppler echocardiography, tissue Doppler imaging and computed-tomography conclusively established the diagnosis of CCP in all 12 (100%) patients (**Figures 1 and 2**). Five (41.7%) patients underwent cardiac magnetic resonance (CMR) to define both morphological and functional changes (**Figures 3A-3C**).

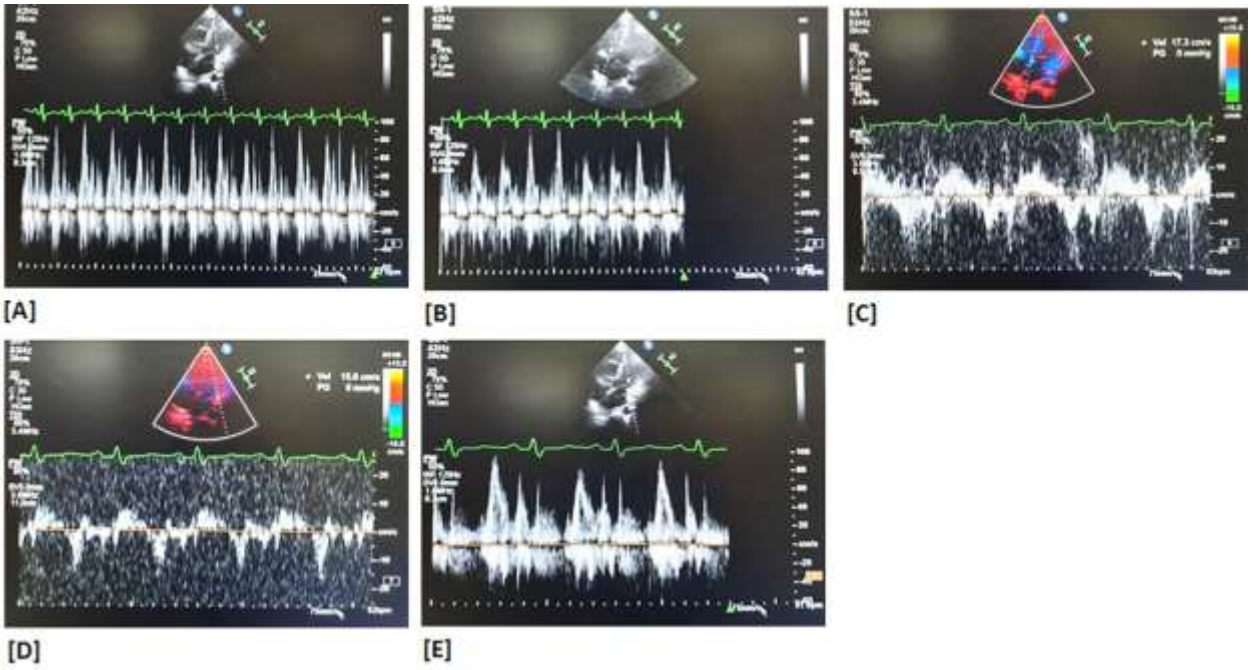


Figure 1: *Preoperative echo images in a patient with chronic constrictive pericarditis. [A] Pulse wave Doppler signals at the mitral valve showing increased respiratory variations. [B] Pulse wave Doppler signals at the tricuspid valve showing increased respiratory variations. [C & D] Doppler signals using Tissue Doppler Imaging (TDI) in apical 4-chamber view with sample volume placed at the medial and lateral annulus of mitral valve respectively showing annulus reversus. [E] Mitral valve inflow e/as >1.5.*

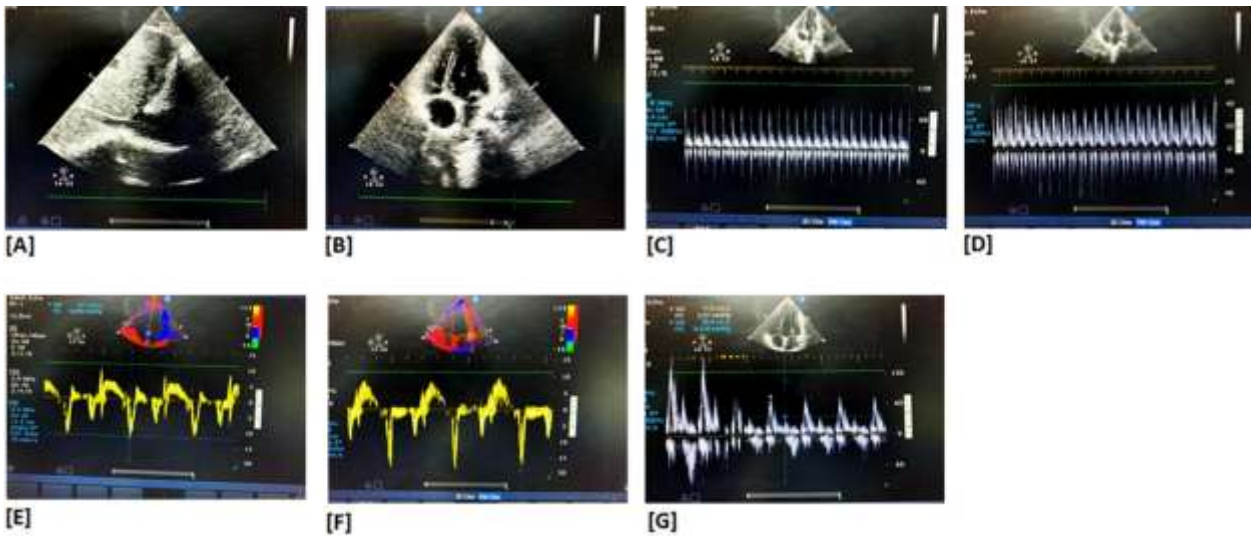
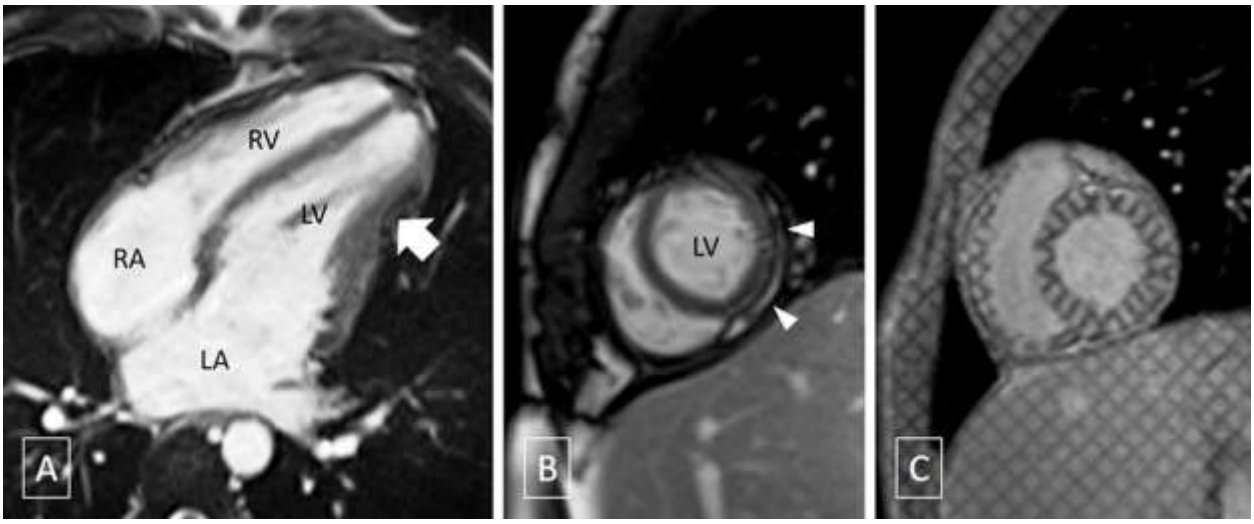


Figure 2: *Postoperative echo images of the same patient with chronic constrictive pericarditis showing [A] Normal sized IVC. [B] Apical 4-chamber view (2D image) normal chamber geometry. [C] Pulse wave Doppler signals at the mitral valve showing normal respiratory variations. [D] Pulse wave Doppler signals at the tricuspid valve showing normal respiratory variations. [E & F] Doppler signals using Tissue Doppler Imaging (TDI) in apical 4-chamber view with sample volume placed at the medial and lateral annulus of mitral valve respectively showing normalization of annulus reversus. [G] Mitral valve inflow e/a normal.*



Figures 3A-3C: *Four chamber MRI cine image (A) shows tubular ventricles with indentation (thick white arrow) along the LV free wall. Short axis cine image (B) shows thin pericardial collection with thickened pericardium (arrowheads) adherent along the inferolateral wall of LV. Short-axis image from tagged cine sequence (C) shows adherence and immobility of the pericardial–myocardial interface.*

Preoperative cardiac catheterization was performed on 3 (25%) patients to quantify right and left heart pressures, assess coronary anatomy, and obtain endomyocardial biopsy. The rest did not have catheterization because of their class III and IV symptoms with hepatic dysfunction, renal dysfunction or the echocardiographic findings were unequivocal. All demonstrated the findings considered diagnostic of constrictive pericarditis: an elevated right atrial pressure usually with a M-or-W shaped contour, and abnormally high right ventricular end-diastolic pressure with a characteristic dip-plateau diastolic configuration, and a ratio of right ventricular end-diastolic pressure to right ventricular systolic pressure of ≥ 0.30 (**Table 1**).

Profile		Number (%)	
Number of patients		12 (100%)	
Males		9 (75%)	
Age in years, Mean±SD, Median, IQR, Range		25.17±14.42, Median 21 IQR: 19.75-26.5, Range: 7-70 years	
Body weight (kg), Mean±SD, Median, IQR, Min-Max		49.88±11.76, Median: 53.25, IQR: 46.50-57.25, Min-Max: 18-63	
Body surface area (m^2), Mean±SD, Median, IQR, Min-Max		1.47±0.25, Median: 1.54, IQR: 1.41-1.63, Min-Max: 0.80-1.73	
Duration of illness (months), Mean±SD, Median, IQR		19.33±8.15, Median: 18, IQR: 13.50-21	
Preoperative NYHA functional class	II III IV	1 (8.3%) 9 (75%) 2 (16.7%)	
Dyspnoea on exertion		11 (91.7%)	
Paraoxsysmal nocturnal dyspnea		1 (8.3%)	
Orthopnea		3 (25%)	
Congestive heart failure		5 (41.7%)	
Distended jugular vein in sitting position		10 (83.3%)	
Peripheral oedema		11 (91.7%)	
History of pedal oedema, ascites		11 (91.7%)	
Pleural effusion		7 (58.3%)	
Hepatomegaly		8 (66.7%)	
Pericardial knock		8 (66.7%)	
Ascites		6 (50%)	
Pulsus paradoxus		2 (16.7%)	
Renal derangement (serum creatinine $\geq$ 2mg/dl)		5 (41.7%)	
Hyperbilirubinemia (Serum bilirubin $\geq$ 2 mg/dl)		2 (16.7%)	
Hypoproteinemia (Serum albumin) $\leq$ 3.5 gm/dl		7 (58.3%)	
Pericardial calcification on chest X-ray		5 (41.7%)	
Pulmonary infiltrates		9 (75%)	
Tuberculosis on culture or history, and/or histopathology		5 (41.7%)	
Atrial fibrillation	Preoperative Postoperative	5 (41.7%) 4 (33.3%)	
Hb gm/dl mean $\pm$ SD (range)		12±1.35, Median: 12, Range: 11-13	
ESR mm/hr, Mean $\pm$ SD, Median, Range		11.23±3.78, Median: 11, Range: 8.7-13.25	
Mitral regurgitation		2 (16.7%)	
Tricuspid regurgitation		2 (16.7%)	
Pericardial thickness >43mm		12 (100%)	
Surgical approach	Median sternotomy Left antero-lateral thoracotomy	7 (58.3%) 5 (41.7%)	
Total pericardiectomy		12 (100%)	
Operative mortality		0	
Late death		0	
ICU stay (days), Mean±SD, Median, IQR, Min-Max		6.75±3.57, Median: 5, IQR: 4.0-9.25, Min-Max: 3-14	
Duration of inotropic support (days), Mean±SD, Median, IQR, Min-Max		5.58±3.29, Median: 4.5, IQR: 3-7.25, Min-Max: 2-13	
At last follow-up	Asymptomatic Symptomatic	12 (100%) 0	
Postoperative NYHA functional class II		12 (100%)	
		Preoperative	**Immediate postoperative**
Right atrial pressure (mmHg), mean±SD (range) Asymptomatic patients (n=12)		14.17±2.82	6.25±1.48

ESR=Erythrocyte sedimentation rate; SD=Standard deviation

Table 1: *Demographic, operative and perioperative data of the study group*

Echocardiographic Studies and Measurements

All patients had comprehensive evaluation with M-mode, two-dimensional (2-D) and pulsed-wave Doppler echocardiography with a respirometer recording and tissue Doppler imaging (TDI) before and after pericardiectomy using a Phillips iE 33 with 2.0 to 5.0 MHz transducer. Left ventricular ejection fraction (LVEF) was calculated by 2-D echocardiography with a modification of the method of Quinones and colleagues. [18] Left atrial volume was measured by the modified biplane area-length method. [19] Right ventricular systolic function was visually assessed. By using pulsed wave Doppler echocardiography, the following variables were measured: trans-mitral and trans-tricuspid peak velocities of early (E) and late filling (A) and E wave deceleration time (DT). On TDI, peak annular velocities were measured from the apical four chamber

view at systole (s'), early (e') and late (a') diastole with a 2- 5 mm tissue Doppler sample volume placed at the septal corner and at the mitral and tricuspid lateral annuli. In patients with atrial fibrillation, five consecutive signals were measured and averaged. Inferior vena caval (IVC) diameter was assessed in subcostal sagittal view.

On Doppler, two flow velocity envelopes can be seen during diastole in persons with sinus rhythm: the E-wave, representing the early, passive filling of the ventricle, and the A-wave, that happens late in diastole, representing the active filling, the atrial contraction. For both mitral and tricuspid valve E and A wave measured. Mitral or tricuspid regurgitation was assessed semi-quantitatively as grade 1+ to 4+. A constrictive pattern was defined as 25% or greater increase in mitral E-velocity with expiration as compared with inspiration and an augmented (25% or more) diastolic flow reversal in the hepatic vein after the onset of expiration compared with inspiration. On tissue Doppler imaging, lateral mitral e′, represents early diastolic myocardial relaxation velocity below the baseline as the annulus ascends away from the apex with cursor at lateral annulus; medial mitral e′ and lateral tricuspid e′ are same velocities measured at mitral medial annulus and tricuspid lateral annulus respectively. The mitral lateral s′ velocity represents the systolic myocardial velocity at lateral mitral annulus. The medial mitral s′ and lateral tricuspid s′ are same velocities measured at mitral medial annulus and tricuspid lateral annulus respectively

Constrictive pericarditis was considered to be hemodynamically significant when there were clinical features of constriction with supportive echocardiographic and hemodynamic criteria as outlined earlier.

Strain by Speckle Tracking

A more recent echocardiographic approach to strain analysis is speckle tracking. Speckle tracking is a post-processing computer algorithm that uses the routine greyscale digital images. Although several manufacturers have devised speckle-tracking echocardiographic approaches, the fundamental approach is similar. [40-47]

Briefly, routine greyscale digital images of the myocardium contain unique speckle patterns. A user-defined region of interest is placed on the myocardial wall. Within this region of interest, the image-processing algorithm automatically subdivides regions into blocks of pixels tracking stable patterns of speckles. Subsequent frames are then automatically analyzed by searching for the new location of the speckle patterns within each of the blocks using correlation criteria and the sum of absolute differences. The location shift of these acoustic markers from frame to frame representing tissue movement provides the spatial and temporal data used to calculate velocity vectors. Temporal alterations in these stable speckle patterns are identified as moving farther apart or closer together and create a series of regional strain vectors.

Because strain information is not dependent on the Doppler angle of incidence like tissue Doppler imaging strain, several more strain analyses are possible, including longitudinal, circumferential, radial, and rotational. Currently, most echocardiography laboratories continue to use the subjective visual assessment of wall motion for resting and stress imaging for everyday clinical use, and strain imaging has been more often regarded as a research tool. Adoption of strain imaging in clinical practice appears to have been gaining momentum more recently so do forconstrictive pericarditis. [40-47]

We had assessed improvement in myocardial mechanics in CCP patients undergoing pericardiectomy using speckle tracking.This recently developed technique for characterization and quantification of myocardial deformation provided data noninvasively to better evaluation of the effectiveness of pericardiectomy. (i.e. in CCP, the epicardial dysfunction leads to depressed Global Circumferential Strain (GCS) and Left Ventricular Torsion (LVT), whereas Global Longitudinal Strain (GLS) and Global Radial Strain (GRS) are preserved and its strengths and weakness, and the potential present and future clinical applications.

Postoperative Studies

These included 3-monthly clinical examinations, electrocardiogram and chest radiographs. A minimum of 2 months follow-up was mandatory for this study. Preoperative studies were performed within 7 days before surgery. Postoperatively, all survivors were followed echocardiographically at the time of discharge and at 3 months. All late echoes have been grouped into one time period (3 months) with a range of no greater than 3 months. Echocardiographic data were measured according to American Society of echocardiographic criteria. [36]

Definitions and Acceptable Normal Values (Electronics)

For uniformity with other studies, total pericardiectomy was defined as wide excision of the pericardium with the phrenic nerves defining the posterior extent, the great vessels including the intrapericardial portion of superior vena cava and superior vena cava‐right atrial junction defining the superior extent, and the diaphragmatic surface, including the inferior vena cava‐right atrial junction defining the inferior extent of the pericardial resection.[3,4,8] Radical pericardiectomy was defined as excision of the pericardium as defined under total pericardiectomy including the removal of the pericardium posterior to the phrenic nerve and the diaphragmatic pericardium. Constricting layers of the epicardium were removed whenever possible. The atria and venae cavae were decorticated as a routine in all cases in this study group. Pericardiectomy was considered partial if both ventricles could not be decorticated completely because of dense myopericardial adhesions or calcification. [3,4,8]

The importance of unrecognized constricting epicardial (visceral pericardial) peel was described by Harrington in 1944 and successful pericardiectomy requires decortication of the ventricular epicardium and relief of all constricting layers. [3,4,7]

Transthoracic two‐dimensional, color‐flow Doppler echocardiographic studies, speckle tracking echocardiography were performed on all patients before and after the operation. Mitral, tricuspid, superior vena cava, hepatic vein, and pulmonary flow velocities were measured. Mitral or tricuspid regurgitation was assessed semi‐quantitatively as Grade 1 + to 4+. Ejection fraction was calculated using modified Quinones method. A constrictive pattern was defined as 25% or greater increase in mitral E‐velocity with expiration as compared with inspiration and an augmented (25% or more) diastolic flow reversal in the hepatic vein after the onset of expiration compared with inspiration.

Low output syndrome was diagnosed if the patient required inotropic support dopamine (4–10 μg/kg/min), dobutamine (5–10 μg/kg/min), epinephrine (0.01–0.1 μg/kg/min), milrinone (50 μg/kg intravenous bolus followed by 0.375–0.75 μg/kg/min), either isolated or in combination, in the operating room or intensive care unit to maintain stable hemodynamics in the absence of mechanical external compression after correction of all electrolytes or blood gas abnormalities and after adjusting the preload to its optimal value. Low output syndrome was also diagnosed if there was an increasing requirement of the above‐mentioned inotropes along with afterload reduction with sodium nitroprusside. Patients who received < 4 μg/kg/min of dopamine to increase renal perfusion were not considered to have low‐output syndrome. [3,4,8]

Accordingly, under the definition of low output syndrome after pericardiectomy, an integration of relevant clinical, laboratory and bedside echocardiographic criteria were used. The criteria for diagnosis were as follows: cold extremities, absent pedal pulses, decreased toe temperature, reduced systolic pressure, impaired renal function and oliguria (<1.0

$mL.kg^{-1}.h^{-1}$), metabolic acidosis, increased serum lactate levels ≥2.0 mmol/L, ≥2 hours), low mixed venous oxygen saturation (≤50%), and blunt sensorium. [3,4,8]

Perioperative mortality was defined as that occurring within 30 days after surgery. Cardiac-related death was defined as death due to cardiac causes, such as progressive congestive cardiac failure.6-10 Hypoproteinemia was defined as serum albumin level < 3.5 gm/dl. Renal dysfunction was defined as serum creatinine >2.0 gm/dl. [3,4,8,48]

Statistical Analysis

Statistical analysis was carried out using Stata 11.0 (College Station, Texas, USA). Continuous data were presented as mean±standard deviation, whereas categorical variables were presented as frequency distribution and percentage. Qualitative data were analysed by using 2 test or student's t test. Normality assumptions for continuous variables were assessed using Shapiro-Wilks test. Comparisons between two groups were done with the t-test. Echocardiographic parameters over a period of time between various clinical parameters were tested using Friedman's test. The correlation between mitral annular systolic velocities and left ventricular ejection fraction was assessed using Spearman's rank correlation. The p value of <0.05 was considered as statistically significant.

Results

There was no early death. Eleven (91.6%) patients had low-cardiac-output in the immediate postoperative period. All patients were routinely started on dopamine ($4\mu g.kg^{-1}.min^{-1}$) to increase renal perfusion on operation table after completing excision of the thickened pericardium. Patients with normal renal function were administered oral angiotensin-converting enzyme (ACE) inhibitors before weaning from inotropic agents. Postoperatively, digoxin, diuretics and ACE-inhibitors were weaned at varying time intervals.

Patients considered to have low output syndrome (n=11) required dopamine (4-10 $\mu g.kg^{-1}.min^{-1}$), epinephrine (0.01-0.1 $\mu g.kg^{-1}.min^{-1}$) and milrinone (50 µg/kg intravenous bolus followed by 0.375-0.75 $\mu g.kg^{-1}.min^{-1}$) either isolated or in combination. Median duration of inotrope requirement was 4 days (range 2-7 days) in these patients. Patients with normal renal function were administered oral angiotensin-converting enzyme inhibitors before weaning from inotropic agents. Two (16.66%) patients required intraoartic balloon counter pulsation as an additional support. There was marked reduction of filling pressure within 24 hours in the majority of patients (n=10) after total pericardiectomy [mean= right atrial pressure (RAP) 19.82±4.6 (18-29) to 6.11±0.85 (6-9); p<0.001]. Echocardiographically, diastolic filling characteristics remained abnormal in 3 (25%) patients of the study group in the immediate postoperative period. There was no late death. Reoperation was not required for any patients.

Follow-up

Follow-up was 100% complete (range 2-36 months, median 19) and yielded 19 patients-years of data.

At closing interval, 2 (16.6%) patients continued to remain in NYHA class II, and had persistent abnormalities of the diastolic filling pattern (p<0.05) on Doppler echocardiography. Pairwise comparison between symptomatic (n=2, 16.7%) and asymptomatic (n=10, 83.3%) patients revealed significant abnormality of the indexed IVC diameter (p<0.05) and increased left ventricular end-diastolic internal diameter (LVID) (p<0.05) in all patients of the symptomatic group. Nine of these symptomatic patients continued to remain in atrial fibrillation. Preoperatively, these symptomatic patients (n=2) were in NYHA class IV and were in atrial fibrillation. Thus, 2 (16.7%) of 5 patients who had preoperative atrial fibrillation continued to remain in atrial fibrillation. This could be the causative factor for alteration of left atrial mechanics and the left ventricular filling pressure which could lead to ongoing symptoms. Surgical techniques did not affect the outcome of atrial fibrillation.

These symptomatic patients (n=2, 16.7%) had significantly higher right atrial pressure in the immediate preoperative period compared to the asymptomatic group (n=10, 83.3%) [mean RAP=21.8±3.8 (symptomatic) vs 19.82±4.6 mmHg (asymptomatic), p<0.05]. Postoperatively, despite total pericardiectomy, the right atrial pressure of the symptomatic group continued to remain higher than the asymptomatic group [mean RAP=9.1±0.7 (symptomatic) vs 6.1±0.85 mmHg (asymptomatic), p<0.001)]. There were no differences of TDI-derived systolic and diastolic annular velocities of the mitral and tricuspid valves between symptomatic and asymptomatic patients in the preoperative period. Tissue Doppler imaging-derived mitral and tricuspid annular velocities failed to predict the postoperative outcome of patients undergoing pericardiectomy.

Data analyses and study interpretation of echocardiographic data (Tables 2 & 3)

Echo parameters	Mean±SD	Median (IQR)	Min - Max
RA Pressure (Preoperative)	14.17±2.82	14.00 (12.50-17.00)	9.0 - 18.0
RA Pressure (Postoperative)	10.25±1.48	10.00 (9.75-11.00)	8.0 - 13.0
DT (milliseconds) (1Preoperative)	98.07±12.97	101.10 (88.56-107.60)	76.7 - 116.0
DT (milliseconds) (2Postoperative)	114.77±11.78	118.35 (111.73-123.53)	84.8 - 126.2
DT (milliseconds) (3Follow-Up)	115.36±11.11	118.30 (111.90-123.85)	88.4 - 126.8
Index IVC (mm/mm2) (1Preoperative)	15.58±3.13	15.31 (13.22-17.60)	10.6 - 21.5
Index IVC (mm/mm2) (2Postoperative)	14.09±3.01	14.60 (11.45-15.82)	10.4 - 20.0
Index IVC (mm/mm2) (3Follow-Up)	13.68±2.49	13.75 (11.52-15.77)	10.4 - 18.0
LV IS (mm) (1Preoperative)	22.99±2.76	22.79 (21.42-24.85)	18.3 - 28.3
LV IS (mm) (2Postoperative)	22.74±2.75	22.58 (21.30-24.57)	18.1 - 28.1
LV IS (mm) (3Follow-Up)	22.54±2.80	22.13 (21.16-24.50)	17.9 - 28.0
LVEF (%) (1Preoperative)	47.83±6.81	50.00 (43.75-54.25)	35.0 - 55.0
LVEF (%) (2Postoperative)	50.00±4.77	50.00 (48.75-55.00)	40.0 - 55.0
LVEF (%) (3Follow-Up)	50.00±4.77	50.00 (48.75-55.00)	40.0 - 55.0
LVID (mm) (1Preoperative)	38.99±6.44	39.40 (36.92-43.06)	25.9 - 48.9
LVID (mm) (2Postoperative)	38.73±6.48	39.12 (36.62-42.88)	25.3 - 48.4
LVID (mm) (3Follow-Up)	38.40±6.42	38.98 (36.29-42.36)	25.2 - 48.0
Medial E' (cm/sec) (1Preoperative)	14.07±1.14	14.20 (13.67-14.75)	11.8 - 15.6
Medial E' (cm/sec) (2Postoperative)	14.68±1.36	14.85 (14.57-15.60)	11.8 - 16.1
Medial E' (cm/sec) (3Follow up)	14.95±1.48	15.40 (14.83-15.80)	11.8 - 16.9
Mitral Lateral E' (cm/sec) (1Preoperative)	11.86±1.63	11.95 (10.73-12.75)	9.4 - 14.5

Mitral Lateral E' (cm/sec) (2Postoperative)	13.49±1.95	13.15 (12.20-14.85)	10.8 - 16.7
Mitral Lateral E' (cm/sec) (3Follow up)	14.09±1.76	13.75 (12.85-15.12)	11.8 - 16.9
MVA (cm/sec) (1Preoperative)	42.33±6.01	42.05 (37.57-44.66)	33.8 - 55.5
MVA (cm/sec) (2Postoperative)	53.33±5.96	55.00 (48.00-58.65)	44.0 - 60.0
MVA (cm/sec) (3Follow-Up)	61.35±6.47	61.90 (55.20-66.45)	52.9 - 70.8
MVE (cm/sec) (1Preoperative)	79.57±11.28	80.91 (75.30-85.47)	58.7 - 96.7
MVE (cm/sec) (2Postoperative)	107.03±10.58	106.00 (103.10-112.00)	88.0 - 132.0
MVE (cm/sec) (3Follow-Up)	115.97±11.09	117.20 (112.60-118.85)	92.6 - 140.7
MVE (Respiratory Variation) (cm/sec) (1Preoperative)	43.10±4.37	44.00 (40.30-45.58)	34.7 - 49.3
MVE (Respiratory Variation) (cm/sec) (2Postoperative)	28.77±3.32	30.10 (25.87-30.85)	22.9 - 34.0
MVE (Respiratory Variation) (cm/sec) (3Follow-Up)	22.39±3.11	21.75 (20.62-24.90)	16.8 - 26.9
MVE/A (1Preoperative)	1.88±0.21	1.83 (1.74-2.02)	1.6 - 2.3
MVE/A (2Postoperative)	2.01±0.22	1.99 (1.82-2.18)	1.7 - 2.4
MVE/A (3Follow-Up)	1.87±0.22	1.79 (1.68-2.05)	1.6 - 2.2
SLRD (1Preoperative)	1.42±0.51	1.40 (1.05-1.65)	0.7 - 2.4
SLRD (2Postoperative)	2.58±0.69	2.45 (2.03-3.25)	1.7 - 3.6
SLRD (3Follow-Up)	2.61±0.67	2.50 (2.10-3.25)	1.7 - 3.6
Tricuspid Lateral E' (cm/sec) (1Preoperative)	12.75±1.42	13.10 (12.40-13.62)	8.9 - 14.3
Tricuspid Lateral E' (cm/sec) (2Postoperative)	13.64±1.16	14.05 (13.25-14.27)	10.8 - 14.9
Tricuspid Lateral E' (cm/sec) (3Follow-Up)	14.39±1.28	14.55 (13.78-15.45)	11.5 - 15.8
TVA (cm/sec) (1Preoperative)	28.86±2.47	29.09 (27.75-30.30)	23.7 - 33.0
TVA (cm/sec) (3Follow-Up)	43.12±6.37	43.40 (41.90-45.32)	29.2 - 53.6
TVA (cm2) (2Postoperative)	42.68±6.52	43.40 (40.48-45.17)	28.9 - 52.8
TVE (cm/sec) (1Preoperative)	52.94±5.69	52.40 (48.82-56.49)	43.8 - 64.3
TVE (cm/sec) (2Postoperative)	51.04±4.99	50.65 (47.80-54.65)	44.8 - 60.6
TVE (cm/sec) (3Follow-Up)	53.71±5.11	52.20 (50.72-58.65)	46.5 - 62.2
TVE (Respiratory Variation) (%) (1Preoperative)	26.33±1.96	26.55 (25.75-27.80)	21.9 - 28.5
TVE (Respiratory Variation) (%) (2Postoperative)	21.02±1.14	20.80 (20.28-21.60)	19.5 - 23.8
TVE (Respiratory Variation) (%) (3Follow-Up)	26.33±1.96	26.55 (25.75-27.80)	21.9 - 28.5
TVE/A (1Preoperative)	1.84±0.21	1.83 (1.68-2.00)	1.5 - 2.1
TVE/A (2Post Operative)	1.21±0.14	1.17 (1.12-1.23)	1.1 - 1.6
TVE/A (3Follow-Up)	1.26±0.20	1.17 (1.15-1.32)	1.1 - 1.6

DT=Deceleration time; IVC=Inferior vena cava; IVS=Interventricular septum; LVEF=Left ventricular ejection fraction; LVID=Left ventricular end-diastolic internal diameter; LVIS=Left ventricular end-systolic internal diameter; MVA=Transmitral late diastolic filling velocity; MVE=Transmitral early diastolic filling velocity; PW=Posterior wall of left ventricle; SD=Standard deviation; TVA=Transtricuspid late diastolic filling velocity; TVE=Transtricuspid early diastolic filling velocity; p<0.05=significant

Table 2: *Two-dimensional Doppler echocardiographic and tissue Doppler imaging derived annular velocities before and after pericardiectomy of all 12 patients in this study*

Echo parameters	Mean±SD	Median (IQR)	Min - Max
GCS (1Preoperative)	24.43±3.17	25.35 (24.42-25.92)	16.9 - 28.6
GCS (2Postoperative)	28.77±2.55	29.35 (28.22-29.95)	23.8 - 32.7
GCS (3Follow-Up)	30.08±2.61	30.30 (29.38-30.83)	24.9 - 35.5
GLS (1Preoperative)	19.63±2.98	19.95 (18.60-20.75)	13.9 - 24.2
GLS (2Postoperative)	21.66±2.61	21.75 (20.60-22.32)	17.8 - 26.8
GLS (3Follow-Up)	22.31±2.62	22.30 (21.08-23.02)	18.4 - 26.8
GRS (1Preoperative)	46.28±7.39	45.80 (42.92-50.17)	33.8 - 57.0
GRS (2Postoperative)	54.24±5.53	52.80 (50.70-57.77)	45.4 - 62.8
GRS (3Follow-Up)	54.26±5.54	52.80 (50.70-57.77)	45.4 - 62.8
GLS: AAL (1Preoperative)	18.39±4.22	19.15 (16.20-20.95)	10.2 - 25.3
GLS: AAL (2Postoperative)	20.37±3.92	20.75 (18.80-22.35)	13.4 - 27.3
GLS: AIS (1Preoperative)	21.76±5.94	21.60 (18.42-22.68)	13.7 - 35.1
GLS: AIS (2Postoperative)	22.28±5.63	21.95 (19.17-22.90)	14.8 - 35.2
GLS: BAL (1Preoperative)	18.03±2.41	18.60 (17.42-19.73)	12.4 - 20.6
GLS: BAL (2Postoperative)	22.22±1.69	22.15 (21.37-23.65)	19.3 - 24.3
GLS: BIS (1Preoperative)	19.05±2.13	18.95 (16.95-21.23)	16.3 - 21.8
GLS: BIS (2Postoperative)	19.67±1.76	19.75 (18.30-21.40)	17.1 - 21.8
GLS: MAL (1Preoperative)	20.18±3.84	19.00 (18.35-21.45)	14.5 - 28.2
GLS: MAL (2Postoperative)	22.95±3.80	22.00 (19.75-24.95)	18.8 - 30.3
GLS: MIS (1Preoperative)	21.62±5.25	20.20 (18.72-23.08)	14.2 - 33.8
GLS: MIS (2Postoperative)	22.56±4.64	21.10 (19.88-23.53)	17.4 - 33.8
GCS: MA (1Preoperative)	22.95±3.79	23.25 (21.28-25.88)	16.1 - 27.9
GCS: MA (2Postoperative)	29.15±3.60	29.10 (28.12-30.50)	21.3 - 34.6

GCS: MAL (1Preoperative)	23.53±3.19	23.90 (21.90-25.32)	16.8 - 29.1
GCS: MAL (2Postoperative)	30.70±1.64	31.05 (29.75-31.72)	27.5 - 33.4
GCS: MAS (1Preoperative)	23.45±3.68	23.30 (20.53-25.65)	18.3 - 29.3
GCS: MAS (2Postoperative)	25.82±2.83	26.40 (23.95-27.12)	21.4 - 30.7
GCS: MI (1Preoperative)	27.88±4.95	29.70 (25.82-31.13)	17.3 - 33.7
GCS: MI (2Postoperative)	28.64±5.03	30.25 (25.22-32.07)	18.8 - 34.7
GCS: MIL (1Preoperative)	22.82±4.30	22.65 (20.68-25.75)	14.3 - 29.3
GCS: MIL (2Postoperative)	30.32±3.25	28.75 (28.37-33.20)	25.9 - 35.5
GCS: MIS (1Preoperative)	26.02±3.81	26.15 (24.18-28.60)	18.2 - 31.6
GCS: MIS (2Postoperative)	28.02±2.81	29.00 (26.42-29.68)	22.1 - 31.9

AAL= Apical anterolateral; AIS= Apical inferioseptal; BAL= Basal anterolateral; BIS= Basal inferior septal; MAL= Mid anterolateral; MIS= Mid inferior septal; MA= Mid anterior; MAL= Mid anterolateral; MAS= Mid anteroseptal; MI= Mid inferior, GCS= Globus circumferential strain, GLS= Global longitudinal strain, GRCS= Global radial strain

Table 3: *Speckle tracking echocardiographic parameters before and after pericardiectomy of all 12 patients in this study*

To assess the characterization of the mitral and tricuspid annular velocity changes and speckle echocardiographic derived myocardial mechanics in patients undergoing pericardiectomy for constructive pericarditis, Friedman's test analysis revealed the following results:

Global circumferential strain (GCS)

Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the GCS changed significantly over time.

The mean GCS increased from a minimum of 24.43 at the I-preoperative timepoint to a maximum of 30.08 at the III-follow-up timepoint. This change was statistically significant (Friedman Test: $\chi 2 = 24.0$, $p = <0.001$).

As a significant change was observed in GCS over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the GCS differed significantly from the I-preoperative timepoint (**Table 4**)

Comparison of GCS at Various Time points vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II.Postoperative – I.Preoperative	4.33 (1.35)	4.10 (1.05)	2.50 - 6.90	0.038
III.Follow-Up - I-Preoperative	5.65 (2.07)	4.95 (1.62)	3.40 - 10.40	<0.001

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 4: *Global circumferential strains (GCS)*

The GCS differed significantly from the I-preoperative timepoint at the following timepoints: II-Postoperative, III-follow-up. The maximum change from the I-preoperative timepoint was observed at the 3 follow-up time point (**Figure 4**).

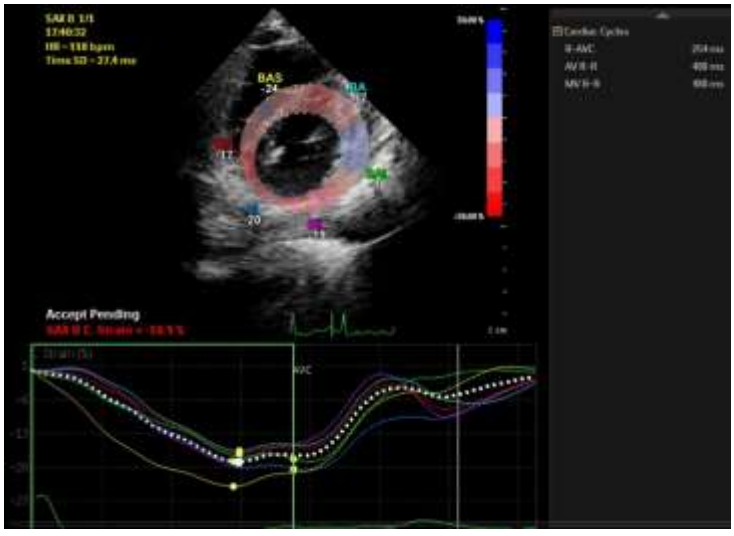


Figure 4: *Two-Dimensional speckle echocardiography showing global circumferential strain (GCS pattern) - Postoperative*

Global longitudinal strain (GLS): Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the GLS changed significantly over time.

The mean GLS increased from a minimum of 19.63 at the I-preoperative timepoint to a maximum of 22.31 at the III-follow-up timepoint. This change was statistically significant (Friedman Test: $\chi 2 = 23.5$, $p = <0.001$).

As a significant change was observed in GLS over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the GLS differed significantly from the I-preoperative timepoint (**Table 5**).

Comparison of GLS at Various Time points vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II-Postoperative - I-Preoperative	2.02 (0.85)	2.00 (0.65)	0.90 - 3.90	0.029
III-Follow-Up - I-Preoperative	2.68 (0.81)	2.60 (0.60)	1.40 - 4.60	<0.001

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 5: *Global longitudinal strain (GLS)*

The GLS differed significantly from the I-preoperative timepoint at the following timepoints: II-postoperative, III-follow-up. The maximum change from the I-preoperative timepoint was observed at the III-Follow-Up timepoint (**Figures 5 and 6**).

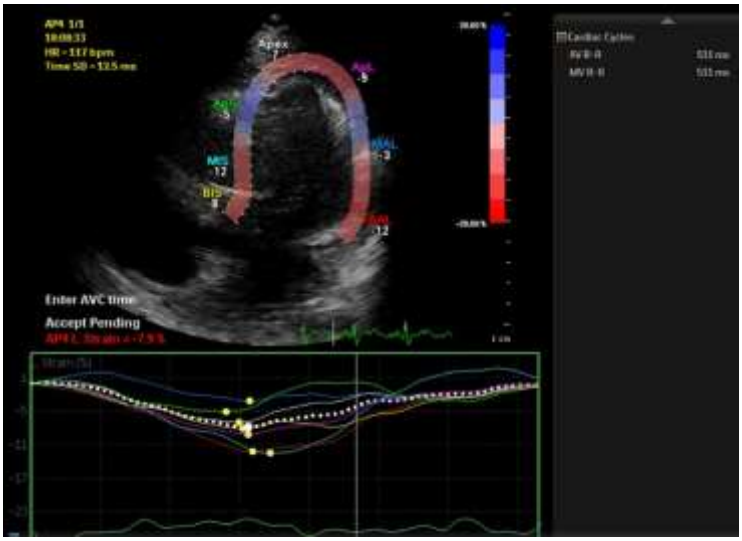


Figure 5: *Two-Dimensional speckle echocardiography showing global longitudinal strain (GLS pattern) – Preoperative*

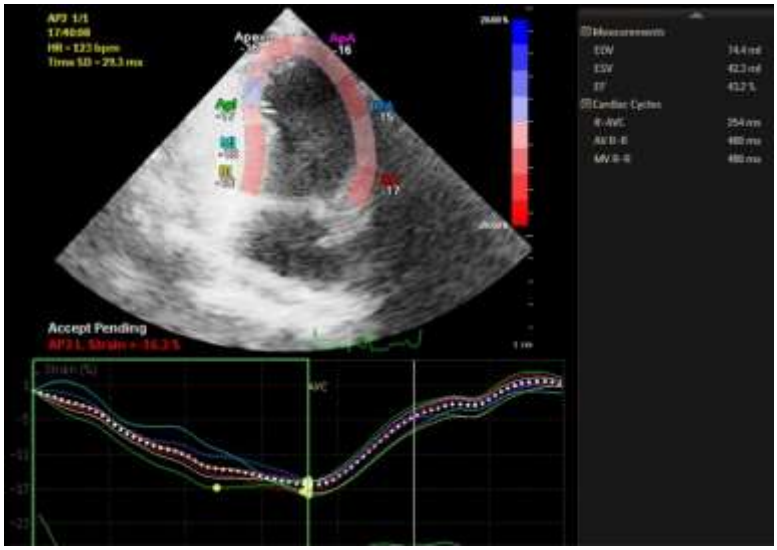


Figure 6: *Two-Dimensional speckle echocardiography showing global longitudinal strain (GLS pattern) – Postoperative*

Global radial strain (GRS): Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the GRS changed significantly over time.

The mean GRS increased from a minimum of 46.28 at the I-preoperative timepoint to a maximum of 54.26 at the III-follow-up timepoint. This change was statistically significant (Friedman Test: $\chi 2 = 23.4$, $p = <0.001$).

As a significant change was observed in GRS over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the GRS differed significantly from the I-preoperative timepoint (**Table 6**)

Comparison of GRS at Various Timepoints vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II-Postoperative - I-Preoperative	7.96 (3.54)	7.20 (2.43)	3.60 - 15.50	0.001
III-Follow-Up - I-Preoperative	7.97 (3.54)	7.20 (2.57)	3.60 - 15.50	<0.001

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 6: *Global radial strain (GRS)*

The GRS differed significantly from the I-preoperative timepoint at the following timepoints: II-Postoperative, III-follow-up. The maximum change from the I-preoperative timepoint was observed at the III-Follow-Up timepoint (**Figure 7**).

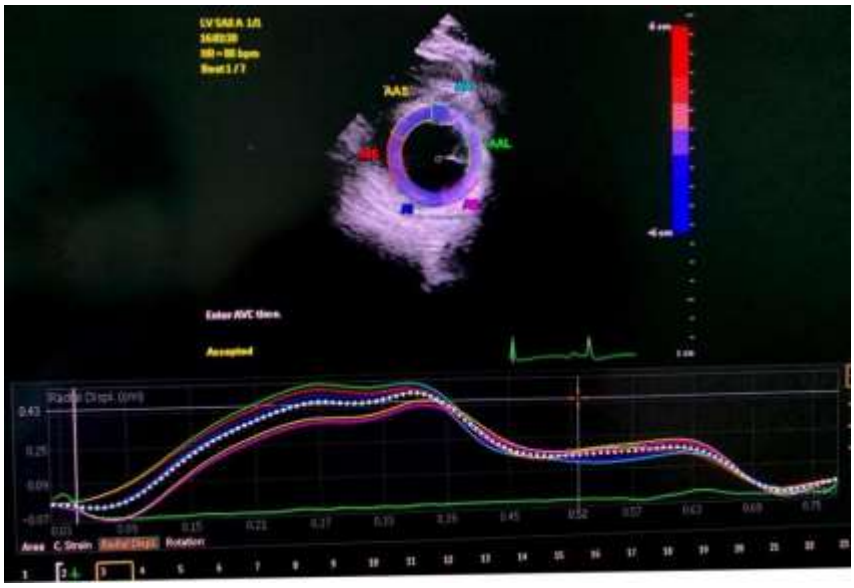


Figure 7: *Two-Dimensional speckle echocardiography showing global radial strain (GRS pattern) – Postoperative*

Indexed IVC diameter (mm/m^2)

Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the Index IVC (mm/m^2) changed significantly over time.

The mean Index IVC (mm/m^2) decreased from a maximum of 15.58 at the I-preoperative timepoint to a minimum of 13.68 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi 2$ = 20.8, p = <0.001).

As a significant change was observed in Index IVC (mm/m^2) over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the Index IVC (mm/m^2) differed significantly from the I-preoperative timepoint (**Table 7**).

Comparison of Index IVC (mm/m^2) at Various Time points vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II-Postoperative - I-Preoperative	-1.48 (1.01)	-1.55 (1.90)	-2.80 - -0.10	0.002
III-Follow-Up - I-Preoperative	-1.90 (0.98)	-2.14 (1.28)	-3.50 - -0.24	<0.001

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 7: *Indexed IVC diameter (mm/m^2)*

The Index IVC (mm/m^2) differed significantly from the I-preoperative timepoint at the following timepoints: II-Postoperative, III-Follow-Up.

The maximum change from the I-preoperative timepoint was observed at the III-Follow-Up timepoint.

Left ventricular end-systolic internal diameter (LVIS)

Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the LV IS (mm) changed significantly over time.

The mean LVIS (mm) decreased from a maximum of 22.99 at the I-preoperative timepoint to a minimum of 22.54 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 24.0, p = <0.001).

As a significant change was observed in LV IS (mm) over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the LV IS (mm) differed significantly from the I-preoperative timepoint (**Table 8**)

Comparison of LV IS (mm) at Various Timepoints vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II-Postoperative - I-Preoperative	-0.25 (0.15)	-0.22 (0.25)	-0.50 - -0.03	0.038
III-Follow-Up - I-Preoperative	-0.45 (0.19)	-0.49 (0.31)	-0.68 - -0.12	<0.001

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 8: *Left ventricular end-systolic internal diameter (LVIS)*

The LV IS (mm) differed significantly from the I-preoperative timepoint at the following timepoints: II-Postoperative, III-Follow-Up.

The maximum change from the I-preoperative timepoint was observed at the 3Follow-Up timepoint.

Left ventricular ejection fraction (LVEF)

Non-parametric tests (Friedman test) were used to make statistical inference as data were not normally distributed. Friedman test was used to explore whether the LVEF (%) changed significantly over time.

The mean LVEF (%) increased from a minimum of 47.83 at the I-preoperative timepoint to a maximum of 50.00 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 12.0, p = 0.002).

As a significant change was observed in LVEF (%) over time using the Friedman Test, post-hoc pairwise analysis was performed to explore at which timepoints the LVEF (%) differed significantly from the I-preoperative timepoint (**Table 9**) There was no significant difference between any of the timepoints as compared to the I-preoperative timepoint in terms of LVEF (%).

Comparison of LVEF (%) at Various Timepoints vs I-Preoperative	Mean (SD) of Difference	Median (IQR) of Difference	Range of Difference	p value
II-Postoperative - I-Preoperative	2.17 (2.52)	0.50 (5.00)	0.00 - 5.00	0.158
III-Follow-Up - I-Preoperative	2.17 (2.52)	0.50 (5.00)	0.00 - 5.00	0.158

Post-Hoc pairwise tests for Friedman test performed using Nemenyi Test method for p value correction. Green background denotes statistically significant difference at p < 0.05.

Table 9: *Left ventricular ejection fraction (LVEF)*

Left ventricular end-diastolic internal diameter (LVID)

The mean LVID (mm) decreased from a maximum of 38.99 at the I-preoperative timepoint to a minimum of 38.40 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 24.0, p = <0.001).

Mitral medial E'

The mean Medial E' (cm/sec) increased from a minimum of 14.07 at the I-preoperative timepoint to a maximum of 14.95 at the III-Follow up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 12.9, p = 0.002).

Mitral lateral E'

The mean Mitral Lateral E' (cm/sec) increased from a minimum of 11.86 at the I-preoperative timepoint to a maximum of 14.09 at the III-Follow up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 23.5, p = <0.001).

Transmitral late diastolic filling velocity (MVA)

The mean MVA (cm/sec) increased from a minimum of 42.33 at the I-preoperative timepoint to a maximum of 61.35 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 24.0, p = <0.001)

Transmitral early diastolic filling velocity (MVE)

The mean MVE (cm/sec) increased from a minimum of 79.57 at the I-preoperative timepoint to a maximum of 115.97 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 24.0, p = <0.001).

MVE (Respiratory Variation) (cm/sec) over time

The mean MVE (Respiratory Variation) (cm/sec) decreased from a maximum of 43.10 at the I-preoperative timepoint to a minimum of 22.39 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2$ = 24.0, p = <0.001).

Assessment of change in MVE/A over time

The mean MVE/A increased from 1.88 at the I-preoperative timepoint to a maximum of 2.01 at the II-Postoperative timepoint, and then decreased to

1.87 at the III-Follow-Up timepoint. This change was not statistically significant (Friedman Test: $\chi2 = 5.1$, $p = 0.076$).

Change in Tricuspid Lateral E' (cm/sec)

The mean Tricuspid Lateral E' (cm/sec) increased from a minimum of 12.75 at the I-preoperative timepoint to a maximum of 14.39 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2 = 22.0$, $p = <0.001$).

Assessment of change in transtricuspid late diastolic filling velocity

The mean TVA (cm/sec) increased from a minimum of 28.86 at the I-preoperative timepoint to a maximum of 43.12 at the III-Follow-Up timepoint, and then decreased to 42.68 at the II-Postoperative timepoint. This change was statistically significant (Friedman Test: $\chi2 = 21.6$, $p = <0.001$).

Transtricuspid early diastolic filling velocity (TVE)

The mean TVE (cm/sec) decreased from 52.94 at the I-preoperative timepoint to a minimum of 51.04 at the II-Postoperative timepoint, and then increased to 53.71 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2 = 8.7$, $p = 0.013$).

Assessment of change in TVE (Respiratory Variation) (%) over time

The mean TVE (Respiratory Variation) (%) decreased from a maximum of 26.33 at the I-preoperative timepoint to a minimum of 21.02 at the II-Postoperative timepoint, and then increased to 26.33 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2 = 24.0$, $p = <0.001$).

TVE/A

The mean TVE/A decreased from a maximum of 1.84 at the I-preoperative timepoint to a minimum of 1.21 at the II-Post Operative timepoint, and then increased to 1.26 at the III-Follow-Up timepoint. This change was statistically significant (Friedman Test: $\chi2 = 17.9$, $p = <0.001$).

Discussion

So far as we are aware, there have been few published studies in the literature investigating the role of tissue Doppler imaging-derived parameters of mitral and tricuspid annular motion on global and regional ventricular function and speckle echocardiographic derived variables of myocardial mechanics and their role in differentiating CP from RCM.[7-17]

The principal findings of this investigation include:

1. Significant reduction in indexed IVC diameter and significant improvement of early and late diastolic filling of both left and right ventricle in the immediate as well as late postoperative period in the majority of patients.
2. Presence of "annulus reversus" of mitral valve, where mitral lateral e′ velocity was lower than medial e′ velocity in all patients in this study before surgery.
3. Significant decrease of mitral medial e′ velocity in early as well as late postoperative period. Following pericardiectomy, the lateral e′ velocity of the mitral valve exhibited insignificant reduction in the immediate postoperative period and significant reduction in the late postoperative period.
4. The identification of "annulus reversus" of the tricuspid valve in all patients.
5. Exhibition of normalization of tricuspid lateral/medial e′ following pericardiectomy during follow-up.
6. Proportionately greater postoperative reduction in tricuspid lateral e′ velocity compared to mitral annulus values.
7. Demonstration of significant reduction in mitral and tricuspid systolic annular velocity (lateral and medial) following pericardiectomy in the postoperative period.
8. Exhibition of inspiratory decreases in peak transmitral flow and inspiratory increase in transtricuspid flow in all patients in the preoperative period. Following pericardiectomy, transmitral early diastolic filling velocity continued to remain abnormal in 10 (18.5%) patients upto 6 months. These symptomatic patients (n=10, 18.5%) continued to have higher indexed IVC diameter and persistent atrial fibrillation (n=9) in the postoperative period. Tissue Doppler imaging-derived mitral and tricuspid annular velocities failed to predict the postoperative symptomatic status of patients undergoing pericardiectomy.
9. Preoperative atrial fibrillation was a predictor of poor prognostic outcome following pericardiectomy.
10. The Global Circumferential Strain (GCS) significantly increased from preoperative value of 24.43±3.17 to 28.77±2.55.and it further improved to 30.08±2.61 on 6-month follow-up.
11. The Global Longitudinal Strain (GLS) slightly increased from preoperative value of 19.63±2.98. to 21.66±2.61.and on 6 month follow-up to 22.31±2.62.
12. The Global Radial Strain(GRS) also increased from preoperative value of 46.28±7.39. to postoperative of 54.24±5.53 with no significant improvement on 6 month follow-up.(54.26±5.54.).

Currently, tissue Doppler imaging is an integral part of an echocardiography examination in various areas of cardiology. Tissue Doppler imaging offers a quantitative measurement of regional and global myocardial tissue function. In particular, the assessment of longitudinal mitral annular motion provides an accurate estimate of global left ventricular function [12-16] and it has further facilitated the detection of constrictive pericarditis. Since the mechanoelastic properties of the myocardium are preserved in constrictive pericarditis, the longitudinal mitral annular velocities remain normal or can be exaggerated as lateral expansion in constrictive pericarditis is limited. Garcia and colleagues were the first to report that measurement of longitudinal axis expansion by tissue Doppler imaging provided a clinically useful distinction between constrictive pericarditis and restrictive cardiomyopathy. [16] Rajagopalan and colleagues showed that a peak e' velocity ≥ 8 cm/s could discriminate between constrictive pericarditis and restrictive cardiomyopathy with high sensitivity (89%) and specificity (100%). [51] Studies by Ha and colleagues and by Sohn and colleagues recommended that e' velocity can provide a helpful diagnostic indicator and should be measured routinely in the evaluation of heart failure or suspected constrictive pericarditis. [30,31] Ha and colleagues recommended the same 8 cm/s cut off value for diagnosis of constrictive pericarditis, where e' velocity is equal or greater than 8 cm/s, with 95% sensitivity and 96% specificity.[49] Ha and colleagues also evaluated the role of tissue Doppler imaging in the diagnosis of constrictive pericarditis in patients without diagnostic respiratory variation of transmitral early diastolic filling velocity. It was confirmed that e' velocity was well-preserved independent of any respiratory variation in mitral inflow velocities. Other studies suggested that e' should be used with caution if constrictive pericarditis is combined with myocardial diseases, extensive annular calcification or segmental non-uniform myocardial velocities. [35]

Several investigators have shown that E/e' ratio correlates well with left ventricular filling pressure. [36] E/e' >15 identifies increased left ventricular filling pressure while E/e' <8 describes normal filling pressure. Ha introduced the concept of annulus paradoxus, which describes the paradoxical behavior of the mitral annulus in constrictive pericarditis. [49] Ha found that an inverse relationship exists between E/e' and left ventricular filling pressure, which can be explained by the fact that in constrictive pericarditis the mitral annulus has an exaggerated longitudinal motion leading to an increase in e', despite high filling pressures.[49]

In normal subjects, the mitral lateral annulus e' velocity is higher than the medial annulus e' velocity. Reuss and colleagues identified the reversal of the normal relationship of mitral lateral e' and medial e' velocities in constrictive pericarditis, where mitral lateral e' velocity is lower than medial e' velocity, therefore lateral/medial e' ratio is inverted and called, "annulus reversus". [33] This finding is based on the tethering of the adjacent fibrotic and scarred pericardium, which influences sthe lateral mitral annulus in patients with constrictive pericarditis. In a patient with preserved mitral e' velocities (> 8 cm/sec) and a low E/e' ratio (< 8) with high left ventricular filling pressure, the recognition of, "annulus reversus" should alert to the diagnosis of constrictive pericarditis.[11-18]

In general, tissue Doppler imaging offers incremental diagnostic information to M-mode, 2D echo and transmitral flow Doppler for detecting constrictive physiology, with a reported sensitivity and specificity of 88.8% and 94.8%, respectively. [11-18]

Kim JS and colleagues examined the medial annular velocities in patients with constrictive pericarditis after pericardiectomy in 16 patients and found that e' decreased significantly after pericardiectomy. [38] However, there is no substantive data on mitral annulus systolic velocity and tricuspid annulus velocity in constrictive pericarditis and no data on the effect of pericardiectomy on these annular velocities. The effect on pericardiectomy on mitral and tricuspid annular velocities, which may provide further insight into the mechanism of annulus motion, is unknown.

Building on these observations from a small number of patients, our aim was to provide a comprehensive evaluation of tissue Doppler imaging at both mitral and tricuspid annuli in a larger number of patients and follow their evolution after pericardial resection.

Early diastolic mitral annulus velocity

We confirmed the presence of "annulus reverses" in patients with constrictive pericarditis. Based on the hypothesis that the lateral annulus motion is restricted by the constricting pericardium and that medial annulus diastolic motion increases in compensation, it may be anticipated that medial mitral annulus velocity decreases and lateral annulus velocity increases after pericardiectomy, and that mitral lateral/medial e' ratio normalizes. In this study, both was confirmed.

Early diastolic tricuspid annulus velocity

In this study, all patients exhibited a reduction in tricuspid lateral e' velocity after pericardiectomy. The phenomenon of "annulus reversus" was observed in all patients in this study. There was reduced lateral tricuspid annular velocity (e') in all patients and normalization of the tricuspid lateral/medial e' ratio following pericardiectomy during the follow up period. Therefore, the above mentioned mechanisms operative at the mitral annulus may as well be responsible for findings at the tricuspid annulus.

Garcia and colleagues were the first to report that the measurement of longitudinal axis expansion by tissue Doppler imaging provided a clinically useful distinction between constrictive pericarditis and restrictive cardiomyopathy.[20] Our studies have confirmed that medial e' velocity was relatively normal or even accentuated in all patients with constrictive pericarditis irrespective of characteristic respiratory variation in mitral E velocity. Characteristic respiratory variation across the mitral valve and tricuspid valve was present in all patients in this study group.

In this study, there was proportionately greater postoperative reduction in tricuspid lateral e' velocity compared to mitral annulus values. As the pericardial disease process is often asymmetric, being more pronounced over the right ventricle, annular motion here may be expected to be most exaggerated before pericardiectomy and following decortication approximate normality.

Similar observations were noted by other investigators. [39] Sengupta and colleagues found higher net twist but no significant increase in torsion post-pericardiectomy, a conclusion limited by small number of patients and early timing of the postoperative studies when restoration of function may have been incomplete.[34,35]

To explain this paradoxical relationship between s' and stroke volume in constrictive pericarditis, Veress and colleagues postulated that systolic and diastolic motion of the mitral annulus are closely coupled in part via elastic recoil mechanisms. [39]

Systolic annulus velocity

s' by tissue Doppler imaging reflects the peak velocity of myocardial fiber shortening in the longitudinal direction and provides a more sensitive assessment of global left ventricle and right ventricle systolic function than 2-D or M-mode imaging. s' has been correlated with peak positive dP/dt and left ventricular ejection fraction in patients with dilated cardiomyopathy, hypertensive heart disease and myocardial infarction. There is little information on mitral and tricuspid s' velocities in patients with constrictive pericarditis. Studies in very small patient population have either compared s' velocity between constrictive pericarditis and restrictive cardiomyopathy or measured changes in s' velocity pre- and post-pericardiectomy. [39]

The mean s' velocity in all patients in this study was lower both before and after pericardiectomy than published normative values [42] and also lower, especially prepericardiectomy. These observations are consistent with previous smaller studies. This finding seems counterintuitive since s' velocity is expected to increase with augmented stroke volume after pericardiectomy.

We postulated that stroke volume in constrictive pericarditis is closely coupled, in part via elastic recoil mechanisms. Thus, in the pre-pericardiectomy setting, both longitudinal systolic and diastolic motion of the annuli are exaggerated while following release of pericardial constraint, both decrease in tandem. This hypothesis is supported by the moderate to high correlation between annular s' and e' as well as s' and a', especially before pericardiectomy when restorative forces may be most operative.

There appeared to be proportionately greater postoperative reduction in tricuspid lateral or right ventricle s' and e' compared to mitral annulus values. As the pericardial disease process is often asymmetric, being most pronounced over the right ventricle, annular motion here may be expected to be most exaggerated before pericardiectomy, and following decortication, approximate normality. However, the disproportionate reduction in tricuspid lateral s' and e' probably seems also from postoperative right ventricular dysfunction, which was moderate in 7 (21.2%) patients.

Left ventricular ejection fraction did not change despite the expected increase in stroke volume after pericardiectomy. It is postulated that after pericardial resection, left ventricular filling increases and other elements of left ventricular shortening including torsion are recruitable, contributing to better cardiac output and compensating for abnormal longitudinal function. [28] Sengupta and colleagues found higher net twist but no

significant increase in torsion post pericardiectomy, a conclusion limited by small patient numbers and early timing of the postoperative studies when restoration of function may have been incomplete. [28] To confirm this hypothesis, detailed analysis of myocardial mechanics in a larger number of patients pre- and post-pericardiectomy will be required. [34,35]

Monitoring of intracardiac pressures during pericardiectomy has been proposed to evaluate the result of decortications but Viola [40] argued against the value of this assessment because further recovery of myocardial failure may occur late after pericardiectomy. In this study, we showed that there is a relationship between the degree of decrease in atrial pressure after pericardiectomy and postoperative diastolic function. Further, early abnormalities in diastolic filling pattern may improve in the late follow-up; however, the long-term hemodynamic result may not be predicted by the immediate postoperative Doppler echocardiographic findings.

Symptoms may persist after successful pericardiectomy in patients with mixed constrictive-restrictive disease because of abnormalities in intrinsic myocardial compliance. In our study group, 2 (16.6%) patients continued to have NYHA Class II symptoms late postoperatively. These patients exhibited higher right atrial pressure (mean±SD= 10.25±1.48 mmHg), raised indexed inferior vena caval diameter, (mean±SD= 14.09±3.01 mm), higher LVID (mean±SD= 40.27.4.7 mm) and persistently abnormal transmitral early diastolic filling velocity (MVE, mean±SD=115.97±11.09 cm/sec, MVE/A 1.87±0.22) in the 6 month follow up.

There are limited studies to assess the extent of myocardial damage with two-dimensional speckle tracking echocardiography (2DSTE). Longitudinal, radial, and circumferential mechanics of the LV were quantified by 2DSTE on 26 patients with constrictive pericarditis and 19 patients with restrictive cardiomyopathy by Sengupta and associates. In comparison with controls, constrictive pericarditis patients had impaired left ventricle circumferential strain (ε) (base; −16±6 vs −9±6%; P.01) significantly reduced in constrictive pericarditis patients when compared to a control group.[55] Amaki and associates validated 2DSTE and cardiac magnetic resonance imaging (CMR) on 28 patients with constrictive pericarditis and 30 patients with restrictive cardiomyopathy.

The global longitudinal scale was higher in patients with constrictive pericarditis than in those with restrictive cardiomyopathy [−18.5% (−20.1 to −15.2) vs −11.6% (−14.6 to −9.3); *P*<.001], and both techniques were found to have similar diagnostic value (area under the curve, 0.84 vs 0.88 for cardiac magnetic resonance imaging and echocardiography, respectively). [56,57] The ratio between lateral and septal longitudinal ε was not significantly different among the 3 groups. Patients with RCM had marginally lower circumferential ε when compared with CP [−23.9 (−28.3 to −20.2) vs −19.3 (−23.3 to −16.0)%, *P*=.07].[57]

Negishi and colleagues investigated 83 patients with CCP whether pericardiectomy improves myocardial mechanics using 2DSTE. Besides left ventricle ε, the authors studied left ventricle longitudinal and rotational displacement. Longitudinal displacement of left ventricle opposing walls was similar, but decreased in constrictive pericarditis compared to controls. After pericardiectomy, septal displacement decreased, but lateral displacement increased. Septal longitudinal ε was similar between two groups, but lateral longitudinal ε was lower in the constrictive pericarditis group. Septal longitudinal ε decreased significantly (−20.3±5.0% vs −17.7±4.6%, P=.032) after surgery, but lateral longitudinal ε did not (−14.7±5.8% vs −15.2±3.4%, P=.51). Patients with constrictive pericarditis had lower absolute values of Global longitudinal strain (−20.1±1.9 vs −16.2±3.3%, P<.01) and Global circumferential strain (−20.7±5.1 vs −14.7±5.0%, P<.01), with no significant difference in Global radial strain (50.4±16.2 vs40.8±18.8%, P=.07) compared with controls. No significant difference in SLRD between controls and constrictive pericarditis (−2.3±3.3 vs −0.6±3.0°, P=.07). After pericardiectomy, SLRD increased significantly (−0.8±3.3% vs 2.1±3.0, P<.01). No changes in GLS (−15.6±3.9% vs −15.8±3.2, P=.88) and GRS (37.4±18.9% vs 39.1±16.5%, P=.73) after pericardiectomy. GCS increased (−13.5±5.7 vs −17.6±5.5, P<.01) after pericardiectomy.[59]

In this study, speckle echocardiographic derived parameters revealed: i) significant increase of the Global Circumferential Strain (GCS) from preoperative value of 24.43±3.17 to 28.77±2.55 and further improvement to 30.08±2.61 on 6 month follow-up, ii) slight increase of the Global Longitudinal Strain (GLS) from preoperative value of 19.63±2.98 to 21.66±2.61and on 6 month follow-up to 22.31±2.62, and iii) increase of the Global Radial Strain (GRS) from preoperative value of 46.28±7.39 to postoperative of 54.24±5.53 with no significant improvement on 6 month follow- up.

This is in accordance with the study conducted by Negishi and associates who demonstrated increase of GCS among 83 patients with constrictive pericarditis undergoing pericardiectomy 13.5±5.7 to 17.6±5.5, p<.01); with no significant difference in GLS and GRS

In this study, 5 (41.7%) patients had extensive pericardial calcification over the anterior and inferior surfaces of the right and left ventricle. However, total pericardiectomy including removal of the calcified pericardium overlying the anterolateral and diaphragmatic surface of the right ventricle was achieved in all patients of the study group. These patients in the immediate postoperative period required higher inotropic support because of low cardiac output. This phenomenon may reflect underlying myocardial damage or atrophy secondary to long standing encasement and penetration of the myocardium by calcium spurs, and persistent inflammation. We believe that subjecting the newly liberated right, and perhaps left ventricle to even moderately elevated filling pressure led to increased wall stress and deteriorating cardiac function possibly due to fibrous invasion of the myocardium and varying grades of myocardial atrophy. We concur with the observations of other investigators regarding the possibility of residual constriction, fibrous invasion of the myocardium and abnormal ventricular compliance secondary to myocardial alterations.[4,5,40,41] The utility of speckle tracking echocardiography and tissue Doppler imaging in identifying residual constrictive pericarditis requires further investigation on a large cohort of patients correlating the clinical outcomes.

Clinical Implications

With a GCS, GLS and GRS data and mitral inflow profile of increased filling pressure and expiratory hepatic vein diastolic reversals as well as abnormal ventricular septal motion in constrictive pericarditis, the disease can be diagnosed by echocardiography with assistance of cardiac catheterization. The characteristic pattern of annulus velocities revert to normal after pericardiectomy in asymptomatic patients.

It will be of clinical interest to investigate if the extent of postoperative changes in annular velocities can predict clinical outcome after pericardiectomy. Further evaluation of these indices as a marker of successful treatment of constrictive pericarditis is warranted. Further studies are underway to compare these speckled tracking and tissue Doppler derived parameters achieved by median sternotomy and anterolateral thoracotomy approaches on a large number of patients.

Study Limitations

This study included only a small number of patients and 7 (53.8%) patients underwent pericardiectomy via median sternotomy. Hence, the tissue Doppler imaging and speckle derived variables could not be compared with anterolateral thoracotomy approach.

Secondly, we only recorded tissue Doppler imaging of longitudinal axis motion in the 4-chamber view. Due to the local tethering effect, analysis of multiple annular regions could have provided additional helpful data. It would also be helpful to characterize radial and circumferential function for a better understanding of the mechanics of the unique annular motion in constrictive pericarditis and effects of pericardiectomy.

Thirdly, due to its limited resolution, 2D speckle tracking echocardiography might be suboptimal in patients with calcific CCP. Other limiting factors are data procured from postsurgical patients with postoperative adhesions, and patients with atrial fibrillation.

Conclusions

This study demonstrates that patients with congestive heart failure and normal left ventricular ejection fraction, preserved or increased medial e' velocity strongly suggests constrictive pericarditis. The diagnosis is further supported if medial e' is higher than lateral e'. The annulus velocity study demonstrates that in patients with constrictive pericarditis:

i. Mitral medial e' velocity is usually higher than mitral lateral e' velocity which is a reversal of the observed relationship in normal individuals.

ii. As the pericardial disease process is often asymmetric being more pronounced over the right ventricle, annular motion was most exaggerated before pericardiectomy at the tricuspid annular level.

iii. All mitral and tricuspid annular velocities (e', and s') decreases after pericardiectomy with normalization of the lateral/medial e' velocity ratio.

iv. There is statistical significant improvement in the Global cirumferential strain than in global longitudinal and global radial strain after pericardiectomy

We conclude that tissue Doppler imaging and speckle tracking echocardiography are useful investigative modalities for serial evaluation of patients undergoing pericardiectomy. It can be performed serially with a high degree of reproducibility. It can be used for late postoperative assessment, obviating the need for frequent cardiac catheterization. In addition, we propose routine and serial utilization of this modality may be the investigation of choice for assessment of adequacy of pericardial resection and its subsequent effect on ventricular remodeling.

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