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Tissue Doppler Imaging-derived Mitral and Tricuspid Annular Velocities: Non-predictors of Operative Outcome in Patients Undergoing Pericardiectomy for Chronic Constrictive Pericarditis

Ujjwal Kumar Chowdhury[1*], Kartik Patel[1], Lakshmi Kumari[1], Sandeep Seth[2], Sheil Avneesh[1], Anand Kumar Mishra[1], Mani Kalaivani[3] and Suruchi Hasija[4]

*[*1]Departments of Cardiothoracic and Vascular Surgery, All India Institute of Medical Sciences, New Delhi-110029, India*

[2]Department of Cardiology, All India Institute of Medical Sciences, New Delhi-110029, India

[3]Department of Biostatistics, All India Institute of Medical Sciences, New Delhi-110029, India

[4]Department of Cardiac Anesthesia, All India Institute of Medical Sciences, New Delhi-110029, India.

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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 in patients undergoing pericardiectomy for chronic constrictive pericarditis and identify the relationship if any of the tissue Doppler imaging-derived variables with patient's symptomatic status following surgery.

Patients and methods: Fifty-four patients undergoing pericardiectomy for constrictive pericarditis aged 7 years to 70 years (mean 31.0 ± 16.8 years) were studied for 24.4 ± 10.8 months (range 6-42 months). They underwent Doppler flow velocity and TDI studies. Generalized estimating equation was used to test the changes in TDI-derived mitral and tricuspid annular velocities 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, 10 (18.5%) patients continued to be in New York Heart Association class II and 9 of them continued to remain in atrial fibrillation. There were no differences of TDI-derived systolic and diastolic annular velocities of the mitral and tricuspid valves in the preoperative period between symptomatic and asymptomatic patients.

Conclusion: We conclude that preoperative atrial fibrillation is a predictor of poor prognostic outcome following pericardiectomy. Tissue Doppler imaging-derived mitral and tricuspid annular velocities are non-predictors of postoperative outcome following pericardiectomy. Tissue Doppler imaging is a useful investigative modality for diagnosis of constrictive pericarditis and not a useful indicator for postoperative evaluation.

Keywords: Tissue Doppler imaging, Chronic constrictive pericarditis, Pericardiectomy, Echocardiography

INTRODUCTION

Pericardiectomy is usually the only accepted curative treatment for constrictive pericarditis (CP) and several studies including ourselves have shown its efficacy in improving symptoms with normalization of hemodynamics in the majority of cases [1-6]. Despite advances in knowledge about CP, the disease is frequently difficult to diagnose even after comprehensive evaluation and continues to be elusive mimicking restrictive cardiomyopathy (RCM), endo myocardial fibrosis or chronic liver disease [1-8]. No single pathognomonic echocardiographic finding exists for CP. To distinguish CP from other diseases including RCM, a combination of findings need to be used.

Corresponding author: Dr. Ujjwal Kumar Chowdhury, Department of Cardiothoracic and Vascular Surgery, All India Institute of Medical Sciences, Ansari Nagar, New Delhi-110029, India, Tel: 91-11-26588700; Ext. 4835; Fax: 91-11-26588641; E-mail: ujjwalchow@rediffmail.com; ujjwalchowdhury@gmail.com

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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 CP from RCM without overlap [7,8].

Because the mechanoelastic properties of the myocardium are preserved in CP, 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 [7-10]. In constrictive pericarditis, early diastolic septal velocity (medial e′) is preserved or even increased [12,13], 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 [7,13-15]. This mitral annular velocity pattern is relatively specific for CP 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. However, there are limited data on mitral and tricuspid annular velocities in patients with CP and their changes after pericardiectomy [12,16,17]. Furthermore, these publications have not addressed the degree and timing of reduction of these annular velocities and their relationship with the patient's symptomatic status following surgery.

This prospective non-randomized study aims to:

a. Serially evaluate the immediate and late effects of total pericardiectomy on the clinical outcome and left ventricular size and function.

b. Serially assess the effect of total pericardiectomy on mitral and tricuspid diastolic filling velocities and their respiratory variation.

c. Serially assess the effect of pericardiectomy on mitral and tricuspid lateral and medial systolic and diastolic annular velocities.

d. analyze the relationship if any of the mitral and tricuspid annular velocities with global myocardial function before and after total pericardiectomy and,

e. Analyze the relationship of mitral and tricuspid annular velocities with patient's symptomatic status in the pre- and postoperative period.

PATIENTS AND METHODS

Patients were enrolled for this prospective study following institutional ethics committee approval and informed written consent from patients/guardians. Between June 2013 and December 2016, 54 consecutive patients (41 males) undergoing pericardiectomy for chronic constrictive pericarditis at All India Institute of Medical Sciences, New Delhi, operated by a single surgeon (corresponding author) were included in this prospective study. The decision to perform pericardiectomy was based on clinical, echocardiographic, computed tomographic and/or cardiac catheterization criteria. Patients with clinical, operative and pathological features of pericarditis and constriction were included. Patients undergoing creation of pleuropericardial window for pericardial effusion, pericardial biopsy and concomitant pericardiectomy and repair of congenital or acquired heart diseases were excluded. Descriptive characteristics and relevant details are summarized in **Table 1**. Patients age at operation ranged from 7 to 70 years (mean, 31 ± 16.8 years). Duration of symptoms ranged from 8 months to 5 years (mean, 18.4 ± 12.6). Preoperatively, 30 (55.6%) patients and 24 (44.4%) patients were in New York Heart Association (NYHA)-III and IV, respectively. All patients had congestive heart failure as the predominant symptom in the preoperative period. Forty-eight (88.8%) patients had precordial pain, 3 (5.5%) had evidence of cardiac tamponade and atrial fibrillation was found in 26 (48.1%) patients. Ninety-two percent had distended jugular veins, 83% ascites, 79% hepatomegaly, 41% pleural effusion and 17% had pulsus paradoxes.

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

Profile	**Number (%)**
Number of patients	54
Males	41 (76%)
Age in years, mean ± SD (range)	31.0 ± 16.88 (7-70 years)
Age distribution	
7-15 years	7 (12.9%)
16-30 years	21 (38.8%)
31-50 years	18 (33.3%)
>50 years	8 (14.8%)
Body weight (kg), mean ± SD (range)	49.42 ± 15.2 (23-80 kg)
Duration of illness (months), Mean ±SD (range)	18.42 ± 12.6 (8-60)
Preoperative NYHA functional class	
III	30 (55.6%)
IV	24 (44.4%)
Precordial pain	48 (88.8%)
Dyspnoea on exertion	49 (90.7%)
Paroxsysmal nocturnal dyspnea	24 (44.4%)
Orthopnea	14 (25.9%)
Congestive heart failure	54 (100%)
Distended jugular vein in sitting position	50 (92.6%)
Peripheral edema	22 (40.7%)
History of pedal edema, ascites	34 (62.9%)
Pleural effusion	22 (40.7%)
Hepatomegaly	43 (79.6%)
Pericardial knock	18 (33.3%)
Ascites	45 (83.3%)
Pulsus paradoxus	9 (16.6%)
Cardiac tamponade	3 (5.5%)
Renal derangement (serum creatinine ≥ 2 mg/dl)	14 (25.9%)
Hyperbilirubinemia (Serum bilirubin ≥ 2 mg/dl)	19 (35.1%)
Hypoproteinemia (Serum albumin) ≤ 3.5 g/dl	29 (53.7%)
Pericardial calcification on chest X-ray	20 (37%)

Pulmonary infiltrates	9 (16.6%)	
Tuberculosis on culture or history	40 (74%)	
Atrial fibrillation		
Preoperative	26 (48.1%)	
Postoperative	9 (16.6%)	
Hb g/dl mean ± SD (range)	11.94 ± 1.80 (6-14)	
ESR mm in 1st h mean ± SD (range)	12.82 ± 7.0 (4-42)	
ESR>40 mm	19 (35.1%)	
Mitral regurgitation	8 (14.8%)	
Tricuspid regurgitation	8 (14.8%)	
Pericardial thickness 43 mm	54 (100%)	
Surgical approach		
Median sternotomy	34 (63%)	
Left antero-lateral thoracotomy	20 (37%)	
Operative mortality	Nil	
Late death	Nil	
Low cardiac output (immediate postoperative)	50 (92.6%)	
Mean duration of hospitalization, mean ± SD (range)	11.42 ± 7.58 (5-24)	
Duration of inotropic support (days), mean ± SD (range)	4.0 ± 2.8 (1-14)	
At last follow-up		
Asymptomatic	44 (81.5%)	
Symptomatic	10 (18.5%)	
	Preoperative	**Immediate postoperative**
Right atrial pressure (mm Hg), mean ± SD (range)		
Asymptomatic patients (n=44)	16.72 ± 4.08 (7-26)	9.11 ± 0.96 (7-10)
Symptomatic patients (n=10)	20.60 ± 3.60 (15-25)	13.80 ± 3.17 (11-22)

ESR: Erythrocyte Sedimentation Rate; SD: Standard Deviation

Four out of 54 patients with pericardial effusion required tapping and steroid therapy as appropriate. All patients with tuberculosis (n=40, 74%) received multidrug therapy (isoniazid, rifampicin, ethambutol and pyrazinamide) for 3 months followed by triple-drug therapy for 9 months after operation. Preoperatively, all patients were on digitalis and diuretics.

The etiology was considered tubercular if the histopathology of the excised pericardium showed granulomas, caseation, giant cells (n=34, 62.9%) or if the fluid and debris removed at surgery was positive for acid fast bacilli (n=6, 18.5%). A history of pulmonary and lymph node tuberculosis was present in 10 (18.5%) and 4 (7.4%) patients respectively. Fourteen (26%) patients had pyogenic or effusive-constrictive pericarditis not resolving with pericardiocentesis.

Laboratory investigations showed elevated erythrocyte sedimentation rate (range, 40 to 90 mm at 1 h) in 19 (35.1%), renal dysfunction (serum creatinine>2 mg/dl in 14 (25.9%)) and hyperbilirubinemia in 19 (35.1%) patients. Chest roentgenogram revealed pericardial calcification (n=20, 37%), pleural effusion (n=22, 40.7%) and pulmonary infiltrates (n=9; 16.6%) patients. The calcification was distributed over the anterior and inferior surfaces of the heart in 12 (22.2%) patients and all around the heart like a cocoon in 8 (14.8%) patients. None had mitral annular calcification. Electrocardiogram revealed low voltage QRS complex (n=53, 98.1%), flattening or T-wave inversion (n=49, 90.7%), atrial fibrillation (n=26, 48.1%) and premature ventricular complex (n=8, 14.8%). Twenty of twenty-six (76.9%) with atrial fibrillation were in NYHA class-IV.

Echocardiography revealed pericardial thickness (>4 mm, n=54), inferior vena cava dilatation (n=53), right atrial enlargement (n=53), abnormal septal motion (n=52), >25% increase in mitral inflow velocity with expiration compared with inspiratory phase (n=53), moderate mitral regurgitation (grade 2+, n=8) and moderate tricuspid regurgitation (grade 2, n=8). Preoperative cardiac catheterization was performed in 6 patients. 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 consistent with constrictive pericarditis because of an elevated right atrial pressure, usually with a M- or W-shaped contour, an abnormally high right ventricular end-diastolic pressure with a characteristic dip-plateau diastolic configuration, equalization of end-diastolic pressure in all cardiac chambers and a ratio or right ventricular end-diastolic-to-right ventricular systolic pressure of >0.30.

SURGICAL TECHNIQUES

The surgical approach was based on surgeon preference and remained uniform throughout the study period. However, a left anterolateral thoracotomy was the preferred option in the setting of purulent pericarditis to avoid sternal infection. The median sternotomy approach was preferred in the following cases: (1) Annular constrictive pericarditis, (n=9, 16.6%); (2) Calcific pericardial patch compressing the right atrium and right ventricular outflow tract (n=12, 22.2%); (3) Egg shaped calcified pericardium (n=8, 14.8%) and (4) extra cardiac intrapericardial mass (n=3, 5.5%). One patient required institution of cardiopulmonary bypass to control bleeding from right ventricular outflow tract. The detailed operative steps of pericardiectomy via median sternotomy (n=34) and left anterolateral thoracotomy (n=20) have been addressed in our previous publications [4,5].

In patients with gross ascites, a peritoneal drainage catheter was placed in the peritoneal cavity before surgical incision and was kept on continuous drainage. It was removed after 1 or 2 days in intensive care unit depending upon the drainage amount. Surgical manipulation of the heart during pericardiectomy can make thermo dilution calculation and pulmonary artery pressure monitoring unreliable as monitors and hence was not used in this group of patients.

After sternotomy, the thymus and pleural reflections were mobilized laterally to obtain a wide width of the pericardium. Both pleural cavities were widely opened to remove the pleural fluid and to identify the phrenic pedicles on either side [4].

An I-shaped incision was made in the midline over the pericardium up to the level of the pulmonary artery superiorly and diaphragm inferiorly. The dissection of the pericardium off the heart was done using cautery until the fibrous pericardium along with its serous layer. When it was done properly, there was clear visualization of the epicardial fat and the coronary arteries. Inability to visualize the coronaries indicates that the dissection plane was not deep enough.

The cautery was adjusted between 8-10 mV during the process of dissection to avoid cautery induced ventricular fibrillation. Multiple silk stay sutures were then placed on the cut edges of the incised pericardium. The pericardium was initially divided at the bottom portion close to the diaphragmatic reflection over the right ventricle and the lateral pericardial flap was raised superiorly and laterally. Circumferential patches of calcified pericardium were crushed with a thick hemostat and/or bone cutter and were removed avoiding injury to the underlying vascular structures, coronaries and phrenic nerves. We have not used cavitational ultrasonic surgical aspiration system for removal of calcium or nerve stimulator for identification of the phrenic nerve on any patient in this study.

The pericardium covering ventricles, the great vessels, the venae cava and the right atrium was excised 1 cm anterior to the phrenic nerve on either side. The pericardium over the venae cava and right atrium was resected last. The pericardial and pleural cavities were irrigated with normal

saline.

For anterolateral thoracotomy, patients were positioned in left lateral position with groin exposed and prepared [5]. The left anterolateral thoracotomy was carried out through left fourth intercostal space. After entering the left thoracic cavity, pleural reflection was dissected out from pericardium. Anteriorly, the pericardium was mobilised and adhesions between sternum and pericardium was released. This was followed by two full-length parallel incisions 0.5 cm anterior and posterior to the left phrenic nerve and extended until the level of the pulmonary artery superiorly and the diaphragm inferiorly. Multiple silk stay sutures were placed on the incised edges of the pericardium to achieve adequate exposure. Posteriorly, the pericardium was gently dissected from the posterolateral surface of the left ventricle and left atrial appendage. The posterior pericardium was subsequently divided to facilitate adequate mobilization until the levels of left-sided pulmonary veins and excised. The anterior pericardial flap was held between stay sutures and mobilized from the anterolateral surfaces of right ventricle, right ventricular outflow tract, and pulmonary artery. Using cautery, a new cleavage plane was made to develop between the diaphragm and thickened diaphragmatic pericardium all along its length. The diaphragmatic surface of the right ventricle and the left ventricular apex was completely freed from pericardial adhesions. Subsequently, the entire width of diaphragmatic pericardium was excised in toto.

ECHOCARDIOGRAPHIC STUDIES AND MEASUREMENTS

All patients had comprehensive evaluation with M-mode, two-dimensional (2D) 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 2D 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.

POSTOPERATIVE STUDIES

These included 3-monthly clinical examinations, electrocardiogram and chest radiographs. A minimum of 6 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 6 months. All late echoes have been grouped into one time period (6 months) with a range of no greater than 6 months. Echocardiographic data were measured according to American Society of echocardiographic criteria [20].

Definitions

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.

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 [4]. Constricting layers of the epicardium were removed whenever possible. The atria and venae cava 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.

Constrictive pericarditis was considered to be hemodynamically significant when there were clinical features of constriction with supportive echocardiographic and hemodynamic criteria as outlined earlier. 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 g/dl. Renal dysfunction was defined as serum creatinine >2.0 g/dl.

Low output syndrome was diagnosed if the patient required inotropic support (dopamine (4-10 $\mu g.kg^{-1}.min^{-1}$), dobutamine (5-10 $\mu g.kg^{-1}.min^{-1}$), epinephrine (0.01-0.1 $\mu g.kg^{-1}.min^{-1}$), milrinone (50 µg/kg intravenous bolus followed by 0.375-0.75 $\mu g.kg^{-1}.min^{-1}$)), either isolated or in combination, in the operating room or in the 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 with or without intra-aortic balloon counter pulsation along with afterload reduction with sodium nitroprusside. Patients who received less than 4 $\mu g.kg^{-1}.min^{-1}$ of dopamine to increase renal perfusion were not considered to have low output syndrome.

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 h), low mixed venous oxygen saturation (<50%) and blunt sensorium.

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 χ^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 generalized estimating equation with exchangeable correlation analysis. 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. Fifty 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=50) 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 patients required intraoartic balloon counter pulsation as an additional support. There was marked reduction of filling pressure within 24 h in the majority of patients (n=44) after total pericardiectomy (mean=right atrial pressure (RAP) 16.72 ± 4.0 (7-26) to 9.11 ± 0.96 (7-10); p<0.001) **(Table 1)**. Echocardiographically, diastolic filling characteristics remained abnormal in 19 (35.2%) 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 6-48 months, median 28) and yielded 135.9 patients-years of data with a mean follow-up time of 30.2 ± 10.8 months.

At closing interval, 10 (18.5%) 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=10, 18.5%) and asymptomatic (n=44, 81.5%) 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=10) were in NYHA class IV and were in atrial fibrillation. Thus, 9 (34.6%) of 26 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=10, 18.5%) had significantly higher right atrial pressure in the immediate preoperative period compared to the asymptomatic group (n=44, 81.5%) (Mean RAP=20.6 ± 3.6 (symptomatic) vs. 16.72 ± 4 mm Hg (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=13.80 ± 3.17 (symptomatic) vs. 9.11 ± 0.96 mm Hg (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 **(Tables 2 and 3)**. Tissue Doppler imaging-derived mitral and tricuspid annular velocities failed to predict the postoperative outcome of patients undergoing pericardiectomy **(Tables 2 and 3)**.

Table 2. Clinical, echocardiographic and hemodynamic parameters in the preoperative period in symptomatic and asymptomatic patients following pericardiectomy for constrictive pericarditis (n=54).

Parameters (preoperative)	Symptomatic patients (post-pericardiectomy, n=10) Mean ± SD (range)	Asymptomatic patients (post-pericardiectomy, n=44) Mean ± SD (range)	p value
Preoperative parameters			
Preoperative/intraoperative RA pressure via central venous catheter (mm Hg)	20.60 ± 3.60 (15-25)	16.72 ± 4.08 (7-26)	**p<0.05**
Postoperative RA pressure (mm Hg)	13.80 ± 3.17 (11-22)	9.11 ± 0.96 (7-10)	**p<0.001**
Duration of symptoms (months)	5.17 ± 4.18 (0.5-12)	11.22 ± 15.08 (1-60)	0.14
ESR (mm/h)	12.53 ± 8.88 (4-42)	12.50 ± 5.23 (6-28)	0.99
Serum bilirubin (mg/dl)	1.61 ± 0.79 (0.7-3.3)	1.85 ± 1.05 (0.4-1.2)	0.46
Serum albumin (g/dl)	3.51 ± 0.89 (2.3-5.4)	3.61 ± 0.50 (3.0-4.6)	0.69
ICU stay (days)	3.80 ± 2.33 (1-9)	4.06 ± 2.84 (2-14)	0.78
Hospital stays (days)	12.27 ± 9.75 (5-40)	10.17 ± 5.27 (5-25)	0.44
MVE (cm/s)	80.83 ± 8.14 (66-90.3)	77.92 ± 19.82 (48-132)	0.71
MVA (cm/s)	38.01 ± 6.10 (30-48.4)	42.06 ± 12.53 (21-78.9)	0.42
MVE/A	2.12 ± 0.18 (1.8-2.38)	1.93 ± 0.42 (1.3-3.1)	0.26
MVE respiratory variation (%)	33.71 ± 14.08 (22-64)	28.69 ± 6. 6 (20-46)	0.18
DT (ms)	95.57 ± 13.65 (80-120)	98.81 ± 18.27 (50-127)	0.67
TVE (cm/s)	64.70 ± 19.14 (42-94.3)	72.36 ± 18.20 (46-109)	0.33
TVA (cm/s)	30.40 ± 7.10 (20.8-43.4)	37.3 ± 7.45 (28-56)	**p<0.05**
TVE/A	2.11 ± 0.28 (1.8-2.67)	1.953 ± 0.34 (1.4-2.66)	0.220
TVE respiratory variation (%)	26.43 ± 4.28 (21-34)	31.65 ± 5.73 (24-43)	**p<0.05**
Indexed IVC diameter (mm/mm^2)	14.65 ± 2.63 (11.40-20)	12.63 ± 2.20 (7.5-15.7)	p<0.05
Mitral lateral e' (cm/s)	16.01 ± 7.43 (5.30-26.1)	14.60 ± 4.67 (6.52-22.2)	0.54
Medial e' (cm/s)	16.19 ± 2.77 (11.90-21.2)	13.75 ± 4.30 (6.4-22.8)	0.07
Tricuspid lateral e′ (cm/s)	11.28 ± 4.56 (6.1-23.3)	12.11 ± 3.59 (5.4-23.3)	0.56
Mitral lateral s′ (cm/s)	9.01 ± 1.08 (6.83-11)	8.48 ± 1.78 (6.5-12.7)	0.33
Medial s′ (cm/s)	7.90 ± 0.88 (6.45-9.52)	7.66 ± 1.81 (5.07-11)	0.63
Tricuspid lateral s′ (cm/s)	8.87 ± 1.41 (6.63-11.3)	9.33 ± 2.34 (5.8-13.3)	0.51
LVIS (mm)	26.53 ± 3.56 (21-36)	27.06 ± 7.03 (17-39)	0.79
LVID (mm)	38.33 ± 4.06 (33-47)	39.56 ± 8.02 (26-53)	0.59
LVEF (%)	56.43 ± 5.56 (50-60)	58.12 ± 3.73 (45-65)	0.66

DT: Deceleration Time; ESR: Erythrocyte Sedimentation Rate; ICU: Intensive Care Unit; IVC: Inferior Vena Cava; 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; RA: Right Atrium; SD: Standard Deviation; TVA: Transtricuspid Late Diastolic Filling Velocity; TVE: Transtricuspid Early Diastolic Filling Velocity
p<0.05=significant

Table 3. Clinical, echocardiographic and hemodynamic parameters in symptomatic and asymptomatic patients at 6 months after pericardiectomy for constrictive pericarditis (n=54).

Parameters (6 months postoperative)	Symptomatic patients (post-pericardiectomy, n=10) Mean ± SD (range)	Asymptomatic patients (post-pericardiectomy, n=44) Mean ± SD (range)	p value
MVE (cm/s)	111.74 ± 22.38 (83-137)	92.78 ± 16.69 (62.7-134)	**p<0.05**
MVA (cm/s)	58.51 ± 28.09 (35-101)	56.69 ± 13.17 (36-83.8)	0.80
MVE/A	2.12 ± 0.64 (1.34-2.90)	1.70 ± 0.39 (1.07-2.50)	**p<0.05**
MVE respiratory variation (%)	12.14 ± 3.89 (6-18)	13.92 ± 4.22 (8-27)	0.32
DT (ms)	100.71 ± 18.48 (78-126)	109.58 ± 24.74 (75-190)	0.38
TVE (cm/s)	68.54 ± 10.60 (50-82)	68.73 ± 20.70 (33.3-113)	0.98
TVA (cm/s)	41.91 ± 11.45 (30.7-59.8)	43.95 ± 13.63 (16.7-78)	0.72
TVE/A	1.77 ± 0.42 (1.06-2.3)	1.56 ± 0.39 (1.00-2.5)	0.23
TVE respiratory variation (%)	18.43 ± 1.62 (16-20)	18.19 ± 3.58 (11-24)	0.87
Mitral lateral e' (cm/s)	14.50 ± 6.37 (4.78-22.40)	10.93 ± 3.69 (3.7-18.3)	0.06
Medial e' (cm/s)	11.61 ± 2.29 (6.8-13.8)	9.36 ± 2.84 (5.3-16.2)	0.06
Tricuspid lateral e′(cm/s)	9.09 ± 2.92 (3.5-12.5)	10.46 ± 5.13 (3.4-27.70)	0.50
Mitral lateral s′ (cm/s)	7.38 ± 1.41 (5.36-8.97)	6.99 ± 1.57 (4.97-9.94)	0.45
Medial s′ (cm/s)	6.61 ± 1.13 (5.40-8.19)	6.11 ± 1.08 (4.26-8.97)	0.29
Tricuspid lateral s′ (cm/s)	7.07 ± 1.60 (4.39-9.78)	7.19 ± 1.89 (4.6-12)	0.88
Indexed IVC diameter (mm/mm^2)	11.54 ± 1.93 (9.09-14.37)	8.28 ± 2.57 (4.61-14)	**p<0.05**
LVIS	27.67 ± 4.75 (21-36)	25.61 ± 5.03 (17-37)	0.24
LVID	40.27 ± 4.77 (33-48)	36.39 ± 5.66 (26-44)	**p<0.05**
LVEF (%)	57.14 ± 3.93 (50-60)	57.50 ± 4.45 (45-65)	0.85

DT: Deceleration Time; IVC: Inferior Vena Cava; 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; TVA: Transtricuspid Late Diastolic Filling Velocity; TVE: Transtricuspid Early Diastolic Filling Velocity
p<0.05=significant

Data analyses and study interpretation of echocardiographic data (Tables 4 and 5 and Figures 1-3)

To assess the characterization of the mitral and tricuspid annular velocity changes in patients undergoing pericardiectomy for constructive pericarditis, generalized estimating equation analysis revealed the following results:

1. There was statistically significant reduction in indexed IVC diameter in the immediate (p<0.001) as well as late postoperative period (p<0.001). The indexed IVC diameter decreased from a preoperative value of 13.55 ± 2.58 mm/m^2 to 9.27 ± 2.47 mm/m^2 (at discharge) and 8.97 ± 2.78 mm/m^2 (at 6 months follow-up).
2. Doppler flow velocity envelopes revealed statistically significant improvement of both transmitral early diastolic and late diastolic filling velocities in the

immediate as well as late postoperative period. As a result, the mitral valve E/A also improved from 1.98 ± 0.39 (preoperative) to 1.79 ± 0.45; p<0.05 (immediate postoperative) and 1.79 ± 0.47; p<0.05 (late postoperative).

3. There was insignificant change in trans-tricuspid early diastolic filling velocity; however, there was significant improvement of trans-tricuspid late diastolic filling velocity secondary to atrial contraction. Overall, the tricuspid valve E/A improved from a preoperative level of 1.97 ± 0.33 to 1.61 ± 0.35 (p<0.001) at discharge and 1.60 ± 0.40 (p<0.001) in the late postoperative period.

4. All patients demonstrated the classic phenomenon of "annulus reversus" of mitral valve velocities. Following pericardiectomy, in the immediate postoperative period, there was no statistically significant improvement of mitral lateral e′ velocity; however there was statistically significant improvement of mitral lateral e′ velocity at 6 months following pericardiectomy (p=0.001). All patients demonstrated greater significant reduction of medial e′ velocity following pericardiectomy in both immediate and late postoperative period.

5. The lateral and medial e′ velocity of the tricuspid valve also exhibited similar phenomenon. Both medial and lateral tricuspid annular velocities exhibited statistically significant decrease in the late postoperative period and only medial tricuspid annular velocity exhibited significant decrease in the immediate postoperative period.

6. Preoperatively, all patients exhibited an inspiratory decrease in peak transmitral flow (mean E>29.76 ± 8.69%) and an increased transtricuspid flow (mean E>30.55% ± 7.81%).

7. Following pericardiectomy, all patients demonstrated statistically significant reduction in mitral systolic annular velocity (lateral and medial) in both early and late postoperative period (8.72 ± 1.5 cm/s (preoperative) vs. 7.94 ± 1.82 cm/s (immediate postoperative); p=0.001 and 7.08 ± 1.20 cm/s (late postoperative); p<0.001; systolic medial mitral annular velocity (mitral medial s′) 7.77 ± 1.45 cm/s (preoperative) vs. 7.15 ± 1.45 cm/s (early postoperative) and 6.22 ± 1.09; p<0.0001). The correlation between mitral s′ and LVEF was statistically insignificant.

8. Similarly, following pericardiectomy, all patients demonstrated statistically significant reduction in tricuspid lateral annular systolic velocity in both early and late postoperative period [tricuspid s′ (cm/s) 9.12 ± 1.96 (preoperative) vs. 8.20 ± 1.73 (early postoperative); p<0.05 vs. 7.16 ± 1.80 cm/s (late postoperative); p<0.001).

9. The early postoperative left ventricular end-diastolic internal diameter (LVID), left ventricular end-systolic internal diameter (LVIS) and LVEF remained almost same as compared to preoperative measurements. There were no significant changes of the above variables in late postoperative period.

10. Overall, the degree of changes of Doppler and TDI-derived variables was maximal in the immediate postoperative period.

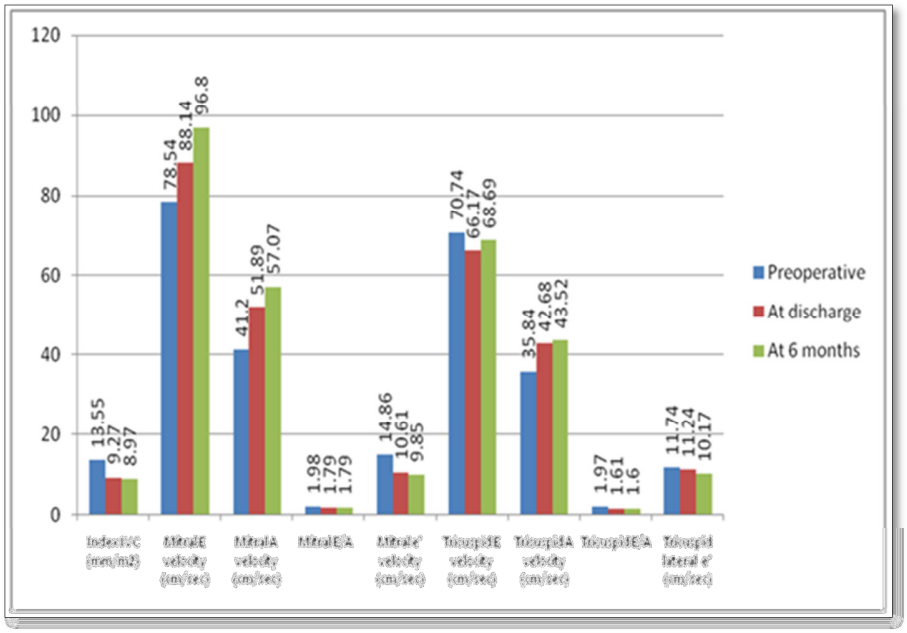

Figure 1. Bar graph showing echocardiographic variables before and after pericardiectomy of all 54 patients in this study. *IVC: Inferior Vena Cava*

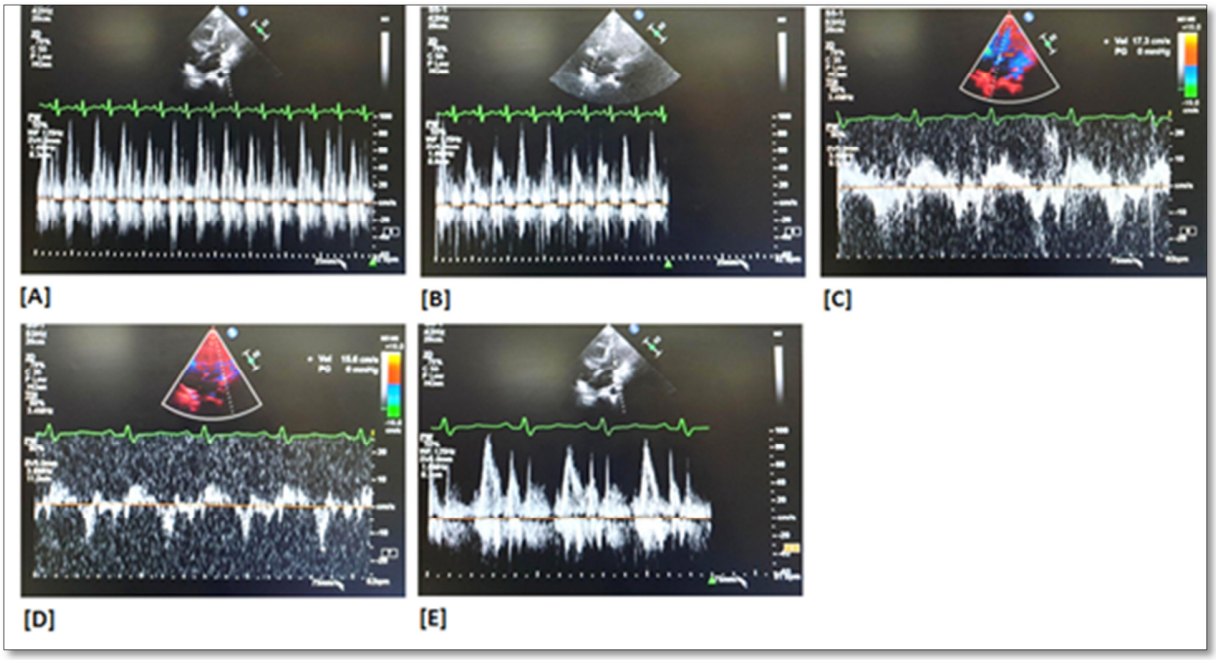

Figure 2. Preoperative echo images in a patient with chronic constrictive pericarditis. **A.** Pulse wave Doppler signals at the mitral valve showing increased respiratory variations. **B.** Pulse wave Doppler signals at the tricuspid valve showing increased respiratory variations. **C&D.** Doppler signals 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/a >1.5.

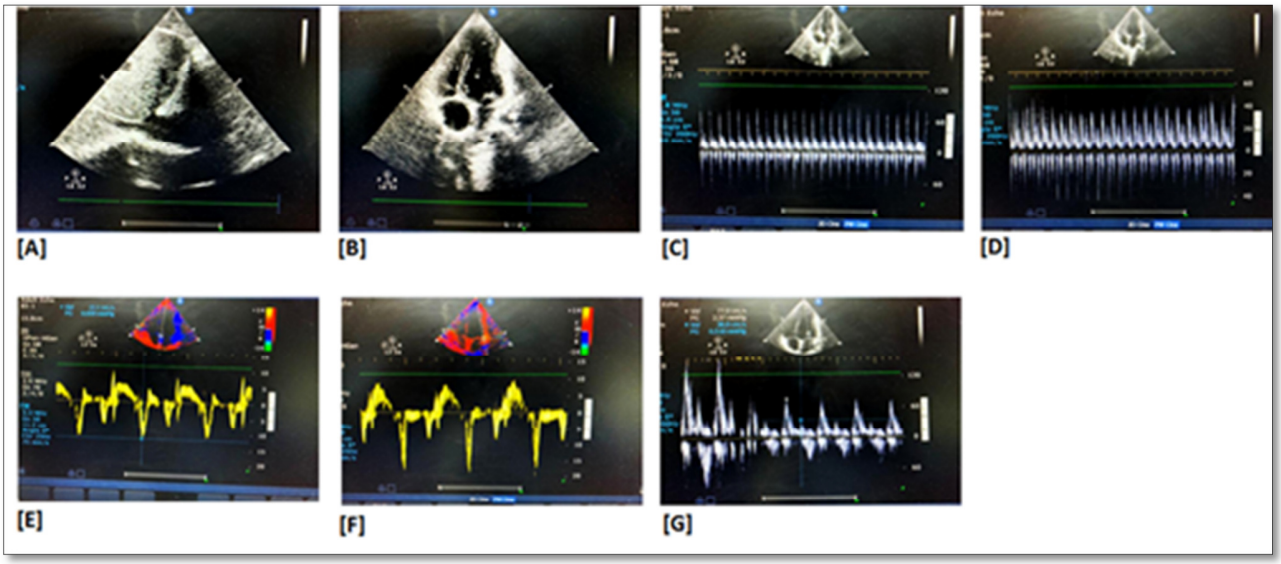

Figure 3. Postoperative echo images of the same patient with chronic constrictive pericarditis showing: **A.** Normal sized IVC. **B.** Apical 4-chamber view (2D image) normal chamber geometry. **C.** Pulse wave Doppler signals at the mitral valve showing normal respiratory variations. **D.** Pulse wave 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.

Table 4. Two dimensional Doppler echocardiographic and tissue Doppler imaging-derived annular velocities before and after pericardiectomy of all 54 patients in this study.

Variables	Pre-operative (n=54) Mean ± SD (range)	At discharge (n=54) Mean ± SD (range)	At 6 months (n=54) Mean ± SD (range)
IVC (mm)	19.63 ± 5.04 (12-33)	13.182 ± 3.9801 **p<0.001**	12.89 ± 4.42 **p<0.001**
INDEX IVC (mm/mm²)	13.55 ± 2.58 (7.5-20)	9.27 ± 2.47 **p<0.001**	8.97 ± 2.78 **p<0.001**
MVE (cm/s)	78.54 ± 17.91 (48-132)	88.14 ± 19.84 **p<0.05**	96.803 ± 19.33 **p<0.001**
MVA (cm/s)	41.20 ± 11.50 (21-78.9)	51.89 ± 16.09 **p<0.001**	57.076 ± 16.85 **p<0.001**
MVE/A	1.98 ± 0.39 (1.3-3.1)	1.79 ± 0.45 **p<0.05**	1.79 ± 0.47 **p<0.05**
DT (ms)	98.12 ± 17.24 (50-127)	108.21 ± 15.15 **p<0.05**	107.70 ± 23.57 **p < 0.05**
MVE respiratory variation (%)	29.76 ± 8.69 (20-64)	14.97 ± 6.14 **p<0.001**	13.55 ± 4.16 **p<0.001**
TVE (cm/s)	70.74 ± 18.38 (42-109)	66.17 ± 17.48 **p=0.176**	68.69 ± 18.87 **p=0.607**
TVA (cm/s)	35.84 ± 7.81 (20.8-56)	42.68 ± 13.14 **p=0.001**	43.52 ± 13.06 **p=0.001**
TVE/A	1.97 ± 0.33 (1.4-2.7)	1.61 ± 0.35 **p=<0.001**	1.60 ± 0.40 **p=<0.001**
TVE Respiratory variation (%)	30.55 ± 5.81 (21-43)	21.85 ± 7.82 **p=<0.001**	17.33 ± 4.22 **p<0.001**
Mitral lateral e′ (cm/s)	14.07 ± 5.27 (5.3-26.1)	14.06 ± 5.24 **p=0.355**	11.69 ± 4.52 **p=<0.001**
Medial e′ (cm/s)	14.86 ± 3.84 (6.4-22.8)	10.61 ± 2.98 **p<0.001**	9.85 ± 2.86 **p=<0.001**
Tricuspid lateral e′ (cm/s)	11.74 ± 4.01 (5.4-23.3)	11.24 ± 3.96 p=0.474	10.17 ± 4.74 p<0.05
Mitral Lateral s′ (cm/s)	8.72 ± 1.51 (6.5-12.7)	7.94 ± 1.82 **p=0.001**	7.08 ± 1.20 **p<0.001**
Medial s′ (cm/s)	7.77 ± 1.45 (5.07-11)	7.15 ± 1.45	6.22 ± 1.09

		p<0.05	**p<0.001**
Tricuspid lateral s′ (cm/s)	9.12 ± 1.96 (5.8-13.30)	8.20 ± 1.73 **p<0.05**	7.16 ± 1.80 **p<0.001**
Mitral E/e'	6.29 ± 3.72 (2.90-16.20)	7.12 ± 3.54 p=0.11	9.75 ± 5.09 **p<0.001**
Tricuspid E/e'	6.88 ± 3.30 (2.94-15.46)	6.81 ± 3.39 p=0.90	8.13 ± 4.58 p=0.17
IVS (mm)	7.61 ± 1.54 (5-11)	7.18 ± 1.42 p=0.193	7.49 ± 1.30 p=0.757
PW (mm)	7.85 ± 1.66 (5-12)	7.52 ± 1.23 p=0.252	7.76 ± 1.00 p=0.801
LVIS (mm)	26.82 ± 5.65 (17-39)	26.79 ± 4.54 p=0.958	26.55 ± 4.94 p=0.846
LVID (mm)	39.00 ± 6.47 (26-53)	38.97 ± 6.04 p=0.959	38.15 ± 5.55 p=0.558
LVEF (%)	57.76 ± 4.15 (45-65)	56.36 ± 4.662 p=0.198	57.42 ± 4.28 p=0.748
Aorta (mm)	24.03 ± 4.54 (15-32)	23.91 ± 4.34 p=0.563	24.18 ± 4.647 p=0.868
Left Atrium (mm)	33.27 ± 8.94 (17-55)	31.42 ± 6.85 p=0.181	33.85 ± 9.414 p=0.786

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 5. Relationship between mitral annular systolic velocity and left ventricular ejection fraction of all 54 patients in this study.

Mitral annular systolic velocity	**Left ventricular ejection fraction**					
	Preoperative (n=54)		**At discharge (n=54)**		**At 6 month (n=54)**	
	r value	**p value**	**r value**	**p value**	**r value**	**p value**
Lateral s′	-0.12	0.48	-0.34	0.06	-0.06	0.73
Medial s′	-0.2	0.23	-0.16	0.38	-0.31	0.08

r: Spearman's rank correlation coefficient, p<0.05=significant

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 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 **(Tables 2-4)**; and
9. Preoperative atrial fibrillation was a predictor of poor prognostic outcome following pericardiectomy.

Tissue Doppler imaging has made possible the acquisition of myocardial wall velocities and 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 [7,8,11-17]. Published data on the effect of pericardiectomy on mitral and tricuspid annular velocities are limited because of limited number of patients and restricted observations [12,16,17]. During systole, the mitral annulus descends towards the apex, with no appreciable motion of the apex in relation to the imaging transducer. Therefore, the annular displacement reflects the extent of myocardial fiber shortening in the longitudinal plane and has a strong linear correlation with global left ventricular function [21]. Since the mechanoelastic properties of the myocardium are preserved in CP, the longitudinal mitral annular velocities remain normal or can be exaggerated as lateral expansion in constrictive pericarditis is limited [12,13,17].

Previous investigators have evaluated the role of tissue Doppler imaging in the diagnosis of CP in patients without diagnostic respiratory variation of transmitral early diastolic filling velocity. They concluded that in patients with preserved mitral e′ velocity (>8 cm/s) and a low E/e′ ratio (<8) with high LV filling pressure, the recognition of "annulus reversus" should alert to the diagnosis of CP [7-9,12-14,22]. Building on the above-mentioned observations, we attempted to evaluate tissue Doppler imaging at mitral and tricuspid annuli in patients undergoing pericardiectomy and identify the relationship if any of the TDI-derived variables with patient's symptomatic status following surgery.

Early diastolic mitral annulus velocity

We confirmed the presence of "annulus reversus" in all patients with CP in the preoperative period. Following pericardiectomy, it may be anticipated that the medial mitral annular velocity decreases and the lateral annular velocity increases, resulting in normalization of lateral/medial e′ ratio. In this study, while the latter was confirmed, both medial and lateral e′ velocities were found to decrease after pericardiectomy and there was no reversal **(Table 4)**.

Veress et al. [18] had similar observations in their study and described the following mechanisms for their observations: Pericardiectomy removes constraint to lateral mitral annular expansion and nullifies the exaggerated longitudinal mitral annular motion as well as the translateral component of lateral e′ velocity related to increased medial excursion.

Early diastolic tricuspid annulus velocity

The phenomenon of "annulus reversus" of the tricuspid valve 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 **(Table 4)**. Therefore, the above mentioned mechanisms operative at the mitral annulus may as well be responsible for findings at the tricuspid annulus.

In this study, mild mitral and tricuspid regurgitation was present in 8 (14.8%) patients. Both of them responded favorably to pericardiectomy and postoperative conservative management. The frequent association of CP with significant tricuspid regurgitation and worsening of tricuspid regurgitation following pericardiectomy in a subset of

patients in the published literature are noteworthy [24].

Systolic annulus velocity

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 and right ventricular systolic function than 2D or M-mode imaging. It was measured via an apical four chamber view at systole (s′) with a 2-5 mm tissue Doppler sample volume placed at the septal corner and at the mitral and tricuspid lateral annuli. s′ has been correlated with peak positive dP/dt and LVEF in patients with dilated cardiomyopathy, and myocardial infarction [25,26]. There is little information on mitral and tricuspid s′ velocities in patients with CP [18,27,28]. In this study, the correlation between mitral s′ and LVEF was statistically insignificant **(Table 4)**.

The mean s′ velocity in all patients in this study was lower both before and after pericardiectomy than published normative values [29] and also lower, especially pre-pericardiectomy **(Table 4)**. These observations are consistent with previous smaller studies [18,27]. This finding contradicts the theoretical basis since velocity is expected to increase with augmented stroke volume after pericardiectomy.

It is 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 other investigators demonstrating 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 [18].

There appeared to be proportionately greater postoperative reduction in tricuspid lateral or right ventricle s′ and e′ compared to mitral annulus values. Asymmetric distribution of the diseased pericardium predominantly over the RV may well be responsible for the above observations. However, the disproportionate reduction in tricuspid lateral s′ and e′ probably seems also from postoperative RV dysfunction, which was moderate in 10 (18.5%) patients.

Left ventricular ejection fraction did not change despite the expected increase in stroke volume after pericardiectomy. It is postulated that after pericardial resection, LV filling increases and other elements of LV shortening including torsion are recruitable, contributing to better cardiac output and compensating for abnormal longitudinal function [18]. Sengupta et al. [30] 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. To confirm this hypothesis, detailed analysis of myocardial mechanics in a larger number of patients pre- and post-pericardiectomy will be required.

Monitoring of intracardiac pressures during pericardiectomy has been proposed to evaluate the result of decortications but Viola [31] 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. Secondly, 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.

It has been shown that diastolic filling characteristics remain abnormal in a substantial number of patients with CP; even after successful pericardiectomy, these abnormalities may resolve gradually. Moreover, diastolic filling abnormalities after pericardiectomy correlate well with clinical symptoms and tend to occur in patients who had long- standing preoperative symptoms [1-4,6,32].

In our study group, 10 (18.5%) patients continued to have NYHA Class II symptoms late postoperatively. However, none of them had raised jugular venous pulsation, hepatomegaly or ascites. These patients exhibited higher RA pressure measured via central venous catheter, increased indexed IVC diameter, higher LVID and persistently abnormal transmitral early diastolic filling velocity in the postoperative period, as compared to the asymptomatic patients (**Tables 2-4**). During surgery, these 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. 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.

It is pertinent to state that 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. Therefore, the TDI-derived mitral and tricuspid annular velocities failed to predict the symptomatic status of patients undergoing pericardiectomy. It is also worthwhile to mention that 9 out of 10 patients who were symptomatic in the postoperative period continued to remain in atrial fibrillation. Therefore, the presence of atrial fibrillation in the preoperative period may be a predictor of poor prognostic outcome following pericardiectomy. The utility of tissue Doppler imaging in

identifying residual constrictive pericarditis requires further investigation on a large cohort of patients correlating the clinical outcomes.

STUDY LIMITATIONS

Majority of patients in this study underwent total pericardiectomy via median sternotomy. Hence, the tissue Doppler imaging variables could not be compared with anterolateral thoracotomy approach. The small number of postoperative symptomatic patients in this study is an additional limitation.

Secondly, heart performs complex rotational and translational movement inside the chest, thus distorting the measurements of myocardial velocities. In this study, 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. Studies are underway to analyze radial and circumferential function for a better understanding of the mechanics of the unique annular motion in constrictive pericarditis and effects of pericardiectomy.

CONCLUSION

This study demonstrates that patients with congestive heart failure and normal LVEF, preserved or increased mitral medial e′ velocity with "annulus reversus" is diagnostic of constrictive pericarditis. This characteristic pattern of annular velocities return to normal after pericardiectomy. The extent of postoperative changes is maximal in the immediate postoperative period. Tissue Doppler imaging-derived mitral and tricuspid annular velocities cannot predict the postoperative outcome of patients undergoing pericardiectomy. Tissue Doppler imaging is a useful investigative modality for diagnosis of constrictive pericarditis and not a useful indicator of postoperative evaluation.

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