(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 5, Issue 6 , Page No: 540-555
November-December 2022

IJMSCR

A Study On Hemodynamic And Tissue Doppler Correlates In Constrictive Pericarditis

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Type of Publication: Original Research Paper
Conflicts of Interest: Nil

Abstract

Background: In constrictive pericarditis no physical sign or procedure is diagnostic. A constellation of clinical features along with chest radiography, echocardiography, CT scan, and cardiac catheterization is often necessary for confirming the diagnosis. Since constrictive pericarditis manifests mainly as an altered physiological state, the use of advanced methods of echocardiography such as Tissue Doppler Imaging (TDI) may help in assessing and even serve as a diagnostic modality and also assess the severity of the disease in constrictive pericarditis.

Aim Of The Study: In constrictive pericarditis no physical sign or procedure is diagnostic. A constellation of clinical features along with chest radiography, echocardiography, CT scan, and cardiac catheterization is often necessary for confirming the diagnosis.

Methods: This Single-center, nonrandomized, observational study was conducted in the department of cardiology, government Mohan Kumar Mangalam medical college, Salem, Tamil Nadu, India in the year 2020-2021. The patient population consisted of patients with the clinical diagnosis of constrictive pericarditis who subsequently had surgical confirmation. The research proposal was approved by the institutional review board of the ethical committee for clinical research.12 patients all prospectively underwent a rigorously detailed clinical evaluation, biochemical investigation, chest radiography, Computed Tomography (CT) scan of the chest, and Echocardiography including M-Mode, 2D Echo, Tissue Doppler imaging (TDI) and Transesophageal Echocardiography (TEE). The patients subsequently underwent a detailed hemodynamic cardiac catheterization study before confirmation by surgical treatment and pathological confirmation.

Results: Pericardial calcification was present in two patients, (16.6%) and appears as thick shaggy calcification of pericardium over the Right ventricle, diaphragmatic surface, and apex of LV. Calcification was best seen in lateral view.Pleural effusion was present in 10 patients (83%).It was bilateral in one patient (8.3%) with right more than left. The remaining patients had unilateral pleural effusion with left-sided effusion in 5 patients (41.6%) and right-sided pleural effusion in 4 patients (33.3%) The mean value of Tricuspid early diastolic peak velocity (E) was 58.4cm/s (range:30.8 to 86.6) during inspiration and 41.6 cm/s (range 23.6 to 63.7) during expiration. Respiratory variation in the Tricuspid early diastolic peak velocity (E) of >40% increase during inspiration compared to expiration was present in 8 patients (66.6%). The variation ranged from 13% to 62.5%. The mean value of peak late diastolic mitral annular velocity (A') was 6.37 cm/s.The mean value of peak systolic mitral annular velocity (S') was 7.2 cm/s (normal range: 7 to 7.5 cm/s) and was suggestive of normal LV systolic function. The mean value of the ratio (E/E' ratio) between peak early diastolic mitral inflow

velocity (E) and peak early diastolic mitral annular velocity (E') was 5.85 (range: 3.29 to 11.17). The mean value of peak early diastolic Tricuspid annular velocity (E') was 13.49 cm/s. The mean value of peak late diastolic Tricuspid annular velocity (A') was 7.9 cm/s.The mean value of peak systolic Tricuspid annular velocity (S') is 9.36 cm/s normal value of S' > 11.5 cm/s). The mean value of the ratio (E/E' ratio) between peak early diastolic Tricuspid inflow velocity (E) and peak early diastolic Tricuspid annular velocity (E') was 4.71 (range: 2.83 to 7.5**).**

Conclusion: Patients with clinically suspected constrictive pericarditis who have classic findings on Doppler echocardiography can now undergo pericardiectomy without the need for cardiac catheterization. Only the patients in whom there have been equivocal findings either on clinical presentation or on noninvasive testing then undergo further hemodynamic assessment. Echocardiography has replaced invasive cardiac catheterization for various hemodynamic assessments. Of all of the hemodynamic measurements used in daily clinical practice, LV filling pressure is one of the most frequently used. With the advent of TDI and other recent developments in the field of echocardiography, it has become a truly versatile and reliable hemodynamic imaging tool. And, echocardiography has become a noninvasive Swan-Ganz catheter.

Keywords: Hemodynamic, Tissue Doppler, Correlatlation with Constrictive Pericarditis

Introduction

With the advent of newer imaging tools, there has been a shift to noninvasive imaging particularly when chronic constrictive pericarditis (CCP) is suspected. Cardiac catheterization nonetheless remains diagnostic of the constriction physiology especially when anatomic information from other imaging tools is inconclusive. [1] In this hemodynamic review we shall briefly discuss hemodynamic alterations seen in patients with pericardial constriction and how they are different compared to other pericardial compression syndromes and restrictive cardiomyopathy (RCMP) proximal-most great arteries and caval veins are intrapericardial, except for the left atrium (LA).[2] Pericardial cavity, a potential space between two layers of the pericardium can have 15e35 ml of serous fluid. As a result of relatively inelastic physical properties, the pericardium limits acute dilatation and enhances mechanical interactions of the cardiac chambers.4 Accumulation of a large amount of fluid especially if rapid or thickening of the pericardium results in pericardial compression syndrome characterized by increased pericardial pressure. When both fluid and pericardial constriction exist together it is referred to as effusive constrictive pericarditis.[3] In constrictive pericarditis the easily distensible, thin, parietal, and visceral pericardium linings become inflamed, thickened, and fused. Because of these changes, the potential space between the linings is obliterated. Venous return to the heart becomes limited and ventricular filling is reduced. [4] One of the distinguishing characteristics of this disease is the equally elevated left and right ventricular end-diastolic pressures. Symptoms consistent with congestive heart failure (CHF), especially right-sided heart failure, develop as a result of the inability of the heart to increase stroke volume. [5] Cardiac output gradually becomes inadequate, at first with exercise and then at rest. Systolic function is rarely affected until late in the course of the disease, presumably secondary to infiltrative processes that affect the myocardium, atrophy, or scarring/ fibrosis of the myocardium from the overlying adjacent pericardial disease. The hemodynamic study for suspected pericardial pathology should be carefully planned. Attention must be paid to the fluid status to avoid evaluation during low volume, low output state. Patients can be mildly sedated but should remain awake during the study to simulate hemodynamic changes at rest. Oxygen supplementation should be limited to patients with hypoxia and should be started after obtaining samples for oximetry to calculate cardiac output.[6] Irregular rhythm (atrial fibrillation, frequent ventricular ectopics) during the hemodynamic study is an important source of error. Temporary pacing if used in such circumstances can improve diagnostic accuracy.[7] The importance of accurate calibration and positioning of the pressure transducer system cannot be overemphasized. Although high-fidelity manometer-tip catheters are

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more accurate, fluid-filled catheters are generally sufficient in clinical practice. It is also important to analyze appropriate beats for proper assessment of respiratory variation in ventricular pressures. Peak inspiratory and expiratory beats are identified by the lowest and highest diastolic nadir in LV and pulmonary capillary wedge pressure (PCWP) respectively.[8]

Methods

his Single-center, non-randomized, observational study was conducted in the department of cardiology, government Mohan Kumar Mangalam medical college, Salem, Tamil Nadu, India in the year 2020-2021. The patient population consisted of patients with the clinical diagnosis of constrictive pericarditis who subsequently had surgical confirmation. The research proposal was approved by the institutional review board of the ethical committee for clinical research.12 patients all prospectively underwent a rigorously detailed clinical evaluation, biochemical investigation, chest radiography, Computed Tomography (CT) scan of the chest, and Echocardiography including M-Mode, 2D Echo, Tissue Doppler imaging (TDI) and Transesophageal Echocardiography (TEE). The patients subsequently underwent a detailed hemodynamic cardiac catheterization study before confirmation by surgical treatment and pathological confirmation.

Inclusion criteria: All patients with the clinical and Echocardiographic features consistent with the diagnosis of constrictive pericarditis.

Exclusion Criteria: Patients with preexisting severe co-morbid conditions may preclude invasive evaluation and surgical treatment. Patients with the Echocardiographic features of thickened pericardium but without clinical and Doppler features of constrictive pericarditis.Patients with associated Rheumatic Heart Disease. All the patients were screened fluoroscopically for pericardial calcification in PA, Lateral RAO, and LAO views. Cardiac catheterization was performed with the patient in the fasting state. A femoral venous and arterial access site was used in all patients. All patients received 2500 U heparin IV at the start of the procedure. Right-sided heart pressure waveforms obtained with a 6F Cournand catheter advanced retrogradely into the Right ventricle. Left-sided heart pressure waveforms obtained with a 6F pigtail catheter advanced retrogradely into the left ventricle the same calibration procedure was performed using simultaneous and equisensitive transducers. Right-sided heart catheterization was performed and the catheter was advanced into the pulmonary tree until a pulmonary wedge contour was observed. Confirmation of the wedge position was obtained with an oxygen saturation >95%. For measurement of right-sided heart pressures in all chambers, the right-sided heart catheter was pulled back sequentially into the pulmonary artery, right ventricle, and mid-right atrium. Patients were instructed to inspire deeply during dynamic respiratory measurements. All pressure recordings and tracings were recorded at 25, 50, and 100 mm/s for at least 1 minute at each speed during normal respirations. Baseline pressure waveforms were measured simultaneously in the right and left sides of the heart at end-expiration, incorporating an average of five consecutive beats. These measurements included the LVSP, RVSP, LVEDP, and RVEDP, mean PCWP, mean pulmonary artery pressure, PASP, MRAP, and mean aortic pressure. Systolic measurements were taken from the peak of the pressure waveform, whereas end-diastolic pressures were measured just before the onset of the ventricular contraction. The height of the RFW was measured from the left ventricular pressure minimum in the early diastole to the mid-diastolic pressure plateau. Analysis of the dynamic respiratory changes in hemodynamics was made from the recordings. To assess whether an abnormal dissociation of intrathoracic and intracardiac pressures was present, the PCWP minus the minimum early LVDP gradient was measured during the inspiratory and expiratory phases of respiration. Inspiration was defined as the first ejection beat that followed the first inspiratory diastolic filling period, and the first expiratory ejection beat was the beat after the first expiratory diastolic filling period. The maximal increase and decrease of the PCWP were also measured during the same respiratory cycle. Evidence for abnormal ventricular interdependence was assessed by analyzing the simultaneous left ventricular and right ventricular waveforms during respiration. The onset of inspiration (beat number one) was defined as the first ejection beat after a decline in early LVDP. Maximum inspiration was defined as the ejection phase of the beat after the diastolic filling phase with the lowest early LVDP. The peak LVSP and RVSP

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were measured for beat number one as well as the following beats throughout one respiratory cycle.

Result

Table 1 Clinical Signs Profile In Patients With Constrictive Pericarditis

Name	JVP cm	JV wave	Kussmaul sign	Pulsus paradoxus	Ascites	Edema	Jaundice	Peri knock	murmurs	Liver	Spleen
1	10	X<Y	+	−	+	+	−	+	−	+	+
2	15	X<Y	−	−	+	+	−	+	−	+	+
3	8	X<Y	+	−	+	−	−	+	−	+	−
4	15	X<Y	+	−	+	−	−	+	−	+	−
5	13	X=Y	+	+	+	+	−	−	−	+	−
6	12	X=Y	+	+	+	−	+	+	+	+	+
7	10	Y	+	−	+	−	−	−	+	+	−
8	15	X<Y	+	−	+	+	+	+	−	+	+
9	12	X<Y	+	−	+	+	−	+	−	+	−
10	14	X<Y	+	+	+	−	−	−	−	+	+
11	11	X<Y	+	−	+	+	−	+	+	+	−
12	12	X<Y	+	−	+	+	−	+	−	+	−

Table :1 The absence of inspiratory decrease or inspiratory increase (in JVP) of Kussmaul‘s sign was present in 10 patients (84%). Pulsus Paradoxus is present in one-fourth of the patients. Tense ascites were present invariably in all patients. Pedal edema was absent in 5 patients (41%). The early diastolic pericardial knock was present in 9 patients (75%) and systolic murmur of mitral regurgitation was present in 3 patients (25%). There was no audible murmur of tricuspid regurgitation or pericardial rub Hepatomegaly was invariable but the spleen was palpable in one-third of the patients.

Table:2 Ecg Findings In Patients With Constrictive Pericarditis

Name	*Rate/bpm*	*Rhythm*	*Notched P wave*	*Low QRS voltage*	*IVCD*	*ST depression*	*T wave inversion*
1	100	Sinus	+	−	−	V4-V6	L II,III,aVF V4-V6
2	112	Sinus	+	+	−	V2-V6	L I,II,III,aVF V4-V6
3	134	Sinus	−	−	−	V2-V6	L II,III,aVF V2-V6

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4	120	Sinus	–	+	–	V4-V6	L II,III,aVF V2-V6
5	100	Sinus	–	+	–	V1-V3	V1-V3
6	130	Sinus	–	+	–	V2-V6	L II,III,aVF V3-V6
7	106	Sinus	+	–	–	V5-V6	L II,III,aVF V4-V6
8	124	Sinus	+	+	–	V2-V6	L II,III,aVF V2-V6
9	120	Sinus	+	+	–	V3-V6	Flat LI,II,V4-V6 ↓V2 –V3
10	110	Sinus	–	–	–	V2-V4	V2-V6
11	75	Sinus	–	–	–	L II, III,aVF V2-V5	L II,III,aVF V2-V5
12	84	Sinus	+	+	–	V5-V6	L I,II,III,aVF V5-V6

Table:2 Abnormal notched P wave was present in 50% of the patients. And low voltage QRS complexes in 7 patients (58.3%). There were no significant conduction abnormalities observed. The ECG was abnormal in all the patients. ST segment in precordial leads and inverted T wave abnormality were present in all the patients. A flat T wave was present in one patient along with other changes.No patient had normal ECG.

Table :3 Chest Radiography Findings In Patients With Constrictive Pericarditis

Name	***CTR%***	***Straight RHB***	***Peri calcific***	***LAE***	***Pleural eff***	***Pulm Venous cong***	***SVC***
1	0.53	+	Diaph surface	–	left	–	–
2	0.58	–	–	–	right	+	+
3	0.50	–	–	–	right	–	+
4	0.50	–	Diaph surface	–	right	+	+
5	0.55	+	–	–	left	+	+
6	0.60	–	–	–	right>left	+	+
7	0.60	–	–	+	–	+	+
8	0.55	+	–	–	–	+	+

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9	0.55	+	–	+	right	+	+
10	0.55	–	–	–	left	–	+
11	0.50	–	–	–	left	–	+
12	0.55	+	–	–	left	+	–

Table :3 Straightening of the right heart border was present in 5 patients (41.6%). Left atrial enlargement was present in two patients (16.6%). Pulmonary venous congestion was present in 8 patients (66.6%). The severity of pulmonary venous congestion was Grades I to II. Frank's pulmonary edema was not seen. Distention of SVC was present in 10 patients(83%). Pericardial calcification was present in two patients, (16.6%) and appears as thick shaggy calcification of pericardium over the Right ventricle, diaphragmatic surface, and apex of LV. Calcification was best seen in lateral view. Pleural effusion was present in 10 patients (83%).It was bilateral in one patient (8.3%) with right more than left. The remaining patients had unilateral pleural effusion with left-sided effusion in 5 patients (41.6%) and right-sided pleural effusion in 4 patients (33.3%).

Table 4 **. CT scan findings in patients with constrictive pericarditis**

Name	*PEF*	*Peri Thickening (mm)*	*Distribution of thickening*	*Peri calcific*	*Tube like chambers*	*I dilation*	*Pleural effusion*
1	–	10	RA/RV>LV	+	RV	+	L
2	+	12	RA/RV>LV	+	RV	+	R>L
3	+	20	RA>LV>RV	–	–	+	R
4	+	16	RA/RV>LV	–	–	–	R
5	++	17	RA>RV>LV	–	–	–	R>L
6	+	18	Global	–	–	+	R>L
7	+	10	Global	–	–	+	–
8	–	15	Global	–	–	+	R
9	–	10	Global	+	–	+	R>L
10	++	25	Global	–	–	+	L>R
11	+	04	LV	–	–	–	R>L
12	–	14	RA/RV>LV	+	RV	+	L

Table 4 Pericardial calcification was present in one-third of the patients. Calcification was over RV and Right AV groove in 3 patients (25%)and extensive involving RV, LV, and Right AV groove in 1 patient. Deformed chambers appearing as the tube-like structure were present in 5 patients (41.6%)- deformed LV 2 patients; deformed RV 3 patients.IVC dilatation was present in 7 cases (58.3%). Pleural effusion was present absent in one patient; bilateral in 6 patients (50%); unilateral (R or L) in 5 patients (41.6%).RA or RV thrombus was not seen in any of the patients.

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Table 5. M-Mode M-Mode Echocardiography

Name	Peri Thickness (mm)	Peri calcific	PEF(mm)	LV Dimension(cm) LVEDD/LVESD/EF%	LVPW flat	Septal notch
1	8.1	+	+	4.5/3.3/53%	+	+
2	14	–	+	4.6/3.4/50%	+	+
3	Ant 11 Post 15	–	Mild	3.7/2.5/62%	+	+
4	6.6	–	–	3.2/2.4/52%	+	+
5	Post 19	–	Ant 10 Post 15	3.4/1.9/65%	+	+
6	11.4	–	++	4.2/3.1/49%	+	+
7	Ant 3.5 Post 12.9	–	+	4.4/3.0/60%	+	+
8	Ant 9.3 Post 23.1	–	–	4.1/2.8/62%	+	+
9	16	–	+	3.0/1.9/69%	+	+
10	39	–	+	3.6/2.8/44%	+	+
11	12	–	–	3.9/2.6/64%	+	+
12	15.7	+	Ant 3 Post 6	3.8/2.6/58%	+	+

Table:6 2D Echocardiography

Nam e	***Atria l dil***	***LA-LV angle***	***Tube Like chamber s***	***PEF***	***Peri calcific***	***Septal bounce***	***IV C Jul***	***Thromb us***	***SEC***
1	–	obtuse	RV	–	+	+	+	–	IVC/ RA
2	–	obtuse	–	–	–	+	+	–	–

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3	–	obtuse	RV	+	–	+	+		
4	+	obtuse	LV	–	–	+	+	RA/RV	IVC/RA/RV
5	–	obtuse	–	+		+	+		
6	–	obtuse	–	++	–	+	+	IVC/ RA/RA A	IVC/RA/RV
7	–	obtuse	–	+	–	+	+	–	–
8	–	obtuse	RV	Min RA/RV	–	+	+	–	
9	–	obtuse	–	RA/RV/LV	R AV groove	+	+	RA	IVC/ RA
10	–	obtuse	–	++	–	+	+	–	IVC/RA
11	–	obtuse	–	–		+	+	–	I
12	–	obtuse	LV	+	+	+	+	–	RA

Table:6Mean size of IVC was 2.08 cm during inspiration (range; 1.55 to 2.53) and 1.8 cm during expiration (range; of 1.43 to 2.26 cm). All patients had <50% respiratory variation of IVC and in 3 patients (25%) IVC showed an absence of respiratory variation.LA- LV angle was abnormal in one patient. Pericardial calcification was evident in 3 patients. The thickened pericardium was seen in 11 patients. Deformed ventricular morphology – tube-like chambers were present in 5 patients (41.3%).with tubes like RV in 3 patients and LV in 2 patients. Pericardial effusion was present in 8 patients (66.6%).

Table 7. Pulmonary Venous Doppler (By Tee)

Name	***Systolic(S) Cm/s***		***Diastolic(D) Cm/s***		***S/D ratio***		***(D) Resp Variation %***	***Atrial Reversal (AR) Cm/s***
	Ins	***Exp***	***Ins***	***Exp***	***Ins***	***Exp***		
1	22.0	26.8	29.7	38.5	0.74	0.69	29.6%	14.5
2	40.7	48.7	38.7	68.2	1.05	0.71	76.2%	24.3
3	23.3	28.5	31.5	38.8	0.73	0.73	23.1%	12.9
4	32.4	41.5	37.4	46.8	0.86	0.88	25.1%	46.2
5	17.7	19.6	32	40.5	0.55	0.48	26%	34.2
6	29.6	38.6	38.4	49.2	0.77	0.78	28.1%	32.5

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7	24.8	30.6	30.8	41.5	0.80	0.73	34%	30.8
8	39.2	46.8	42.5	51.6	0.92	0.90	21.4%	28.5
9	23.1	32.5	31.3	40.7	0.69	0.79	30%	13.0
10	20	29.1	33.0	45.7	0.60	0.63	38.4%	23.1
11	25.4	46.4	41.3	57.6	0.61	0.80	39.4%	17.6
12	32.2	42.0	43.9	48.5	0.73	0.86	10.4%	14.1

Table:7 Hepatic Venous systolic(S) flow analysis showed an increased flow velocity during inspiration than during expiration Inspiration: mean S 54.8 cm/s (range: 19.1 to 38.6). Hepatic Venous Systolic flow reversal (SR) showed expiratory exaggeration with a mean value of 15.9 cm/s (range:8.2 to 29.3) during inspiration and a mean value of 19.2 to cm/s (range:9.5 to34.2) during expiration. Hepatic Venous diastolic (D) flow analysis showed a mean value of 22cm/s (range:10.6 to 0.1). Analysis of Hepatic Venous diastolic flow reversal (DR) analysis during inspiration and expiration revealed an exaggerated expiratory increase of diastolic flow reversal (9.7% increase during expiration). Diastolic flow reversal (DR) during inspiration – mean 21.4 cm/s (range: 13.3 to 32.8). Diastolic flow reversal (DR) during expiration – mean 31.1 cm/s (range: 21.6 to 55.1).

Table 8 Hepatic Vein Doppler

Name	*Systolic(S) cm/s*	*Systolic Reversal(SR) cm/s*		*Diastolic(D) cm/s*	*Diastolic Reversal(DR) cm/s*	
		Insp↑	*Exp↑*		*Insp↑*	*Exp↑*
1	25.7	8.2	9.5	17	18	23.7
2	22.3	11.6	16.8	15.8	19.6	27.5
3	21.9	13.4	15.4	15.1	18.4	25.8
4	36	13.6	24.3	40.1	32.4	55.1
5	26.2	18.5	27.9	14.5	13.3	26.4
6	19.1	15.7	19.5	28.4	32.8	36.8
7	19.7	14.4	26.1	18.6	24.5	34.2
8	38.6	29.3	34.2	28.7	25	30.7
9	24.9	11.2	10.9	10.6	17.2	35.3
10	28.9	18.8	11.8	18.3	16.5	26.2
11	22.2	17.6	18	19.1	15.6	21.6
12	21.7	18	15.5	38.3	23.9	31.9

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Table 9 IVC: M- Mode& 2D-Echo

Name	*IVC size (cm)*		*Respiratory Variation*			*Estimated RAP (mm Hg)*	*SEC*
	Ins	*Exp*	*>50%*	*<50%*	*Absent*		
1	2.53	2.12	–	+	–	15-20	+
2	2.45	2.26	–	+	–	15-20	+
3	1.70	1.70	–	–	+	10-15	–
4	1.60	1.60	–	–	+	10-15	+
5	1.55	1.43	–	+	–	10-15	–
6	1.93	1.90	–	–	+	10-15	+
7	1.78	1.44	–	+	–	10-15	–
8	1.84	1.76	–	+	–	10-15	+
9	2.48	1.35	–	+	–	15-20	+
10	2.5	2.3	–	+	–	15-20	+
11	2.19	1.8	–	+	–	10-15	+
12	2.21	1.92	–	+	–	10-15	+

Table:10 Analysis of mitral annular TDI

	Mitral Annulus(cm/s)				Tricuspid Annulus(cm/s)			
Name	S’	E’	A’	E/E’	S’	E’	A’	E/E’
1	7.1	11.7	8.6	38.5/11.7 =3.29	7.8	11.2	6.4	36.8/11.2=3.2
2	6.4	12.8	6.6	58.8/12.8=4.6	7.6	10.6	7.0	55.1/10.6=5.2
3	6.6	13.2	5.5	54.5/13.2 =4.12	7.3	9.6	6.6	39.1/9.6 =4.07
4	7.4	17.4	5.5	100.1/17.4=5.75	8.5	15	6.1	86.6/15=5.77
5	5.3	7.4	5.6	82.7/7.4 =11.17	10.3	14	7.4	95.2/14=6.8
6	6.6	9.9	5.4	65.1/9.9 =6.57	9.8	18.6	8.8	57.7/18.6=3.10
7	7.9	14.1	4.3	97.5/14.1 =6.91	12.9	18.7	3.7	81.4/18.7=4.35
8	9.7	15.1	6.6	58.6/15.1 =3.88	11.2	16.4	4.8	80.6/16.4=4.92
9	6.68	19.5	9.47	81/19.5=4.15	9.6	10.7	5.2	43.8/10.7=4.1
10	7.12	8.87	5.36	67.5/8.87=7.6	8.77	6.24	14.7	47.2/6.24=7.5
11	7.9	11.4	6.2	90.8/11.4=7.96	10.9	17.1	16.4	48.4/17.1=2.83
12	7.8	12.5	7.4	53.6/12.5 =4.28	7.7	13.8	7.8	65.6/13.8=4.76

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Table:10 The mean value of peak early diastolic Tricuspid annular velocity (E') was 13.49 cm/s. The mean value of peak late diastolic Tricuspid annular velocity (A') was 7.9 cm/s.The mean value of peak systolic Tricuspid annular velocity (S') is 9.36 cm/s normal value of S' > 11.5 cm/s). Mean value of the ratio (E/E' ratio) between peak early diastolic Tricuspid inflow velocity (E) and peak early diastolic Tricuspid annular velocity (E') was 4.71 (range: 2.83 to 7.5).

Table 11; Hemodynamic Data Of Patients With Constrictive Pericarditis

Name	*Mean RAP*	*Peak RVS P*	*RV RF W*	*Ins↓ RA P*	*RV ED P*	*Peak PAS P*	*Mean PAP*	*Mean PCW P*	*Peak LVS P*	*LV RF W*	*LV ED P*	*Mean Ao Pressure*
1	16	33	8	–	19	28	20	20	100	10	18	90
2	18	40	17	–	18	40	30	22	120	20	20	100
3	18	30	8	–	18	28	22	20	86	8	21	70
4	21	30	12	–	18	26	16	16	90	20	21	70
5	20	32	7	+	22	30	24	20	96	5	21	70
6	24	36	8	–	20	34	28	20	120	12	18	100
7	20	40	9	–	24	36	28	24	129	22	26	80
8	24	50	8	–	24	48	36	22	96	10	20	80
9	20	38	10	–	20	32	26	21	86	11	18	70
10	22	36	9	–	20	34	24	20	100	12	21	70
11	21	40	10	–	22	40	30	22	110	8	20	74
12	16	30	8	–	21	32	26	19	94	7	19	70

Table 12:Analysis of Hemodynamic Data for Conventional and Dynamic Catheterization Criteria

Name	*Square root sign*	*LVEDP-RVEDP (<5mmHg)*	*RVEDP>1/3 RVSP*	*PASP <55mm Hg*	*Insp↓ MRAP*	*VID*	*PCWP-LVEDP> 5mm Hg*
1	RV&LV	2	+	+	absent	+	+
2	RV&LV	2	+	+	absent	+	+
3	RV&LV	3	+	+	absent	+	+
4	RV&LV	3	+	+	absent	+	+
5	RV	1	+	+	present	+	+
6	RV&LV	4	+	+	absent	+	+
7	RV&LV	2	+	+	absent	+	+

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8	RV&LV	4	+	+	absent	+	+
9	RV&LV	2	+	+	absent	+	+
10	RV&LV	1	+	+	absent	+	+
11	RV&LV	2	+	+	absent	+	+
12	RV&LV	2	+	+	absent	+	+

Table 13Hemodynamic and TDI correlation in patients with Constrictive Pericarditis (Mitral Annulus)

Name	***PCWP (Cath Data)***	***E/E' (TDI Data)***	
		Mitral	***Tricuspid***
1	**20**	**3.29**	**3.2**
2	**22**	**4.6**	**5.2**
3	**20**	**4.12**	**4.07**
4	**16**	**5.75**	**5.77**
5	**20**	**11.17**	**6.8**
6	**20**	**6.57**	**3.10**
7	**24**	**6.91**	**4.35**
8	**22**	**3.88**	**4.92**
9	**21**	**4.15**	**4.1**
10	**20**	**5.36**	**7.5**
11	**22**	**7.96**	**2.83**
12	**19**	**4.28**	**4.76**

Table 14 Hemodynamic and TDI correlation in patients with Constrictive Pericarditis – Tricuspid Annular TDI& Mean RAP

Name	***Cath Data mRAP(mm Hg)***	***TDI Data E/E'***
1	**16**	**3.2**
2	**18**	**5.2**
3	**18**	**4.07**
4	**21**	**5.77**
5	**20**	**6.8**
6	**24**	**3.10**
7	**20**	**4.35**

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8	**24**	**4.9**
9	**20**	**4.1**
10	**22**	**7.5**
11	**21**	**2.8**
12	**16**	**4.76**

Discussion

It is important to make the diagnosis of constrictive pericarditis because untreated patients have progressive hemodynamic and physical deterioration and a limited life span. Conversely, complete resection of all constrictive pericardium can result in marked improvement in symptoms as well as prolongation of life with a possible cure. Of the 12 patients in the study group, the clinical and echocardiographic features of constrictive pericarditis had an excellent correlation with CT scans and cardiac catheterization studies. [9] Elevated jugular venous pressure, a hallmark of constrictive pericarditis was present in 11 out of 12 patients. Its absence of one patient could be related to the prior diuretic therapy. The typical pattern of the jugular venous waveform (y>x) was present in three fourth of the patients. Two patients had equally prominent x and y waves and moderate pericardial effusion demonstrated subsequently during echocardiography, consistent with the diagnosis of Effusive – constrictive pericarditis. Kussmaul's sign was present in 10 patients (84%). Pulsus Paradoxus was present in 3 patients (25%) among which, two patients had features of Effusive - constrictive pericarditis. The characteristic auscultatory sign of constrictive pericarditis- early diastolic pericardial knock was audible in three fourth of the patients. This is higher than in Western studies. [10] Systolic murmur of mitral regurgitation was audible in one-fourth of the patients, although mitral regurgitation was demonstrable in half of the patients during echocardiography (mild MR-5 patients; moderate MR -1 patients). There was no audible murmur of tricuspid regurgitation or pericardial rub. All the patients in the study were in sinus rhythm. Although atrial fibrillation has been documented in up to 50% of Western patients, it was notably absent in this study. [11] Pericardial calcification was seen in 3 patients (25%)by transthoracic echocardiography and in 5 patients(50%) by TransEsophageal echocardiography. Also, TEE was more sensitive to diagnosing AV groove calcification which was present in 3 patients(25%).

But CT scan demonstrated pericardial calcification in one-third of the patients who also had pericardial calcification in fluoroscopy. Similar to TEE, AV groove calcification was present in 3 patients (25%).

M-Mode echocardiography was insensitive for confirming the presence or absence of pericardial calcification(2 patients had calcification). Pericardial thickening was demonstrable by M-mode echocardiography in all the patients. The mean thickness was 16.1 mm posteriorly. [12] Anterior pericardial thickening was seen in 3 patients (range: 3.5 to 11 mm) 2D echocardiography could not demonstrate pericardial thickening in one patient which was evident in TEE and CT scan. This patient had a minimal thickening of all- 4 mm by CT scan and 6.3 mm by TEE. Thus TEE and CT scans are more sensitive than 2D echocardiography to demonstrate pericardial thickening. The mean pericardial thickness was 15 mm (range: 4 to 35 mm) by CT scan and 13.9 mm (LV);10.7 mm(RV). Pericardial thickness was maximal over RA in one-third of the cases. RA and RV were involved more frequently and severely than LV. [13]Pericardial effusion was present in two third of the patients by TEE and CT scan. Prominent pericardial strands were better visualized by TEE. Prominent pericardial strands are usually associated with a risk of future development of constriction, if not developed earlier. [14] This high incidence of SEC and intracardiac thrombus are secondary to venous stasis with raised venous pressure. The intracardiac thrombus is a potential source of pulmonary embolism with significant morbidity and mortality. Thus TEE is very essential in the evaluation of patients with

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constrictive pericarditis. It provides excellent data on pericardial thickening and calcification, and organized/loculated effusion. TEE is as sensitive as a CT scan for pericardial thickening. In addition, it provides valuable information regarding pericardial strands and intracardiac thrombi which are important during pericardiectomy. Also TEE provides a better Doppler evaluation of pulmonary venous flow and involvement of the AV groove by the constricting pericardium. These pathophysiological abnormalities have been applied to Doppler echocardiographic studies in which the dissociation of intrathoracic and intracardiac pressures is manifested by an inspiratory increase in the peak tricuspid flow velocity and a simultaneous decrease in mitral flow velocity, with opposite changes occurring in expiration.[15]Although these Doppler findings are usually diagnostic in the presence of other clinical and noninvasive findings consistent with constrictive pericarditis, both false-positive and false-negative results exist. [16] Severe lung disease with marked respiratory changes in intrathoracic pressures and movement of the sample volume relative to the heart can cause changes in the mitral flow velocity curves mimicking those of constrictive pericarditis. Marked increases in left atrial pressures may mask these Doppler respiratory changes.[17] In this study, the dynamic respiratory changes in cardiac hemodynamics in patients with constrictive pericarditis were assessed by cardiac catheterization and correlated with the TDI. The finding of increased ventricular interaction, as assessed by respiratory discordance of left ventricular and right ventricular pressures, was the most reliable hemodynamic factor for distinguishing patients with constrictive pericarditis from those with other disease entities. [18] In patients with constrictive pericarditis, there was a consistent increase in right ventricular pressure during peak inspiration, a time when left ventricular pressure is lowest. The assessment of LV filling pressure is clinically important in patients with established heart disease and usually requires invasive hemodynamic measurement. 19 The high incidence of RA/RAA thrombus in patients with constrictive pericarditis as highlighted in this study, emphasizes the role of TEE in planning surgical treatment and preventing significant morbidity and mortality from pulmonary embolism. Since a significant proportion of the patients with constrictive pericarditis have marked pericardial thickening around RA, total pericardiectomy including the pericardium around RA is mandatory for good surgical results. This finding can better be evaluated by TEE.[20]

Conclusion

Patients with clinically suspected constrictive pericarditis who have classic findings on Doppler echocardiography can now undergo pericardiectomy without the need for cardiac catheterization. Only the patients in whom there have been equivocal findings either on clinical presentation or on noninvasive testing then undergo further hemodynamic assessment. Echocardiography has replaced invasive cardiac catheterization for various hemodynamic assessments. Of all of the hemodynamic measurements used in daily clinical practice, LV filling pressure is one of the most frequently used. With the advent of TDI and other recent developments in the field of echocardiography, it has become a truly versatile and reliable hemodynamic imaging tool. And, echocardiography has become a noninvasive Swan-Ganz catheter.

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