Scholars LITERATURE

International Journal of Integrative Cardiology

Case Report

DOI: https://doi.org/10.47275/2690-862X-109
Volume 2 Issue 1

A Rare Case of Effusive-constrictive Pericarditis

Maheshwar Honnalli and Sagar Mali*

SDM Narayana Heart Centre, SDM medical college, Sattur, Dharwad-580009, Karnataka, India

Abstract

Constrictive pericarditis and effusive-constrictive pericarditis are part of a clinical spectrum rather than two separate diseases. The diagnosis of his entity requires a systemic approach. A failed treatment with diuretics, anti-inflammatory therapy, and pericardiocentesis in reversing pathophysiology of effusive-constrictive pericarditis warrants for surgical approach i.e. pericardiectomy. We are reporting a rare case of effusive-constrictive pericarditis.

Keywords: Effusive-constrictive Pericarditis; Cardiac Catheterization; Pericardiectomy

Correspondence to: Sagar Mali, SDM Narayana Heart Centre, SDM medical college, Sattur, Dharwad-580009, Karnataka, India; E-mail: sagar5838@gmail.com

Citation: Honnalli M, Mali S (2020) A Rare Case of Effusive-constrictive Pericarditis. Int J Integr Cardiol, Volume 2:1. 109. doi: https://doi.org/10.47275/2690-862X-109.

Received: June 01, 2020; **Accepted:** June 24, 2020; **Published:** June 30, 2020

Learning objectives

- Effusive constrictive pericarditis is still an elusive clinical entity for cardiologists.
- In the Indian scenario, M. tuberculosis should be ruled out as a causative organism because of its high prevalence rate.
- Only partial decortication around ventricles during pericardiectomy for difficult constrictive pericarditis cases may help patients to normalize cardiac hemodynamics and avoid risking patients of complications like a puncture of atria while performing complete decortication.

Introduction

Around 300 years ago, constrictive pericarditis was called Concretio Cordis [1]. Even though its cardiac hemodynamics was described 50 years back [2,3] our current understanding of constrictive pericarditis is limited. Over the last two decades, studies have tried to elaborate on complex mechanisms behind non-compliant pericardium due to pericardial inflammation which leads to constrictive pericarditis or effusive-constrictive pericarditis [4]. Constrictive pericarditis and effusive-constrictive pericarditis are part of a clinical spectrum rather than two separate diseases. The prevalence of effusive-constrictive pericarditis ranges from 2.4% to 14.8%. It is caused by viral infection/idiopathic cause (42-49% of cases), cardiac surgery (11-37% of cases), radiation (9-31% of cases), connective tissue disorder (3-7% of cases) or tuberculosis (3-6% of cases) [5,6]. Patients usually present with raised jugular venous pressure (JVP), peripheral edema, ascites (due to right-sided congestion); dyspnoea, effort intolerance (due to inadequate ventricular filling); and rarely pulmonary edema.

A failed treatment with diuretics, anti-inflammatory therapy, and pericardiocentesis in reversing pathophysiology of effusive-constrictive pericarditis warrants for surgical approach i.e. pericardiectomy.

Case presentation

A 56-year-old male presented to the cardiac out-patient department with a history of fever, associated with chills of one-day duration. He also complained of progressive breathlessness and cough predominantly dry in nature which aggravated deep breathing. The patient didn't give any history of paroxysmal nocturnal dyspnoea or pedal edema. The patient had undergone visual internal urethrotomy and transurethral incision of prostate and bladder neck contracture two weeks back.

On examination, he was conscious, co-operative, and well oriented to time, place, and person. His vital data showed pulse rate-108 bpm, blood pressure-91/60 mmHg, respiratory rate-18/min, SPO2-99% at room air and he was afebrile. General physical examination of the patient showed a pallor and distended neck veins. Jugular venous pressure was 15 cm and pulsatile. His thyroid gland was not enlarged and could not be felt on palpation. The rest of the physical examination was inconclusive.

On investigation, ECG showed sinus tachycardia, T wave inversion in the chest leads V3-V6 and low voltage complexes (Figures 1 and 2).

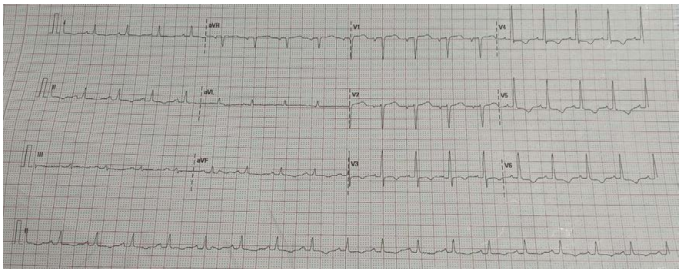

Figure 1: ECG showing low voltage complexes.ECG showed sinus tachycardia, T wave inversion in chest leads V3-V6 and low voltage complexes.

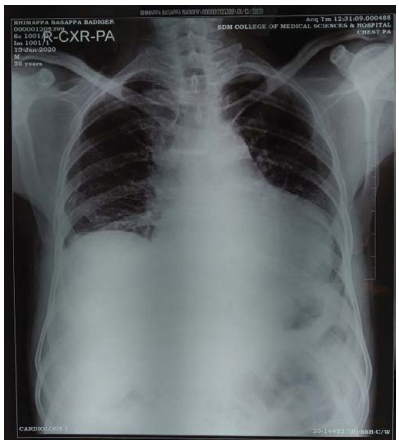

Figure 2: Chest X-ray showing cardiomegaly and clear lungs.

Echocardiography showed pericardial effusion without tamponade with pericardial thickening (26 mm posterolaterally near left ventricle and 8mm anteriorly near right atrium) and mild PAH (pulmonary artery hypertension) with normal left ventricular function (Figure 3).

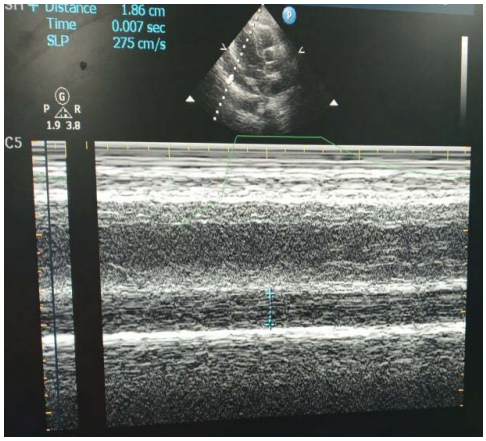

Figure 3: Echocardiographic image pericardial effusion and thickening.

Patient's plain CT scan revealed moderate pericardial effusion with maximum thickness of 26 mm and associated thickening of parietal pericardium. It showed mild inter-lobar effusion on left side and few subcentimetric pre-para tracheal and perivascular lymph nodes (Figure 4).

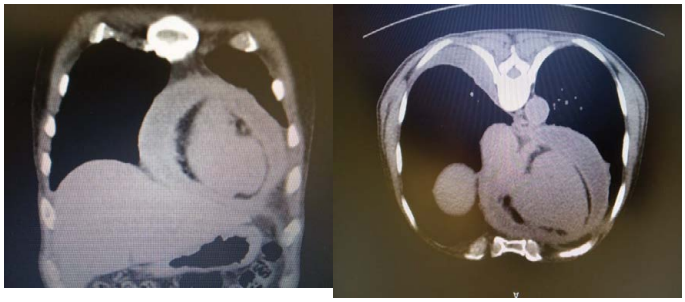

Figure 4: Plain CT scan of chest showing moderate pericardial effusion with maximum thickness 26 mm and associated thickening of parietal pericardium.

He was admitted to the cardiac intensive care unit for pericardiectomy. Coronary artery angiography revealed no significant coronary artery disease but showed limited motion at apical LAD (Left Anterior Descending) and proximal RCA (Right Coronary Artery) suggesting pericardial constrain of the anterior surface of RV (Right Ventricle) and RA (Right Atrium) groove, apical portion of the interventricular groove, leaving a posterior and basal portion of heart free. The patient's cardiac hemodynamics was studied by cardiac catheterization. It revealed the square root sign on ventricular pressure tracing with a marked decrease in inspiratory LV pressure compared to expiratory LV pressure (Figure 5).

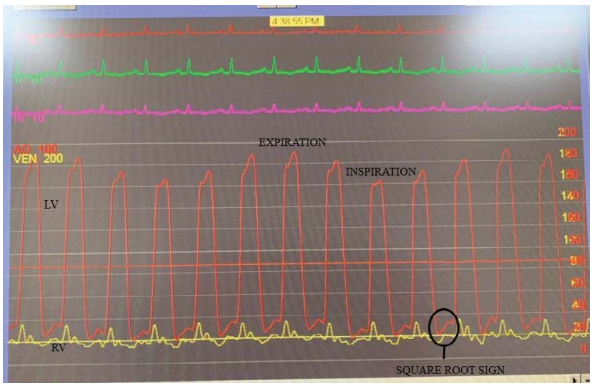

Figure 5: Cardiac catheterization study showing LV (left ventricular) and RV (right ventricle) pressure tracing with square root sign.

Lab values are summarized in (Table 1).

He underwent pericardiectomy procedure. On sternotomy, anterior surface of RV, RVOT (Right Ventricular Outflow Tract) showed thick, adherent pericardium extending to the aorta. There was pericardial fluid covering the lateral surface and posterior surface of LV with fibrous strands. Pericardial fluid and tissue from pericardial space were collected during the procedure and sent for analysis. Biochemistry and microbiological tests were performed on pericardial

Table 1: Laboratory investigation report showed no deranged parameter.

Parameter	Result	Ref. Range
Blood investigation		
Hemoglobin	12.9 gm/dl	12-16
Total count	9,840 cells / mm^3	4000-12000
ESR	12 mm/hr	1-13 mm/hr
Renal function tests		
Blood urea	10 mg/dl	15-45
Serum Creatinine	0.96 mg/dl	0.5-1.2
Liver function tests		
Total protein	7.3 gm/dl	-
Albumin	2.5 gm/dl	3.5-5
AST	28 U/L	15-59
ALT	37 U/L	13-72
Alkaline phosphatase	139 U/L	38-126
GGT	17 U/L	12-73
Viral markers		
HIV	Non-reactive	-
HCV	Non-reactive	-
HBsAg	Negative	-
Pericardial fluid analysis		
Microscopy: Gram stain- Occasional pus cells, no organisms seen, ZN stain- Negative for acid fast bacilli		
Biochemistry		
Protein	5.4 mg%	15-45
Sugar	10 mg%	70-110
Thyroid function tests		
Serum T3	3.3 nmol/L	1.2-3.1
Serum T4	1.1 nmol/L	66-181
TSH	2.12 mcIU/ml	0.7-6.4
Hematology		
Prothrombin time	11.7 sec	control 12.4 sec
INR	0.9	-

fluid (Table 1). Histopathology analysis of pericardial tissue showed coagulative necrosis. The patient succumbed to death due to RA (Right Atrium) puncture and excessive blood loss from the peritoneal surface during the procedure.

Discussion

Constrictive pericarditis is due to thickened, fibrotic, adherent (at times calcified) non-compliant pericardium restricting diastolic filling of ventricles. The first stage of acute pericarditis causing fibrin deposition and pericardial effusion slowly progresses to the sub-acute stage of organization and resorption of effusion over a while. In the chronic stage, fibrous scarring and thickening of pericardium lead to the obliteration of pericardial space. Due to non-compliant pericardium, a decrease in intrathoracic pressure during inspiration is not transmitted to intracardiac pressures. This leads to a further decrease in pulmonary venous pressure causing low left-sided preload and stroke volume during inspiration. Such changes in stroke volume with respiration are minimal in the normal heart. A stiff ventricular -pericardial unit greatly exaggerates pericardial coupling causing ventricular interdependence in constrictive pericarditis [7].

On examination, almost all patients have raised JVP. An increase in JVP during inspiration (Kussmaul's sign) indicates stiff right-sided heart resisting abdominal venous return. JVP also has prominent x and y descent. High pitched pericardial knock can be heard on auscultation along the left sternal border. Among laboratory tests, raised BNP suggests restrictive cardiomyopathy.Echocardiography can be a diagnostic tool in constrictive pericarditis showing ventricular interdependence, intrathoracic intracardiac dissociation, respirophasic septal shift, increased inspiratory tricuspid flow velocity, abnormal beat-to-beat septal motion (shudder), inferior vena cava plethora, expiratory hepatic vein reversal and low diastolic forward flow [8,9].

In constrictive pericarditis, a chest X-ray can show enlarged cardiac silhouette and pericardial calcification. Chest CT and MRI can help to detect pericardial tethering and thickening indicating the presence of constrictive pericarditis. Chest MRI can help detect active pericardial inflammation and cardiomyopathic processes [10].

Cardiac catheterization is considered a gold standard diagnostic tool to assess increased filling pressures and diastolic equalization in all four chambers [11]. Few patients respond to palliative medical treatment with diuretics and anti-inflammatory therapy if constriction is due to acute inflammation. Curative treatment is pericardiectomy. The left anterolateral thoracotomy provides good exposure to the left ventricle for resection of the thickened pericardium. The incision can be extended superiorly and laterally onto the right side of the heart. Few studies also suggested that decortication of the anterior and diaphragmatic surface of ventricles can normalize cardiac hemodynamics. The outcome of the surgery also depends on the extent of myocardial involvement and duration of illness [12].

In our case, the patient was diagnosed with effusive-constructive pericarditis based on a clinical picture (progressive dyspnoea, raised JVP), echocardiography, chest CT scan, and cardiac catheterization findings. Histopathology analysis of resected tissue during pericardiectomy showed coagulative necrosis. Due to puncture during decortication near the right atrium, the patient was put on a heart-lung machine. The patient had excessive bleeding from the peritoneal surface and he received 12 units of blood and blood components. Despite all measures, he succumbed to death.

Acknowledgement

We would like to thank Dr Vivekanand Gajapati, Head of Cardiology Department, for his expert advice and encouragement. I would also like to thank Dr Kirti L for her support.

References

1. Myers RB, Spodick DH (1999) Constrictive pericarditis: Clinical and pathophysiologic characteristics. Am Heart J 138:219-232.https://doi.org/10.1016/S0002-8703(99)70105-5
2. Spodick DH, Kumar S (1968) Subacute constrictive pericarditis with cardiac tamponade. Dis Chest 54:62-66.https://doi.org/10.1378/chest.54.1.62
3. Burchell HB (1954) Problems in the recognition and treatment of pericarditis. J Lancet 74:465-470.
4. Gentry J, Klein AL, Jellis CL (2016) Transient constrictive pericarditis: current diagnostic and therapeutic strategies. Curr Cardiol Rep 18:41.https://doi.org/10.1007/s11886-016-0720-2
5. Sagristà-Sauleda J, Angel J, Sánchez A, Permanyer-Miralda G, Soler-Soler J (2004) Effusive-constrictive pericarditis. N Engl J Med 350:469-475.https://doi.org/10.1056/NEJMoa035630
6. Imazio M, Brucato A, Maestroni S, Cumetti D, Belli R, et al. (2011) Risk of constrictive pericarditis after acute pericarditis. Circulation 124:1270-1275.https://doi.org/10.1161/CIRCULATIONAHA.111.018580
7. Lorell BH, Braunwald E (1992) Pericardial disease. Braunwald E (Eds.) Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia: WB Saunders Co. 1465-516.
8. Oswal MP, Ponde CK (2019) Right-sided constrictive pericarditis: A rare variant of an uncommon disease.J Ind Acad Echocardiogr Cardiovasc Imag 3:23-26.https://doi.org/10.4103/jiae.jiae_39_18
9. Coylewright M, Welch TD, Nishimura RA (2013) Mechanism of septal bounce in constrictive pericarditis: a simultaneous cardiac catheterisation and echocardiographic study. Heart 99:1376.http://dx.doi.org/10.1136/heartjnl-2013-304070
10. Muehlberg F, Toepper A, Fritschi S, Prothmann M, Schulz-Menger J (2016) Magnetic resonance imaging applications on infiltrative cardiomyopathies. J Thorac Imag 31:336-347.https://doi.org/10.1097/RTI.0000000000000199
11. Geske JB, Anavekar NS, Nishimura RA, Oh JK, Gersh BJ (2016) Differentiation of constriction and restriction: complex cardiovascular hemodynamics. J Am Coll Cardiol 68:2329-2347.https://doi.org/10.1016/j.jacc.2016.08.050
12. Rajarajan PA (2019) Clinical and surgical outcomes of pericardiectomy done by total anterolateral thoracotomy. IAIM 6:22-26.