

Case Report

Transient Constrictive Pericarditis in Multiple Myeloma : Unusual Case

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ABSTRACT

Transient constrictive pericarditis has never been reported in multiple myeloma. 2D echocardiography in a 69 year old male patient of multiple myeloma, at presentation revealed features of constrictive pericarditis which reversed to normal with chemotherapy. Thus a diagnosis of transient constrictive pericarditis was made.

Keywords : Constrictive pericarditis, Multiple myeloma, Tissue doppler, Pericardial fibrosis

Introduction :

Cardiac involvement in multiple myeloma is a rare finding. It usually presents with anemia, hypercalcaemia, renal insufficiency and bone destruction. Classic constrictive pericarditis (CP), a progressive and debilitating condition, is characterized by pericardial fibrosis, with or without calcification, which results in chronic refractory congestive heart failure, the treatment of which involves pericardiectomy. In recent years there have been reports describing a transient form of CP that resolves without surgical intervention. The development of constrictive hemodynamics and subsequent resolution with medical therapy (transient CP) was first described by Sagrista-Sauleda et al. in 1987.¹ We discuss a rare case of transient constrictive pericarditis seen in a patient of multiple myeloma.

Case Report :

69-year-old male was diagnosed in a multispecialty hospital with multiple myeloma for systemic symptoms of easy fatigability, pallor, bone pain & dyspnea on exertion since 1 month. On examination he had pallor & features of congestive cardiac

failure (edema feet, congestive hepatomegaly and raised jugular venous pressure with sharp, deep Y descent). He was investigated, which revealed normocytic normochromic anemia, slightly abnormal liver and kidney functions, congestive hepatomegaly and normal kidneys on ultrasonography. His routine lab evaluation showed hemoglobin 6 gm/dl, leucocyte count of 6400 /cu. mm, platelet count 6,20,000/cu. mm, normal blood urea, serum creatinine 1.6 mg/dl, eGFR 45.78 mL/min/1.73m² by MDRD formula, ionic calcium 1.32 mg/dl, serum Na 132 meq/L & serum K 4.3 meq/L. He was advised 2D echocardiography for cardiac evaluation. 2D echocardiography evaluation at the time of initial presentation was suggestive of constrictive pericarditis. It showed normal left ventricular (LV) function, thickened pericardium, ventricular septal shift (septal bounce), respiratory variation in ventricular filling, congested inferior vena cava (IVC) & mild pulmonary hypertension. However tissue doppler imaging (TDI) was not in tune of constrictive pericarditis. (*Online Figure 1*) He underwent treatment for multiple myeloma for which he received chemotherapy at regular intervals as prescribed by his treating physician. He achieved complete remission of multiple myeloma with above treatment in 8 months. At interval of around 9 months, repeat 2D echocardiography was performed. This time, it showed normal LV function, no septal bounce, normal respiratory variations on mitral valve (MV) inflow, normal PA pressure & normal IVC. (*Online Figure 2*)

Discussion :

Constrictive pericarditis (CP) is an important cause of right and/or left ventricular (RV/LV) failure

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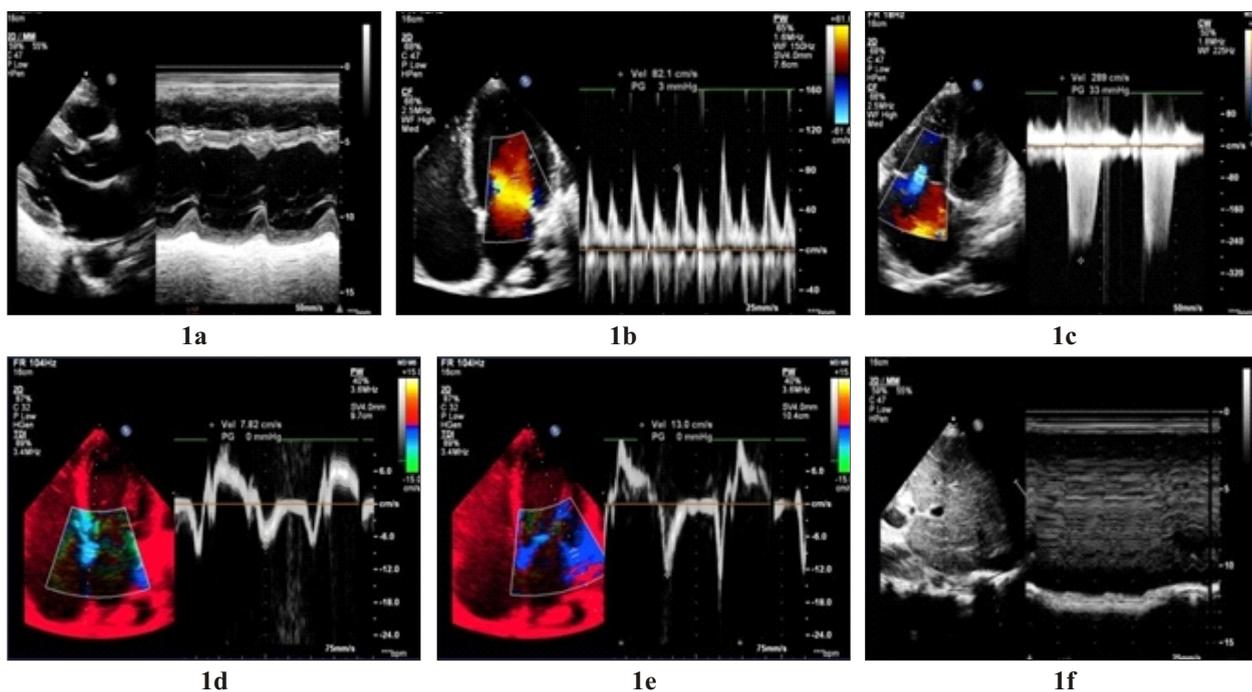


Fig. 1 : 2d echocardiography ap presentation showing ventricular septal shift i.e. septal bounce (1a), respiratory variation in ventricular filling i.e.> 25% respiratory variations in mitral valve inflow pattern (1b), mild pulmonary hypertension (1c), normal tissue doppler imaging (1d, 1e), dilated & congested inferior venacava (1f).

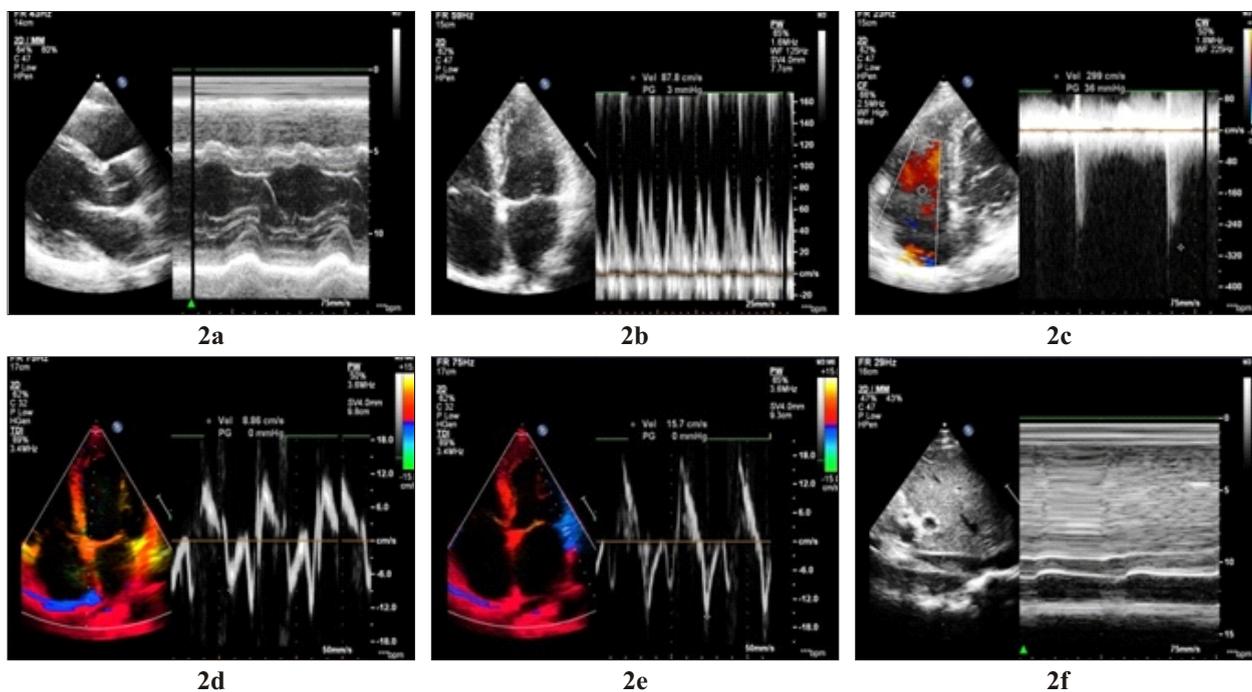


Fig. 2 : Repeat 2d Echocardiography around 9 months showing normal ventricular septal shift i.e. no septal bounce (2a), normal respiratory variation in ventricular filling i.e.< 25% respiratory variations in mitral valve inflow pattern (2b), mild pulmonary hypertension (2c), normal tissue doppler imaging (2d, 2e), normal inferior vena cava (2f).

caused by a reduction in the elasticity of the pericardium resulting in impaired diastolic filling of the heart. All types of CP were thought to be irreversible until 1987. Sagrista-Sauleda et al. described transient nature with objective evidence of constrictive pericarditis. He showed resolution with medical therapy and observation in effusive acute idiopathic pericarditis cases.¹

Most common causes of transient CP are idiopathic, viral pericarditis, and postpericardiotomy pericarditis that usually develops within 3 months of cardiac surgery.² Approximately 10% of patients with acute pericarditis complicates with constrictive physiology.³

In 1980, Hancock described two forms of pericardial constriction, one being elastic and the other more analogous to a rigid shell. The fibroelastic form, he proposed, represents the acute or subacute phase of constriction. He noted that patients with subacute fibroelastic constriction present with more subtle signs and symptoms, rather than the classic findings seen in chronic CP. In many patients who are identified in the course of the initial pericardial inflammatory response, pericardial inflammation continues and pericardial fibrosis and calcification subsequently develop, leading to chronic (rigid) CP.⁴ This may be the reason of normal TDI in our case as there was no fibrosis in pericardium. Usually constrictive findings resolve at an average time of three months.²

Multiple myeloma is characterized by a proliferation of malignant plasma cells and usually presents with bone pain and organic damage. The most frequent presentation is anemia, hypercalcemia, renal insufficiency and bone destruction.⁵

Pericardial involvement in multiple myeloma is very rare. In a study of extramedullary presentation of multiple myeloma, authors reviewed 869 patients & found no patients with pericardial disease.⁶ Few reports have shown cardiac involvement in multiple myeloma.^{7,8} In Mayo series of transient constrictive pericarditis of 36 patients, there was no case associated with multiple myeloma.²

In our patient, at presentation he had constrictive pericarditis with normal TDI which completely resolved after chemotherapy at 9 months. Thus we labelled it as transient. Till date, entity of transient constrictive pericarditis in literature has not been described in multiple myeloma. Moreover author is of opinion that lack of TDI parameters of constriction in our case could be due to absence of fibrosis, a feature of transient constrictive pericarditis.

Conclusions :

Presentation of transient constrictive pericarditis is a rare finding in multiple myeloma. In our case, constrictive pericarditis resolved with complete remission of multiple myeloma.

Learning objective : Absence of TDI findings on 2d echocardiography at presentation of constrictive pericarditis may suggest its transient nature.

Conflicts of interest : None

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