



Constrictive Pericarditis, a Difficult Diagnosis -A Case Report with Review of Literature

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ABSTRACT

The diagnosis of constrictive pericarditis requires a high degree of clinical suspicion as the signs and symptoms of this disease can be falsely attributed to other causes. Here we present a case of a 50-year old male who had presented with bilateral pleural effusion, ascites, fever and history of pulmonary tuberculosis. So he was initially thought to be a case of disseminated tuberculosis or pulmonary tuberculosis associated with IVC (inferior vena cava) obstruction. He was evaluated clinically and required investigations were done which revealed constrictive pericarditis. It sets an example of the difficulty in diagnosing this condition.

KEY WORDS: Constrictive Pericarditis, JVP (jugular venous pressure), Tuberculosis, ATT (antituberculin therapy).

INTRODUCTION

The pericardium is a two-layered sac that encircles the heart consisting of visceral and parietal pericardium. Between these layers is a thin film of liquid, with a total volume of around 50 ml. In conditions like healing of an acute fibrinous or serofibrinous pericarditis or resorption of a chronic pericardial effusion leads to constrictive pericarditis (CP)¹. Scarring and fibrotic changes alter the pericardium and prevent it and the heart from expanding and diastolic filling². In developing nations where the condition is prevalent, most cases are of tubercular origin, but in developed countries the majority cases are idiopathic, postcardiac surgery, traumatic, purulent infection, neoplastic disease (breast cancer, lung cancer, and lymphoma), connective

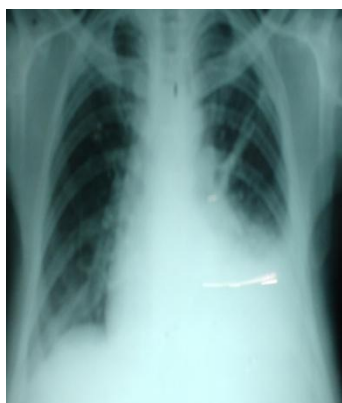
tissue disorders (RA, SLE), post-radiation therapy, or chronic renal failure on chronic dialysis^{1,3}.

CASE REPORT

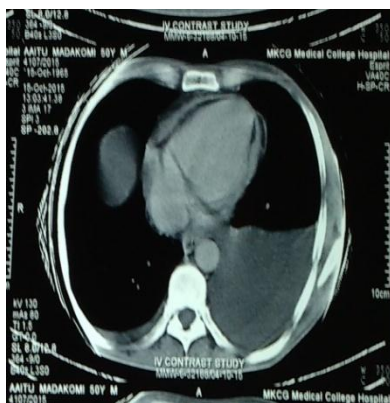
A 50-year-old man presented to our hospital with swelling of both lower limbs for five months, distension of abdomen, breathlessness and fever for 15-days. On admission he was febrile (101.4⁰F) and other clinical examination revealed pallor, pulse rate-92/min with pulsus paradoxus, BP-110/80mmHg, JVP at angle of mandible (6cm) in sitting position, bilateral lower limb edema up to upper thigh which was very firm on palpation indicating chronic edema. Patient was tachypnoeic with a respiratory rate of 22/minute and orthopnoea. Per abdominal examination showed parietal edema, tender hepatomegaly (3cm

below right costal margin), ascites and on examination of respiratory system, moderate left pleural effusion and mild right pleural effusion was found without any added sounds. His labs revealed Hemoglobin of 10.4g/dl, WBC count of $9.2 \times 10^3/\text{cmm}$ with Neutrophil-67%, Lymphocytes-29% ESR was 28mm in first hour, serum creatinine was 1.0 mg/dL, blood urea of 24mg/dl, Sr Na^+ -136, Sr K^+ -3.5, liver function test showed total bilirubin-1.6, direct bilirubin-0.4, AST-62, ALT-68, ALP-139 IU/L and MP (ICT), Hbs Ag, anti HCV, HIV tests were negative. X-ray chest detected moderate left pleural effusion, obliteration of right costophrenic angle, patchy opacity in right upper zone and linear opacity in left mid zone and sputum for AFB was negative. Ultrasound of abdomen showed hepatomegaly (16.6cm), moderate ascites, plethoric inferior vena cava and hepatic veins and bilateral pleural effusion. We started treatment with back rest, oxygen inhalation, Inj

Dexamethasone, Inj furosemide along with CAT-1 ATT with provisional diagnosis of disseminated tuberculosis involving lung parenchyma, pleura and pericardium. But his pleural and ascitic fluid study were transudate in nature with low protein, few lymphocytes and negative for ADA. 2D-ECHO showed thickened pericardium with good systolic function (EF-58%). ECG showed normal QRS complexes, regular in rhythm and inverted T waves in chest leads (V2 to V6). We could do CT scan of thorax only on 14th day of treatment which showed left pleural effusion with normal pericardium and pericardial space. Gradually patient improved clinically with treatment in the form of decrease in lower limb edema, ascites, breathlessness and he became afebrile. On 19th day patient was discharged with Cat 1 ATT + rifampicin 150 mg and oral corticosteroid (Prednisolone). On follow up after 15 days patient was asymptomatic except mild pedal edema.



(A)



(B)



(C)

A: - X-Ray Shows enlarged heart, left pleural effusion with patchy opacity right upper zone and linear opacity in left mid zone (Evidence of healed pulmonary TB).

B: - CT Thorax shows moderate left pleural effusion mild right pleural effusion with normal pericardium and pericardial space.

C: - Cachectic patient (on 14th day of treatment.)

DISCUSSION

The diagnosis of constrictive pericarditis is difficult both for its rarity and is often obscured by other, more common, diagnoses like cardiac tamponade, restrictive cardiomyopathy, right heart failure, RVMI (right ventricular myocardial infarction), tricuspid stenosis and hepatic

cirrhosis¹. In our case, the patient was taking CAT 1 ATT since one month for pulmonary tuberculosis under RNTCP, from local Hospital but at that time lower limb edema, ascites and raised JVP were not given importance.

Pathophysiology of this disease, in causing a thickened and non-compliant pericardial sac,

prevents the diastolic filling phase of the heart. Functionally, the changes during diastole lead to impaired filling of the heart causing venous back pressure and failure of outflow². Patients commonly present with weakness, fatigue, weight gain, increased abdominal girth, abdominal discomfort, edema and in advanced cases, anasarca, skeletal muscle wasting, and cachexia. They sometimes give symptoms of exertional dyspnoea and orthopnoea. The systemic venous congestion will cause the neck veins to be distended with presence of *Kussmaul's sign*. The pulse pressure is normal to reduced and a paradoxical pulse is found in one-third of cases. Congestive hepatomegaly is prominent, sometimes associated with impaired liver function, jaundice and ascites. Ascites disproportionate to the degree of hepatic impairment should raise clinical suspicion of constrictive pericarditis⁴. The cardiac apex is reduced and may retract in systole (*Broadbent's sign*). The heart sounds may be distant, sometimes associated with pericardial knock. The ECG frequently shows low voltage QRS complexes and diffuse flattening or inverted T waves and atrial fibrillation in one-third of patients. X-ray chest shows normal to slightly enlarged heart. Due to the difficulty in the clinical diagnosis, certain investigations prove vital in making the diagnosis. Important investigations include echocardiography, CT, MRI and cardiac catheterisation studies. The transthoracic echocardiogram typically shows pericardial thickening, dilation of inferior vena cava and hepatic veins, and a sharp halt in ventricular filling in early diastole, an early diastolic septal bounce, with normal systolic function. But CT and MRI scanning are more accurate in establishing or excluding the presence of a thickened pericardium^{1, 5, 6}. Cardiac catheterisation studies are necessary for the diagnosis of constrictive pericarditis. It causes early, rapid diastolic filling and equalisation of pressures between the ventricles⁷. In tubercular pericardial disease ATT is the main stay of treatment and pericardiectomy

should be considered if thickened pericardium is found while the patient is receiving antituberculous chemotherapy¹. In tubercular pericardial disease ATT is the main stay of treatment, if evidence of thickened pericardium is found pericardiectomy should be considered while patient is receiving antituberculous chemotherapy¹.

CONCLUSION

There are many causes of systemic venous congestion. Among them, constrictive pericarditis is a rare and commonly missed cause. The diagnosis of constrictive pericarditis is the first and most significant hurdle for which high degree of clinical suspicion is required. Early diagnosis and treatment of pericarditis and its aetiology are required to prevent further progression of the disease and that will improve quality of life and help a long-term survival benefit to the patient. Constrictive pericarditis should, therefore, feature on the differential list when a patient presents with signs of right heart failure.

REFERENCES

1. Eugene Braunwald. Pericardial Disease: Harrison's Principles Of Internal Medicine. 19th Edition. 2015 ; (288):1571-1577.
2. Marnejon Thomas, Gemmel David: The Constricted Heart. *Postgraduate Medicine* 2008, 120(1):8-10.
3. Bertog S, Thambidorai S, Parakh K, Schoenhagen P, Ozduran V, Houghtaling P. Constrictive pericarditis: etiology and cause-specific survival after pericardiectomy. *J Am Coll Cardiol.* 2004 Apr; 43 (8): 1445.
4. Marshall A, Ring N, Lewis T: Constrictive pericarditis: lessons from the past five years' experience in the South West Cardiothoracic Centre. *Clinical Medicine, Journal of the Royal College of Physicians* 2006, 6:592.
5. Himelman RB, Lee E, Schiller NB: Septal bounce, vena cava plethora, and

pericardial adhesion: informative two-dimensional echocardiographic signs in the diagnosis of pericardial constriction. *Am Soc Echocardiogr* 1988, 1(5):333-340.

6. Dal-Bianco JacobP, Sengupta ParthoP, Mookadam Farouk, Chandrasekaran Krishnaswamy, Tajik A Jamil, Khandheria BijoyK: Role of Echocardiography in the Diagnosis of Constrictive Pericarditis. *Journal of the American Society of Echocardiography* 2009, 22(1):24-33
7. Talreja DR, Nishmura RA, Oh JK, Holmes DR: Constrictive pericarditis in the modern era: novel criteria for diagnosis in the cardiac catheterization laboratory. *J Am Coll Cardiol* 2008, 51(3):315-9.