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Medicine

Tubercular constrictive pericarditis: Still a diagnostic dilemma in modern era of advanced molecular diagnostics

Ashish Hota Assistant Professor, Department of Medicine, Raipur Institute of Medical Sciences, Raipur, India

Binod Kumar Pati Assistant Professor, Department of Virology, PGIMER, Chandigarh

ABSTRACT In developed countries, idiopathic constrictive pericarditis is common but tuberculosis is still an important cause of constrictive pericarditis in India. Patients present with nonspecific complaints and features of right heart failure. Therefore, diagnosis is difficult as it requires high degree of clinical suspicion and often delayed treatment results in higher morbidity and mortality. We present here a challenging case of tubercular constrictive pericarditis where the etiology remained illusive until pericardiectomy and histopathological examination of pericardium was done.

KEYWORDS : Tuberculosis, Constrictive pericarditis, India

Introduction

Constrictive pericarditis results from chronic inflammation of both fibrous and serous layers leading to thickening, scarring and loss of elasticity of pericardium. As a result of restricted diastolic filling, venous return is reduced and cardiac output is decreased. Though idiopathic chronic pericarditis and pericarditis following cardiac surgery and mediastinal radiotherapy may lead to constrictive pericarditis, *Mycobacterium tuberculosis* is most common cause in tropical countries like India. Tubercular pericarditis still remains as a diagnostic challenge for the medical practitioners even in endemic area where clinical suspicion is high (Chhina D, 2013). Delay in treatment initiation is considered as main cause of chronic constrictive pericarditis.

Case Report

A 39-year-old male normotensive, nondiabetic medical laboratory technician with no history of smoking and alcohol intake belonging to middle socioeconomic status presented with two months' history of loss of appetite, significant loss of weight (8 k.g.), progressive dyspnoea and ankle swelling. There was no significant history of fever and cough. He was a known asthmatic for last 10 years with adequate control on inhalational steroid and long acting β_2 agonist. General physical examination revealed mild pallor, pitting edema of both feet, raised jugular venous pressure with blood pressure of 100/68 mmHg, pulse rate of 110/ min, and respiration rate of 21/min. Respiratory system examination was consistent with bilateral moderate pleural effusion. On cardiovascular system examination apex beat was not palpable, heart sounds were soft with no parasternal heave. Abdomen was distended with everted umbilicus, liver was palpable 3 cm below costal margin. There was no shifting dullness and bowel sound was normal. On evaluation, hemoglobin was 11.3 g/dl, high ESR (60 mm 1st hour), INR was 1.41, serum creatinine 0.8 mg/dl, total bilirubin 2.4 mg/dl, AST/ALT-65/73 U/L and TSH was 4.5 microIU/ml. Sputum AFB, Montoux test was negative and antinuclear antibody was not detected. He was seronegative for HIV, HBsAg and HCV. Pro BNP was high (861pg/ml). ECG was showing low voltage complexes. Chest Xray showed bilateral pleural effusion (with right side dominance) and normal cardiac shadow. Sonography of abdomen revealed mild ascitis & congestive hepatomegaly with normal echotexture and dilated IVC. Transthoracic echocardiography detected moderate pericardial effusion, no diastolic collapse of RA/RV, dilated IVC, however no regional wall motion abnormality or valvular anomaly was found. Pleural fluid was lymphocyte rich and transudative with low ADA (27 U/L). Both PCR from pleural fluid and repeated Genexpert from pleural fluid as well as ascitic fluid were negative for tuberculosis. CECT chest and abdomen revealed hepatomegaly with normal outline, dilated IVC, mild ascites, bilateral pleural effusion and mild pericardial effusion. However, no mediastinal or abdominal lymphadenopathy were detected and lung parenchyma was normal. Upper GI endoscopy was normal. Twenty-four-hour urinary protein was 417 mg/day. Bilateral therapeutic pleural tap was done and the patient improved with diuretics, i.v antibiotics, multivitamins and high protein diet. He was started with ATT and steroid considering polyserositis and endemic geography. However, after 1 month patient again presented with worsening dyspnoea (NYHA Class IV) and found to have moderate bilateral pleural effusion and required repeat therapeutic tap. A second echocardiography revealed septal bounce, pericardial thickening, dilated IVC with minimal phasic variation. Thickened pericardium (8-9 mm) was detected in CECT. A diagnosis of constrictive pericarditis was established and ATT continued with a future plan for pericardiectomy.

After 3 month of ATT, anterior total pericardiectomy was done by median sternotomy. All reflexion of pericardium from phrenic nerve to phrenic nerve was removed, posterior adhesionolysis was performed and outlet of the heart and root of the aorta and IVC was carefully made free. Histopathological report of removed pericardium revealed dense infiltration of chronic inflammatory cells & granulomas composed of epithelioid cells with few Langhan's type giant cells and granulomatous inflammation consistent with tubercular pericarditis. The postoperative course was uneventful and showed gradual improvement of heart function. He had been doing well without any further complications.

Discussion

This case exemplifies the difficulty in diagnosis of tubercular constrictive pericarditis despite the availability of newer methods of sensitive and rapid diagnosis like Genexpert and TB PCR. Though the patient presented with loss of appetite, loss of weight and later with right heart failure, history of fever was strikingly absent. Huge hepatomegaly with deranged LFT was misleading and pointing towards hepatic cause. Pericardial infection with *M. tuberculosis* occurs via extension of infection from adjacent organs like lungs, tracheobronchial tree, lymph nodes or via miliary spread. Often tuberculous pericarditis represents reactivation disease where primary focus may not be apparent. Tuberculous pericarditis develops in 1-2% of patients with pulmonary tuberculosis (Larrieu, Tyers, Williams, & Derrick, 1980). Literatures often describe four pathological stages of tubercular pericarditis (Mayosi, Burgess, & Doubell, 2005; Ortbals & Avioli, 1979), which include fibrinous exudation, serosanguinous effusion, pericardial thickening with fibrosis and finally constrictive scarring. Constrictive pericarditis develops in 30-60% of patients, despite prompt ATT and use of corticosteroids (Sagrista-Sauleda, Permanyer-Miralda, & Soler-Soler, 1988). Missing an early diagnosis and delay in institution of antitubercular therapy may complicate to constrictive pericarditis often requiring pericardiectomy. Most of the constrictive pericarditis

cases described by Hughes and Lipton were consequences of tuberculosis and pericardiectomy was required for all of them (Hughes & Lipton, 1949). In a series of 143 surgically confirmed cases of constrictive pericarditis Laghari et al. reported increased pericardial thickness in 37%, abnormal septal motion in 49%, atrial enlargement in 61% of patients and all thickened pericardium had evidence of tuberculosis in histopathological examination (Laghari & Tai, 2013).

Conclusion:

Establishing the early diagnosis of constrictive pericarditis is still challenging in an era of advanced molecular diagnosis. High index of suspicion is needed and tuberculosis should never be disregarded to be featured in the list of differential diagnoses in cases of right heart failure in endemic countries. Though surgical morbidity and mortality is high, definitive treatment with pericardiectomy shows better clinical outcome than medical therapy with ATT and steroid.

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