ACUTE FULMINANT MYOCARDITIS – A RARE COMPLICATION OF TUBERCULOSIS

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Introduction

Tuberculosis is generally thought to spare organs like Myocardium, thyroid, pancreas and skeletal muscle. Tuberculous involvement of the heart usually presents as pericarditis.Myocarditis as initial presentation is quite rare, prevalence of which in various series is about 0.2%[1].Herein we present a patient who happens to be a Pulmonary tuberculosis treatment defaulter presenting with Acute fulminant myocarditis. It is important to consider this remote but a real entity, particularly in areas of high prevalence, in view of its favourable prognosis with prompt recognition and timely Anti-tuberculous treatment.

Case Report

A 30 year old male presented to the emergency room with history of high grade fever, cough and dyspnea for a week with worsening orthopnea and paroxysmal nocturnal dyspneafor two days. Clinically he was febrile, in severe congestive cardiac failure, pitting bilateral pedal edema, intense diaphoresis, Pulse rate 120/min and B.P 90/70 mm hg, intense respiratory distress, RR 40/min, SPO_2 48% in room air and 88% with 10 liters of O_2. Cardiovascular examination showed raised JVP, cardiac apex in 6th intercostal space lateral to mid clavicular line, a prominent S3 gallop, soft mitral regurgitation murmur, bilateral extensive crepitation's and rhonchi. No pericardial rub or knock was present. Other systems were unremarkable except for tender congestive hepatomegaly. His past medical history was noteworthy for smear positive pulmonary tuberculosis two months ago and was put on Anti tuberculous therapy in TB Sanatorium which he subsequently stopped after a few weeks on his own due to malaise, fatigue and dyspepsia. There was no history of Diabetes mellitus, Autoimmune disorder, drug addiction, Sexually transmitted disease's or alcoholism.

Clinical differentials considered were Acute fulminant myocarditis which could be commonly Idiopathic, Viral, rarely Drug induced, Giant cell myocarditis and remotely Tuberculous Myocarditis. Tuberculous constrictive pericarditis which is more common is clinically unlikely in our patient as it is chronic in presentation, may have aprominent pericardial knock and usually does not have extensive crepitation's in the chest.

Following are the results of investigations. Hb 15.5 gm/dl, Total leukocyte count 16,500 with 76% neutrophils,15% lymphocytes, No eosinophils or monocytes, platelet count 2.6 lakhs and ESR was elevated to120mm/hr.Total CK and CK-MB were raised. HIV serology was negative. ABG revealed type 1 respiratory failure. Chest X-ray showed cardiomegaly with severe pulmonary edema (Fig 1) and chest X raydone outside two months ago showed bilateral extensive pulmonary tuberculosis (Fig 2). ECG on presentation (Fig 3) showed normal sinus rhythm with sinus tachycardia and nonspecific st-t changes and subsequently showed anteriort wave inversion indicating ongoing myocarditis (Fig 4)

Fig 1:chest x ray showing cardiomegaly with pulmonary edema

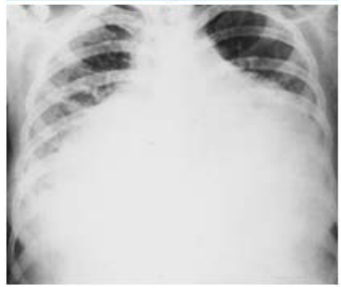


Fig 3 initial ECG showing sinu tachycardia

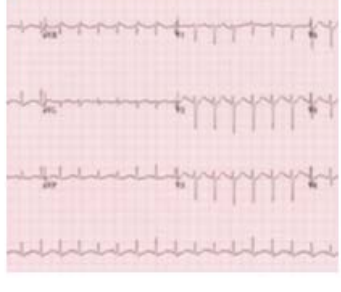


Figure 2:chest x-ray showing bilateral extensive pulmonary tuberculosis

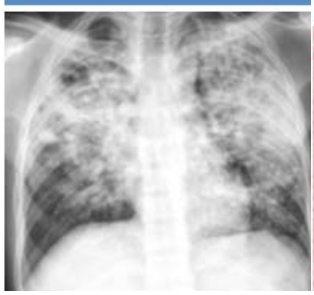
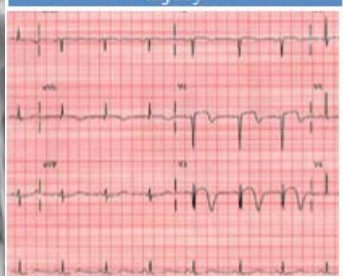


Fig 4: subsequent ECG showing anterior t wave inversion indicating ongoing myocardial injury



Echocardiogram revealed Dilated Left atrium and Left ventricle, prolonged EPSS, Moderate Mitral regurgitation, severe LV dysfunction, Global hypokinesia with 20% Ejection fraction and mild pericardial effusion. There was no evidence of constriction, pulmonary hypertension, mass, vegetation's or shunts. Fig (5, 6, 7 and 8)

Fig 5:parasternal long axis view showing dilated left atrium and left ventricle and mild pericardial effusion

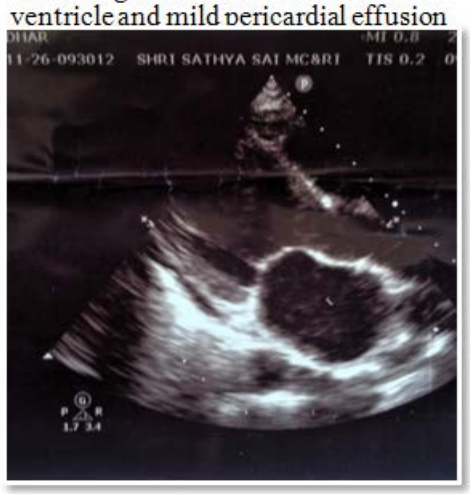


Fig 6:parasternal short axis view showing dilated left ventricle

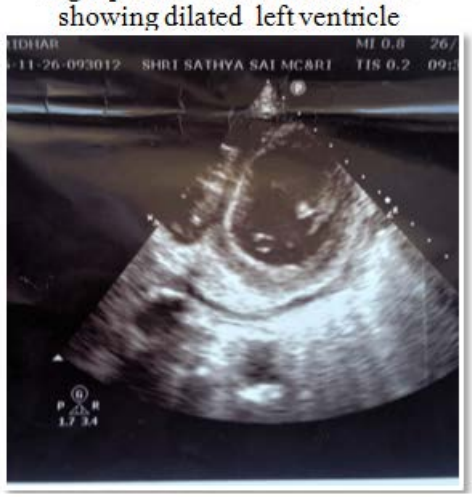


Fig 7: showing restrictive mitral filling pattern

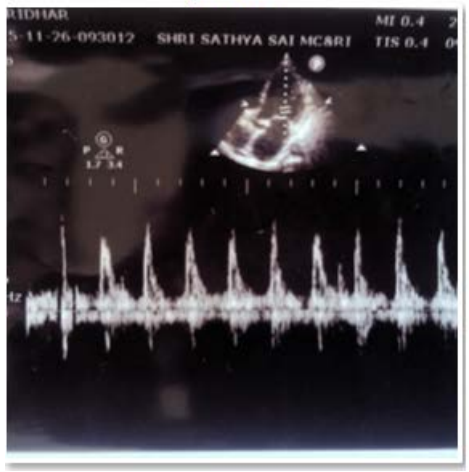
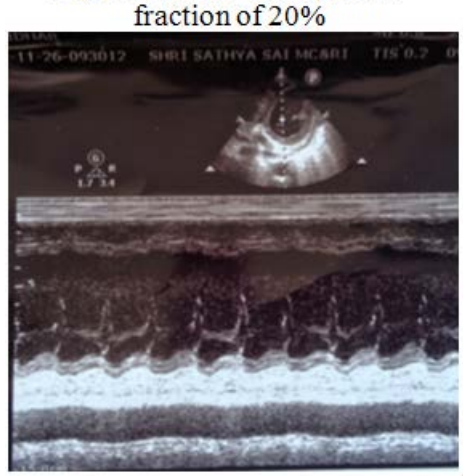


Fig 8:m-mode with ejection fraction of 20%



Patient was shifted to coronary care unit and was started on Inotropes, Vasodilators, Diuretics, Vasopressors, Digoxin, Enalapril, prophylactic LMWH and was restarted on Antituberculous therapy. After 24 hours, his clinical condition stabilized andwas less breathless with stable vitals, Respiratory rate 18/min and SPO_2 97% in room air. The following differentials for Acute fulminant myocarditis were considered and elaborated in the subsequent discussion. Tuberculous myocarditis, although rare it is a definite entity and clinically considered in our patient due to presence of active parenchymal tuberculosis, presenting with myocarditis and with no other obvious predisposition to it. Drugs like INH, Pyrazinamide and Rifampicin[2] are well known to have caused myocarditis albeit very rare. Idiopathic giant cell myocarditis is rare, aggressive and often diagnosed post mortem[3].

Subsequently after few days the patient was referred out to confirm our diagnosis with myocardial biopsy, serum viral titers, cardiac MRI, the results of which are awaited.Anti-

tuberculous therapy was started in view of active pulmonary tuberculosis ,strong clinical suspicion of TB Myocarditis, its association with sudden cardiac death and very low likelihood of ATT altering the investigation results.

Discussion

Tuberculous myocarditis was first repoted in 1664 by Maurocordat and the second case after 97 years in 1761 by Morgagni[4]. As such cases of tuberculous myocarditis reported in literature are few and far between with maximum reported live cases of about four.Tuberculosis commonly affects pericardium and usually has a favorable course with treatment. Myocardial involvement is extremely rare and can present even as sudden cardiac death[5]. Presentation with fulminant myocarditis as in our patient has been reported in about four cases[6] .Tuberculous infection of the myocardium usually occurs through hematogenous, retrograde lymphatic or spread from endobronchial tuberculosis. Horn and Saphir[7] described three histological types of myocardial tuberculosis. Nodular tubercles (granulomas), miliary tubercles of the myocardium and Diffuse infiltrative types.

Myocardial tuberculosis can manifests as Acute fulminant myocarditis, supraventricular arrhythmias, ventricular tachycardia, varying degrees of conduction block, sudden cardiac death, ventricular aneurysms, pseudo aneurysms, aortic insufficiency, coronary arteritis. Myocardial tuberculosis almost always shows evidence of tuberculosis at other sites as in our patient[8]. Even though myocardial involvement by tuberculosis is rare it should be suspected as a cause of congestive cardiac failure in any patient with features of tuberculosis. Prognosis of tuberculous myocarditis is generally favorable[4].

Antituberculous drugs INH, rifampicin and pyrazinamide can cause eosinophilic myocarditis. Presenting characteristics may include a rash, peripheral eosinophilia and multiorgan dysfunction. Myocardial involvement varies but usually does not result in fulminant heart failure as in our patient. Corticosteroids and drug withdrawal usually resolve this syndrome[9].

Idiopathic giant cell myocarditis represents a rare but distinct entity of unknown origin with unfavorable prognosis. Typical IGCM affects young adults and histology shows myocyte damage, eosinophils, foci of lymphocytic infiltrates, and multinucleated giant cells. Although the age and severity of cardiac symptoms in our patient makes IGCM a likely diagnosis, presence of active parenchymal tuberculosis, quick response to standard cardiac care, absence of other extra cardiac autoimmune manifestations in the patient makes it difficult to be certain without Endomyocardial biopsy and myocardial gene expression profiling. However Endomyocardial biopsy is not without pitfalls like availability, poor sensitivity and intra-observer variability and should not be the sole criteria for diagnosis[10]. These factors and clinical scenario of active parenchymal pulmonary tuberculosis led us to prudentially manage this patient empirically as Tuberculous Myocarditis with Anti-Tuberculous therapy with which he stabilized and improved.

In summary here we present a patient with extensive bilateral active pulmonary tuberculosis who presented with clinical, electrocardiographic and echocardiographic features of Acute fulminant myocarditis likely Tuberculous and is recovering on standard cardiac care and Anti-tuberculous therapy.

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