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A CASE ON PERICARDIAL EFFUSION SECONADARY TO TUBERCULOSIS

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ABSTRACT

Background: Tuberculosis (TB) is responsible for approximately 70% of cases of large pericardial effusion and most cases of constrictive pericarditis in developing countries. TB is a dangerous disease with a mortality of 17% to 40%. **Case Prsentation**: We report a case of 22 years old male of pericardial effusion with high grade fever, breathlessness on exertion, orthopnea, non-productive cough, decreased appetite and weight loss. Emergency pericardiocentesis in view of severe pericardial effusion was done and 1400 ml pericardial fluid was drained. Patient was initiated on Antitubercular Treatment (ATT) along with corticosteroids, in view of elevated Adenosine Deaminase (ADA) levels in exudative pericardial fluid. **Conclusion**: TB pericarditis is a type of extra-pulmonary TB. Prompt diagnosis and effective management can be helpful in reducing the progression of the pleural effusion to the cardiac tamponade.

KEYWORDS: Tuberculosis, pericardiocentesis, pericardial effusion.

INTRODUCTION

Pericardial effusion is an abnormal accumulation of fluid in the space between heart and pericardial sac, which will slowly progress and the pericardial sac will stretch to accommodate the fluid and eventually cardiac tamponade will occur.

Tuberculosis is responsible for approximately 70% of cases of large pericardial effusion and most cases of constrictive pericarditis in developing countries. TB is a dangerous disease with a mortality of 17% to 40%.[1] Sub-Saharan Africa and the part of Asia carry largest number of

TB pericarditis patients, in which TB is responsible for majority of clinically significant pericardial effusion (50-70% in non-HIV infected cases and 90% in HIV-infected cases).[2,3]

CASE PRESENTATION

A 22- year old male patient presented on 22nd august 2019, with high grade fever and evening rise of temperature associated with chills and rigors for the past 3 weeks, breathlessness on exertion since 10 days, orthopnea, non-productive cough, decreased appetite, weight loss (2kgs in 1 month). Patient had no history of TB, diabetes mellitus, systemic hypertension or immunosuppresion.

On admission patient was conscious, oriented, febrile (102 F), pulse rate of 122 b/min, BP 90/60 mmHg. Systemic examination shows CVS: S1S2+, muffled heart sounds, RS: B/L air entry+, PA: soft, CNS: NFND.

Laboratory investigations revealed Hb: 11.2 g/dl, PCV: 34.1%, WBC: 8620 cells/cumm, Platelets: 308000, PT: 11.68, INR: 1.71, APTT: 29.1, Albumin 3.4 g/dl, Globulin: 4.5 g/dl. ECG revealed sinus tachycardia with low voltage complexes as shown in figure 1. Chest X-ray revealed money bag appearance with normal lung field suggestive of pericardial effusion as shown in Figure 2.

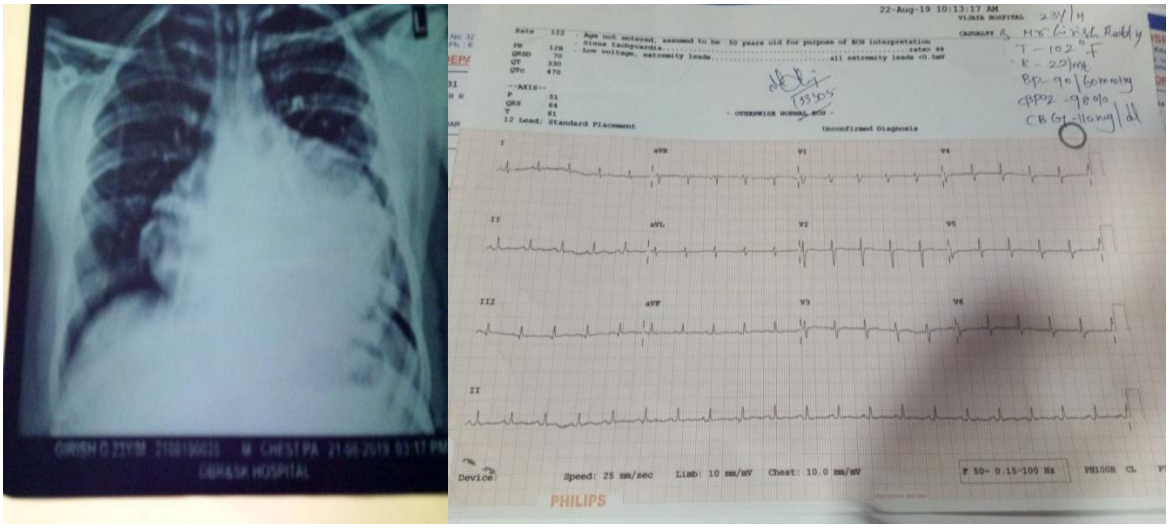

Figure 1 & 2: Chest X-ray & ECG.

2 D ECHO shows pericardial effusion amounting to tamponade, RV systolic collapse, dilated inferior vena cava and hepatic veins, no Regional Wall Motion Abnormality (RWMA), adequate LV function(EF:55%). USG abdomen reveals hepatomegaly, minimal bilateral pleural effusion.

On 23/8, in view of large pericardial effusion, pericardiocentesis was done. About 1400 ml high coloured (black grape juice) fluid was drained and sent for analysis. Fluid analysis revealed high levels of ADA. Mantoux test was positive. On the basis of above findings, patient was started on ATT i.e. Isoniazid(INH) 300 mg, Rifampicin 450 mg, Pyrazinamide 1250 mg and Ethambutol 750 mg. Repeated 2 D ECHO was taken after pericardiocentesis shows moderate pericardial effusion posterior with stared, mild pericardial effusion anteriorly, normal LV function and normal right ventricular function. Patient symptomatically improved and got discharged on 28 august.

DISCUSSION

Tuberculosis pericarditis is seen in 1-8% of patients with extra pulmonary tuberculosis.[4] It is potentially a dangerous condition. Pericardial effusion is mainly due to the hypersensitivity to the tubercular protein.[5] The clinical presentation can be variable, ranging from asymptomatic effusions identified incidentally, chest x-ray to life threatening emergencies associated with cardiac tamponade.[6] The accurate diagnosis is often challenging. The various tests available to confirm tuberculous pericarditis were mycobacterium Tuberculous screening, ADA levels, Polymerase Chain Reactions (PCR) tests, pericardial IFN-g, absolute lymphocyte count, detection of mediastinal lymph nodes on Computed Tomography, 2D Echo etc.

Tuberculous pericarditis presents clinically in 3 forms i.e. pericardial effusion, constrictive pericarditis, and a combination of effusion and constriction. A “definite” diagnosis of tuberculous pericarditis in tuberculosis endemic countries is based on the demonstration of tubercle bacilli in pericardia fluid or on a histological section of the pericardium; “probable” tuberculous pericarditis is based on the proof of tuberculosis elsewhere in a patient with otherwise unexplained pericarditis.[7]

In this study, the patient has developed pericardial effusion secondary to tuberculosis, came with high grade fever with evening rise associated with chills and rigors, breathlessness, orthopnea, non productive cough, decreased appetite, weight loss as presentation. As similar to our case, the ECG was virtually abnormal in all cases of reported tuberculous pericarditis patients and also the ECHO shows large pericardial effusion.

Large pericardial effusion will eventually leads to cardiac tamponade, a complicated stage. In view of large pericardial effusion, emergency pericardiocentesis was done and about 1400ml pericardial fluid was drained.

Pericardial fluid high in ADA activity is of great value in early diagnosis of tuberculous pericarditis, with a sensitivity of 94% and a specificity of 97% for tuberculous pericarditis.[8] In our case the pericardial fluid analysis shown high levels of ADA, which indicates the presence of TB and ATT along with corticosteroids initiated.

CONCLUSION

TB pericarditis is a type of extra-pulmonary TB. Prompt diagnosis and effective management can be helpful in reducing the progression of the pleural effusion to the cardiac tamponade. Emergency pericardiocentesis is helpful in making fast and strong diagnosis as well as therapeutic intervention. ATT should be initiated as soon possible after the confirmed diagnosis with or without adjuvant steroid therapy.

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