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Prevalence of tubercular pericardial effusion in Rohilkhand region, a prospective study

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Abstract
Tuberculous pericardial effusion is common in India. Since, the introduction of HIV infection, the incidence of tuberculous pericardial effusion has increased not only in India but also the world over. It presents with the usual features of tuberculous infection (low grade fever, loss of appetite, loss of weight) along with features of pericardial effusion (dyspnea, cough and enlarged heart). The salient features of pericardial effusion are low volume pulse or even pulsus paradoxus, raised jugular venous pressure Kussmaul's sign, congestive hepatomegaly, ascites and edema over legs. In massive pericardial effusion, patient may go into cardiac tamponade when patient is breathless, restless with poor volume pulse (typical paradoxus), engorged neck veins, sinus tachycardia, fall in blood pressure. Urgent pericardial paracentesis is warranted to reverse the hemodynamic changes with improvement in symptoms and signs. Laboratory tests reveal raised absolute lymphocyte count, raised ESR, cardiomegaly on X-ray chest, low voltage and sinus tachycardia on ECG, Echo-free space seen between two pericardial layers on 2D-echo with heart floating in pericardial sac. Diagnostic pericardial paracentesis shows that pericardial fluid is lymphocytic exudate, with elevated ADA and IFN-g levels.

Keywords: Tuberculous Pericardial Effusion, paracentesis, Biochemical study and cytological study

Introduction
Tuberculosis (TB) is a leading cause of pericarditis in India and a number of other developing countries [1, 2]. This is in contrast to first-world countries where TB is responsible for less than 4% of acute pericarditis [1]. In spite of economic developments and the availability of effective chemotherapy, the burden of TB is increasing. This increase has been partially attributable to the spread of human immunodeficiency virus (HIV) and is characterized by an increasing proportion of extrapulmonary cases [3]. Tubercle bacilli may be isolated on culture, guinea pig inoculation and nowadays by PCR technique. For management of tuberculous pericardial effusion, antituberculous treatment with four standard drugs is started (HRZE). Pericardial paracentesis with needle or even open drainage is useful in relieving symptoms and rapid recovery. Adjunctive corticosteroids are useful for rapid recovery and for prevention of development of constrictive pericarditis.

Aims and objectives
To study the prevalence of Tuberculous pericardial effusion patients in a tertiary care Hospital in Rohilkhand region.

Material and methods
A prospective study was carried out at Rohilkhand Hospital, Bareilly. Patients presenting with pericardial effusions between June 2015 to May 2017 were enrolled. All patients gave written informed consent for participation in the study which was approved by the Ethics Committee of Rohilkhand. A pericardial tap was performed under echocardiographic guidance through a pigtail catheter and fluid sent for biochemistry, microbiology, cytology and differential white cell count. Patients were allocated to diagnostic groups based on pre-determined criteria.
Pericardial effusions were considered to be tuberculous in origin when diagnosed by one or more of the following criteria:

1. isolation of Mycobacterium tuberculosis from the drained pericardial effusion or pericardial biopsy specimen;
2. demonstration of granulomatous inflammation on histopathological examination of the pericardial biopsy sample;
3. presence of a lymphocytic pericardial exudate together with adenosine deaminase (ADA) level o40 U/l;
4. presence of a lymphocytic pericardial exudate associated with clinical features and a good response to anti-tuberculous chemotherapy; and/or
5. presence of a lymphocytic pericardial exudate with a positive sputum ZN stain and/or TB culture.

Inclusion criteria
All patients above 18 years of age.

Exclusion criteria
Patients having tuberculosis other than cardiac cause.

Result
A total of 322 consecutive patients were enrolled. Two hundred fifty seven patients (80%) were TB+/HIV-; 32 of these (10%) had TB+/HIV+, 3.2 patient (4%) had septic pericarditis and 3.2patient (1%) had a uraemic pericarditis, 12.88 patient (4%) had neoplastic, 3.2 patients (1%) had miscellaneous. Only two patients were not tested for HIV; these were classified as HIV negative (x) for the purposes of this study.

Recent developments in the diagnosis of TPE
Polymerase chain reaction (PCR)
PCR technology has been used for nucleic acid amplification in the diagnosis of tuberculosis [7]. PCR was performed with both pericardial fluid and tissue with IS6110 based primers specific for the *Mycobacterium tuberculosis* complex. They concluded that the overall accuracy of PCR approached the results of conventional methods, although PCR was faster. The sensitivity for pericardial fluid was poor and false positive results with PCR remain a concern [8].

Serodiagnosis
The potential for serodiagnosis of TPE is a solid phase antibody competition sandwich ELISA (SACT-CE) method [9]. A monoclonal antibody (CDC/WHO reference number IT39) which was raised against a specific epitope on the *M tuberculosis* 30 kDa antigen was used.

Adenosine deaminase and other markers
Adenosine deaminase levels are believed to reflect T-cell activity. The levels with TPE have varied from 10–303 U/l in one report [10]. The sensitivity was 94% and specificity 68% with a positive predictive accuracy of 80%. On the other hand it has been suggested adenosine deaminase and lysozome levels have to be taken into account in attempting an early diagnosis of TPE [11].

Nuclear imaging
Gallium-67 and indium-111 scintigraphy have been used in the diagnosis of TPE. The results are, however, non-specific and there are other cardiac causes of gallium-67 uptake [12, 13].

Chest computed tomography
Computed tomography allows study of the pericardium and the pleural changes.

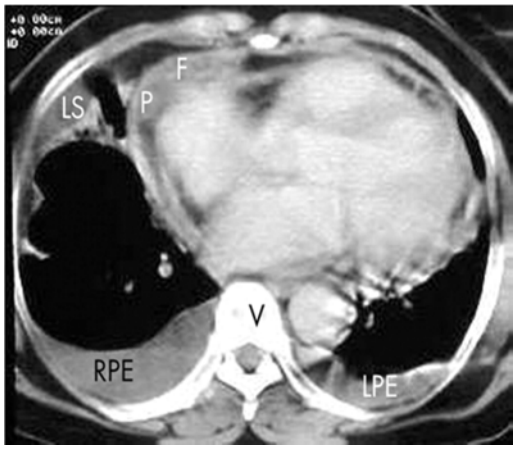


Fig 1: Pre-treatment chest computed tomogram after aspiration showing irregular pericardial thickening (P) and some fluid (F). Right (RPE) and left (LPE) pleural effusion with strands and loculation (LS) in pleural space.

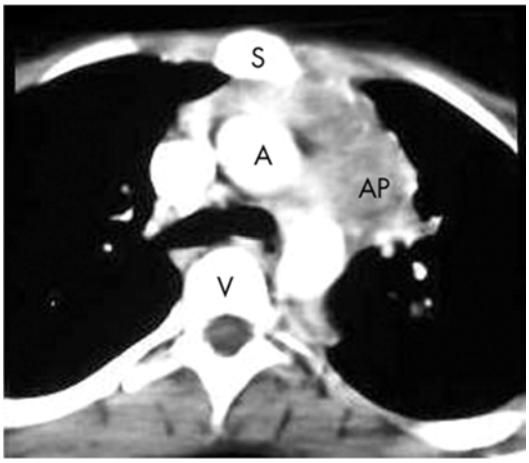


Fig 2: Pre-treatment chest computed tomogram after aspiration showing coalescing aortopulmonary (AP) nodes with matting and hypodense centre. S, sternum; A, aorta; V, vertebra.

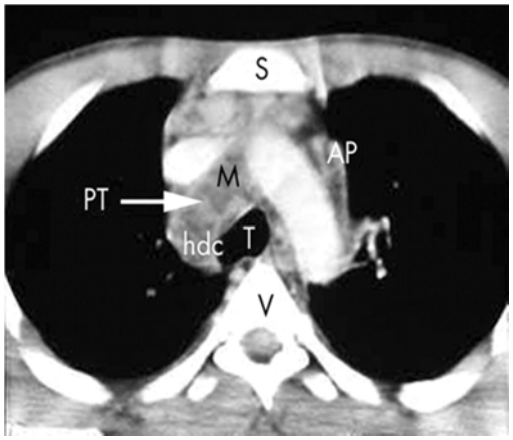


Fig 3: Pre-treatment chest computed tomogram of same patient as in fig 1 showing coalescing aortopulmonary (AP) and paratracheal (PT) nodes with matting (M) and hypodense centre (hdc). S, sternum; T, trachea; V, vertebra.

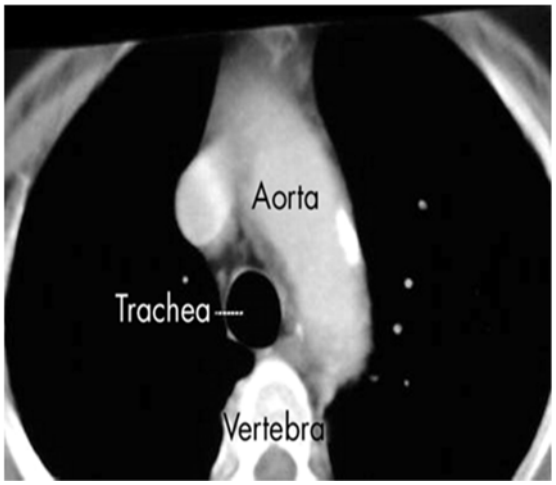
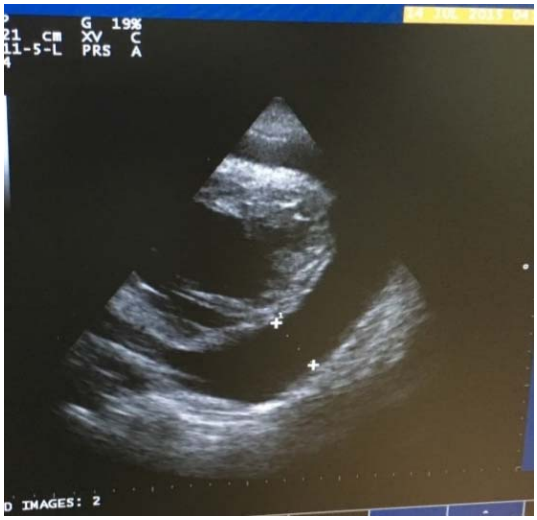
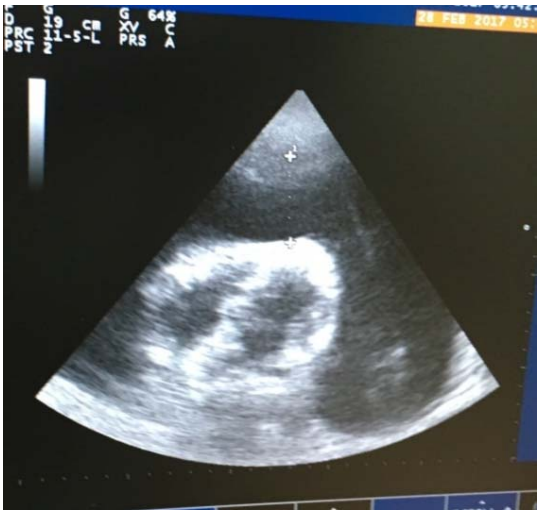


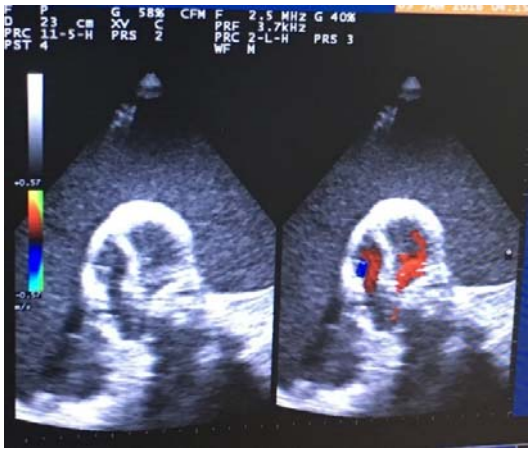
Fig 4: Chest computed tomogram in viral pericardial effusion after aspiration. No lymph nodes seen.

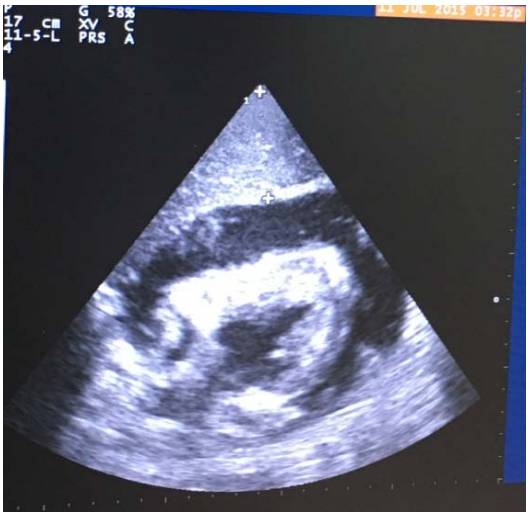
Echocardiography

The pericardial exudate is thick and fibrinous with a tendency to form adhesions and in some instances constriction. On echocardiography there are patchy deposits 4–8 mm in thickness with "fibrinous" strands criss crossing the pericardial space [14]. The appearance is quite characteristic.









Tuberculin skin test

The tuberculin skin test is done using the purfied protein derivative. A positive skin reaction is an induration ⩾10 mm and a strongly positive response is defined as one ⩾15 mm with or without excoriation of the skin. A positive tuberculin skin test has been found in all patients with TPE. In a recent publication the induration measured 16.4 [3] mm. A strongly positive tuberculin skin test is of value when associated with tissue granuloma without acid fast bacilli or when typical non-hilar mediastinal adenopathy is detected on chest computed tomography.

Pericardial fluid

The greatest value of the pericardial fluid is when *M tuberculosis* can be cultured as it is rare to find acid fast bacilli in a spun smear. The raised protein and lactic dehydrogenase values speak for an exudate. Lymphocytosis has been found in some studies [15] but not in others. All these non-specific findings can be found in chronic idiopathic pericardial effusion as well [16].

Culture of mycobacterium tuberculosis

M tuberculosis may be cultured from the sputum, tissue like the pericardium, pleura, scalene node, or other accessible enlarged nodes but such information is patchy. Recovery from the pericardial fluid has varied from 30%–100% [17]. The specimens were cultured in double strength Kirchner culture medium after bedside inoculation and also conventional culture in Stonebrink medium. *M*

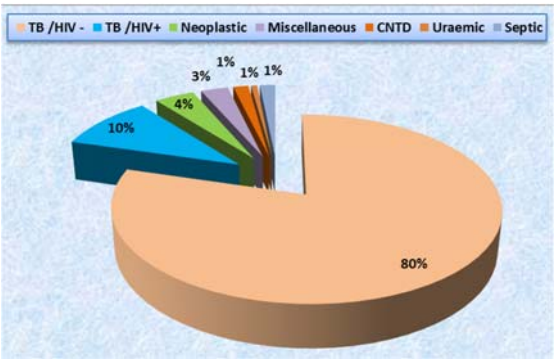
tuberculosis was cultured in all 10 patients, in Kirchner medium in nine and conventional in one.

Histological evidence

Like recovery of *M tuberculosis* from the pericardial tissue or fluid, histological evidence of a tuberculous granuloma with the demonstration of acid fast bacilli would be a definite diagnostic criterion. The typical granuloma is however not always found and the pericardial biopsy may show non-specific findings even when *M tuberculosis* is found in the pericardial fluid

Scalene node biopsy

The report on scalene node biopsy has been discussed earlier [18]. This would be a safe and relatively easy route to a specific diagnosis but so far there has been only one report.



Discussion

Pericardial effusions can result from a number of disease processes. The clinical presentation can be variable, ranging from asymptomatic effusions discovered incidentally by chest X-ray to life-threatening emergencies associated with cardiac tamponade. Numerous studies have reported on the aetiologies of pericardial effusions [4-6], however, the majority are retrospective reviews undertaken in first-world countries and suffer from inconsistencies and lack of thorough evaluation inherent in such a study method.

Conclusion

Tuberculosis is an important etiological cause of pericardial effusion in India. The incidence of tuberculous pericardial effusion has increased not only in India but also all over the world. Improved techniques for recovery of M. tuberculosis, the use of PCR technology, ADA levels, pericardial IFN-g, absolute lymphocyte count, detection of mediastinal lymphnodes on CT and more clearly defined observations on 2D-echo have improved the percentage of proper diagnosis. For management, apart from antibuberculous regime, pericardial pericardiocentosis or open drainage and corticosteroids (oral and/or intrapericardial) have improved the morbidity, mortality and reduced the sequalae of constrictive pericarditis.

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