Pericardial Effusion caused by Non-Tuberculous Mycobacteria in an Immuno-competent Patient
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Citation

Abstract
The finding of non-tuberculous mycobacteria (NTM) in a specimen is clinically significant when repeatedly isolated from sites which are normally unsterile, except from blood cultures or sterile sites where a single isolation is enough. Non-tuberculous mycobacteria have been very rarely isolated from pericardial fluid, a sterile closed cavity, in immuno-competent hosts. Here we describe one such extremely rare case who presented to us for persistence of pericardial effusion despite receiving a combination of anti-tuberculous drugs and steroids empirically for pericardial effusion for a period of six months.

INTRODUCTION
Atypical mycobacterial infection has been described in the medical literature since the mid 1950s [1]. A number of atypical mycobacterial infections exist; the most common forms of diseases being chronic pulmonary disease resembling tuberculosis (occurring mainly in adults), cervical adenopathy in children, skin and soft tissue infections, and disseminated disease in immuno-compromised persons [1]. Mycobacterium avium complex (MAC) and Mycobacterium scrofulaceum are associated in immuno-competent children with lymphadenitis. Table 1 summarizes the most important pathogens with common and less common sites of infection. [1]

Infections caused by NTM were not reported in the past, hence, only few systematically collected data about their frequency and distribution are available. Since NTM have been isolated from pericardial fluid very rarely [1], we report the isolation of NTM from pericardial effusion of an immuno-competent adult.

CASE REPORT
A fifty-seven year old Indian male was diagnosed as having moderate pericardial effusion on the basis of chest skiagram followed by echocardiography performed for complaints of non-anginal chest pain with breathlessness for three days. As there was no evidence of antecedent respiratory tract infection, and tuberculosis being one of the most common causes of pericardial effusion, four drug anti-tuberculous therapy with steroids were started. During follow-up, there was relief in clinical symptoms over a period of one-month; however, the effusion increased in amount as determined echocardiographically over a period of six-months despite adequate therapy for tuberculous pericarditis. This patient was referred to Himalayan Institute of Medical Sciences, Dehradun for the evaluation of the progression of the effusion.

There was history of a single episode of epistaxis with blood loss of approximately 250 ml blood 7 months prior to onset of chest pain and breathlessness; and arthralgias in small joints of hands, both shoulder and elbow joints without morning stiffness. The general and systemic examination was unremarkable. The skin manifestation of BCG vaccination was evident.

The chest skiagram revealed cardio-thoracic ratio of 0.6 with unremarkable lung fields. Echocardiographically, the pericardial effusion was estimated to be about 1000 ml; the fluid appeared viscous without any evidence of pericardial calcification, constriction or tamponade. His investigation profile was as follows- Hemoglobin 10.6 gm/dL, TLC 8640/cu mm, ESR 84 mm/1st hour, MCV 88 fL, MCH 31 pg, fasting blood sugar 88 mg/dL, post prandial blood sugar 130 mg/dL, blood urea nitrogen 26 mg/dL, serum creatinine 0.8 mg/dL, ELISA was negative for HIV-1 and HIV-2, serum was negative for rheumatoid factor and anti-nuclear antibody; liver functions were within normal limits with a serum albumin of 4.2 g/dL; serum TSH 3.9 µmol/L.
Pericardiocentesis was done and 100 ml of straw colored fluid was collected in a sterile bottle. Cytological examination of the fluid revealed a few red blood cells, TLC- 1600/ cumm with 2% neutrophils and 98% lymphocytes. Biochemical examination showed increased protein level (3.6 gm/dL) with a normal sugar level (60 mg/dL). Aerobic culture was sterile and direct smear for acid-fast bacilli was negative. Pericardial fluid carcino-embryonic antigen level was 3.1ng/mL (normal <5). The fluid was subjected to PCR DNA analysis for Mycobacterium tuberculosis (MYCO-SURE) that identified mycobacterium genus; however it was negative for Mycobacterium tuberculosis.

The patient was treated with a combination of rifampicin, isoniazid, azithromycin and ciprofloxacin. After 6 months of continuous treatment, the effusion has regressed and he has improved.

**DISCUSSION**

The development of a rapid radiometric mycobacterial detection system has allowed the distinction of Mycobacterium tuberculosis from other mycobacteria. The increased frequency of atypical mycobacterial infection stems from advances in the diagnostic procedures concerning the infection paired with the prevalence of mycobacterial disease in immuno-compromised patients infected with the human immunodeficiency virus (HIV). [1]

The virulence properties of non-tuberculous mycobacteria allow the microorganisms to establish infection in a variety of sites in immuno-compromised and immuno-competent patients. During disseminated Mycobacterium avium complex disease, some patients may not have fever and may not appear acutely or chronically ill [4] as was seen in this patient. Disseminated lesions have been reported in acquired immunodeficiency syndrome [5] and in patients with or without immune deficiency [6]. Our patient was immuno-competent as there was no evidence suggestive of immunodepression i.e. leukemia, diabetes, intake of steroids, other immunosuppressive drugs and opportunistic infections.

The pericardial effusion was suggestive of tuberculous origin, however, lack of response to conventional anti-tuberculous therapy raised suspicion about the etiology of the effusion being non-tuberculous. Other etiologies of pericardial effusion viz. viral pericarditis, malignancy, collagen vascular disease and hypothyroidism were excluded.

Our patient showed clinical and echocardiographic improvement with treatment. Although the culture and sensitivity testing was not performed, it is presumed that the lack of correlation between in vitro susceptibility and therapeutic efficacy in AIDS patients suffering from Mycobacterium avium complex infections [8,9] possibly holds true for non-tuberculous mycobacterial infections in immuno-competent patients.

The finding of NTM in a specimen is clinically significant when repeatedly isolated from sites which are normally unsterile, except from blood cultures [8] or a single isolation from sterile sites. Infection of pericardial cavity with NTM evidenced by PCR with a high diagnostic sensitivity (96%) and specificity (97-100%) [9]; unresponsiveness to standard anti-tuberculous therapy; and rapid resolution of pericardial effusion with therapy for atypical mycobacteria in this case should be considered to be of pathogenic importance; although, the species was not identified.

Hence, the identification of non-tuberculous mycobacteria from pericardial fluid in an immuno-competent adult merits special attention as only a single case in an immuno-competent child has been reported [4] till now to the best of our knowledge.

**References**

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