A case of massive pericardial effusion caused by Mycobacterium simiae

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Introduction: Mycobacterium simiae can cause disseminated infection in human immunodeficiency virus (HIV)-infected patients, mainly with involvement of pulmonary and reticulo-endothelial systems. Although this organism is also known to cause infections in non-HIV-infected individuals, to our knowledge there has been no report of pericardial effusion caused by M. simiae.

Case presentation: We describe a case report with massive pericardial effusion caused by M. simiae in a non-HIV-infected female patient, who presented with complaints of gradually increasing breathlessness and cough over a period of 1 month. Acid-fast bacilli were isolated from the pericardial effusion and subsequently confirmed as M. simiae by PCR-RFLP.

Conclusion: In an area where Mycobacterium tuberculosis infection is endemic, clinicians and microbiologists must be aware of the possibility of a non-tubercular mycobacterial infection that could be misdiagnosed as a tubercular infection.

Keywords: effusion; Mycobacterium simiae; pericardial.

Introduction

Mycobacterium simiae can cause disseminated infection in human immunodeficiency virus (HIV)-infected patients, mainly with involvement of pulmonary and reticulo-endothelial systems. A fatal disseminated M. simiae infection in a non-HIV patient has also been described (Balkis et al., 2009). Despite an extensive literature search, we did not find any report of pericardial effusion caused by M. simiae. To the best of our knowledge, this is the first report of massive pericardial effusion caused by M. simiae.

Case report

A 49-year-old female presented with complaints of gradually increasing breathlessness and cough over a period of 1 month. She was a known hypertensive, had a history of a left-sided stroke 6 months earlier and was on treatment for hypertension. There was no history of prolonged fever or significant weight loss. On physical examination, she was well nourished, conscious and oriented, with warm extremities. There were no congenital malformations. The liver was palpable 2 cm below the costal margin. The spleen was not palpable. Her blood pressure was 110/70 mmHg, and she had a pulse rate of 118 min⁻¹. Bilateral pedal oedema was present. There was no clubbing or visible cyanosis. No murmurs were audible. Heart sounds were muffled. There was bilateral air entry in her lungs. A chest X-ray showed massive cardiomegaly. A two-dimensional echocardiogram showed evidence of massive pericardial effusion with right atrial and right ventricular collapse. Left ventricular function was preserved. Her white cell count was 16000 mm⁻³, with 80 % neutrophils, 18 % lymphocytes and 2 % monocytes. Her haemoglobin level was 10 g dl⁻¹. An ELISA for antibodies to HIV-1 and 2 was negative. Serum protein levels were 7.98 g dl⁻¹, with an albumin:globulin ratio of 0.82. A diagnosis of massive pericardial effusion with cardiac tamponade was made. Pericardiocentesis was carried out with aseptic precautions through the subxiphoid angle under local anaesthesia. A 7F sheath was introduced and a pigtail catheter was passed through it into the pericardial space from where 1000 ml pericardial fluid was aspirated and drained. Microscopic examination of adequately cellular cytosmears of the pericardial fluid showed reactive...
mesothelial cells forming variable-sized clusters. Some of them were also singly dispersed. There was no evidence of any granuloma/atypia or malignancy in the smears studied. No acid-fast bacilli (AFB) were seen. The total cell count was 2050 mm\(^{-3}\), with 24% polymorphonuclear leukocytes, 60% lymphocytes and 16% mesothelial cells. The pericardial fluid was processed for AFB culture using an MB/BacT (bioMerieux) system and a concentrated smear did not show any AFB. However, AFB were grown on culture after 3 weeks of incubation. The growth was subcultured on plain Lowenstein–Jensen (L-J) medium, as well as on \(p\)-nitrobenzoic acid-incorporated L-J medium. Smooth colonies that became yellow after exposure to light were grown on plain L-J medium. The organism was niacin positive, catalase positive, was hydrolysed by Tween 80, did not reduce nitrate and also grew on \(p\)-nitrobenzoic acid-containing L-J medium.

DNA from the growth harvested from L-J slants was extracted by a procedure established previously using lysozyme (Sigma) and proteinase K (Bangalore Genei) (Telenti et al., 1993) in order to carry out PCR-RFLP for confirmed identification of the organism. A 439 bp fragment encoding \(hsp65\) was amplified using primers and a procedure described previously (Van Embden et al., 1993) followed by restriction analysis by \(Hae\)III. Fragment sizes were estimated by comparison with appropriate controls run in parallel with a reference strain of \(M.\) simiae and molecular mass marker in a gel documentation system using Quantity One software (Bio-Rad). The resulting fragments were matched with the \(M.\) simiae reference strain. The results are illustrated in Fig. 1: the PCR-RFLP result of the strain of \(M.\) simiae isolated from the pericardial fluid is in lane 80 and that of the reference strain of \(M.\) simiae is in lane 43. The patient was referred to another healthcare facility upon her request, before culture results were available, and was subsequently lost to follow-up.

**Discussion**

\(M.\) simiae can cause infections in diverse organs, and especially in the respiratory system, causing variable clinical manifestations (Baghaei et al., 2012). In HIV-uninfected adults, pulmonary manifestations are common, although lymphadenopathy, skin lesions and genitourinary tract involvement have also been described (Rynkiewicz et al., 1998; Onen et al., 2010). There has also been a report of a fatal disseminated \(M.\) simiae infection in a non-HIV patient (Balkis et al., 2009). An extensive literature search did not locate any report of pericardial effusion caused by \(M.\) simiae. Our patient was negative for antibodies to HIV-1 and -2. In an area like ours, which is endemic for tuberculosis, it is particularly important to be aware of the possibility of a non-tubercular mycobacterial infection that could be misdiagnosed as a tubercular infection. It is of particular importance to carry out mycobacterial cultures in order to be able to identify the \(Mycobacterium\) sp. and ascertain drug susceptibility. This is in view of the fact that the optimal treatment regimen for \(M.\) simiae infection has still not been determined. There are no published clinical trials for the treatment of \(M.\) simiae complex disease; however, clarithromycin and fluoroquinolones are recommended in the American Thoracic Society guidelines (Griffith et al., 2007). *In vitro* drug susceptibility results show that \(M.\) simiae is not usually susceptible to most antimycobacterial drugs, and high rates of resistance to rifampin (100%), amikacin (92%), clarithromycin (75%) and ciprofloxacin (30%) have been reported, although there were no recommended interpretive criteria for defining the susceptibility of the \(M.\) simiae isolates (Al-

![Fig. 1. PCR-RFLP of mycobacterial isolates targeting the hsp65 gene region digested with HaeIII. M, marker; lane Rv, \(M.\) tuberculosis (standard strain); lane 70, \(M.\) tuberculosi{}s isolate; lane 11, \(M.\) tuberculosi{}s isolate; lane J31, \(M.\) chelonae (standard strain); lane N2, \(M.\) fortuitum (standard strain); lane 50, \(M.\) fortuitum isolate; lane 43, \(M.\) simiae (standard strain); lane 90, \(M.\) fortuitum; lane 80, \(M.\) simiae isolated from pericardial fluid; lane 60, \(M.\) fortuitum isolate; lane 57, \(M.\) fortuitum isolate; U, uncut.](image-url)
Abdely et al., 2000; Huminer et al., 1993; Chung et al., 2009). We have reported this unusual causative agent of pericardial effusion in a non-HIV-infected individual in order to underscore the need to look for and identify non-tuberculous mycobacteria, even in individuals who have no obvious known risk factors for susceptibility to infection with non-tuberculous mycobacteria. This exercise is even more important in a tuberculosis endemic region in order to ensure that we do not wrongly diagnose infections with non-tuberculous mycobacteria as drug resistant Mycobacterium tuberculosis infections.

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References


