

Clinical Case Report

Chronic knee monoarthritis caused by *Mycobacterium chelonae*

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ABSTRACT

Extrapulmonary tuberculosis occurs in 20% of all patients with tuberculosis and tubercular arthritis occurs in 10% of those with extrapulmonary tuberculosis. Arthritis caused by *Mycobacterium tuberculosis* is not uncommon in India. However, arthritis caused by *Mycobacterium chelonae* has not been reported to the best of our knowledge. We report a patient with arthritis caused by *Mycobacterium chelonae* in whom the diagnosis was confirmed by smear and culture of acid-fast bacilli. Polymerase chain reaction of the synovial fluid using IS6110 was negative.

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INTRODUCTION

Extrapulmonary tuberculosis (EPTB) is seen in about 20% of all patients with tuberculosis and tubercular arthritis occurs in about 10% of such EPTB patients.¹

Conventional bacteriological techniques such as smear and culture have a low sensitivity while polymerase chain reaction (PCR) has been reported to be a more sensitive (73.9%) and rapid technique for the diagnosis of tubercular arthritis.^{2,3} However, species-specific PCR fails to detect tuberculosis caused by atypical mycobacteria. Atypical mycobacterial infections account for 0.5%–30% of all mycobacterial infections⁴ and involvement of the musculoskeletal system occurs in 5%–10% of patients with atypical mycobacterial infections.⁵ Musculoskeletal infection is acquired by contamination during surgical procedures, penetrating injuries or via haematogenous spread.^{6,7} The atypical mycobacterial strains often acquired by trauma are *Mycobacterium fortuitum*, *M. chelonae* and *M. marinum*.⁸ The prevalence of tuberculosis is high in India and atypical mycobacterial infections have also been reported⁹ from different organ systems. However, there is no report of tubercular arthritis caused by atypical mycobacteria. We report a patient with documented arthritis caused by *M. chelonae*.

THE CASE

A 34-year-old man attended the orthopaedic outpatient department of Choithram Hospital and Research Centre, Indore with complaints of pain and swelling of the left knee for 3 months. He gave a

history of trauma in 1991, for which his knee was immobilized for 1 month in a plaster cast. He had a similar episode of swelling of the left knee in January 1999. There were no associated symptoms at that time. The swelling lasted for 8–9 days and a synovial fluid aspiration was done. He had been treated with non-steroidal anti-inflammatory drugs (NSAIDs), allopurinol and sparflaxacin, with which his condition improved. He was also advised an arthroscopy which he did not undergo. He remained symptom-free for 3 years but in December 2002 he again had swelling of the left knee associated with pain and was treated with NSAIDs. No aspiration was done this time. In December 2005, before coming to our centre, he again had swelling of the left knee which was treated with NSAIDs and allopurinol. Uric acid levels had been estimated twice previously, and were 5.7 mg/dl and 5.2 mg/dl. His symptoms were not relieved. He had no history of pain in the other joints, morning stiffness, no bladder or bowel complaints, eye or skin involvement, no history of diabetes, hypertension, pulmonary tuberculosis and no family history of joint pain.

The synovial fluid sample aspirated in 1999 had revealed a glucose level of 90 mg/ml and uric acid of 5.7 mg/dl. The rheumatoid factor and smear for AFB were negative. His haemoglobin was 13.8 g/dl and white cell count 8000/cmm (47% polymorphs, 39% lymphocytes, 6% monocytes and 8% eosinophils). His ESR was 19 mm and the C-reactive protein (CRP) test was positive.

The synovial aspirate done by us showed a white cell count of 1150/cmm with predominance of polymorphs (70%), the glucose level was 90 mg/dl and proteins 6.6 g/dl. Adenosine deaminase (ADA), IgG and IgM antibodies against A-60, gamma interferon levels with specific ESAT-6 and CFP-10 antigens were also not raised and PCR using IS6110 primers specific for *M. tuberculosis* complex was negative. The antibody test, interferon response and PCR did not suggest an infection with *M. tuberculosis* complex.

AFB staining revealed the presence of short AFB. The AFB culture was positive at the end of 2 weeks and the isolate was identified as a rapid grower, which could also grow on MacConkey agar in 3 days. The isolate was further confirmed as *M. chelonae* by biochemical tests.¹⁰ Drug sensitivity testing indicated susceptibility to amikacin, co-trimoxazole, erythromycin, linezolid but resistance to tetracycline, cefotaxime and to all antitubercular drugs except ciprofloxacin.

The patient was given a knee support, advised quadriceps exercises and NSAIDs initially along with ciprofloxacin 500 mg b.i.d and co-trimoxazole b.i.d for 3 months. He responded well to treatment.

DISCUSSION

We report this case to generate awareness about the diagnosis of typical and atypical mycobacterial infections in patients with arthritis. The patient possibly acquired the infection during the episode of trauma in 1991. However, the diagnosis was delayed till January 2006. In patients with atypical mycobacterial infections the rise in ESR or ADA may not be striking and a predominance of neutrophils rather than lymphocytes may be observed. Scanty bacilli in the synovial fluid may not be detected on microscopy unless a sufficient amount of the fluid is centrifuged before preparing a slide. An AFB culture can be more rewarding.

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However, a high index of suspicion is necessary to diagnose atypical mycobacterial infections.

Clinically, musculoskeletal infections caused by atypical mycobacteria are not much different from those caused by *M. tuberculosis*^{6,11,12} although the course of atypical mycobacterial disease is more indolent.¹¹ The onset of atypical mycobacterial infections is with non-specific symptoms that include local pain and swelling, joint stiffness, low-grade fever, sweats, chills, anorexia, malaise and weight loss.^{13,14}

PCR has been shown to be more sensitive for the diagnosis of tubercular arthritis.^{2,3} However, in our patient the species-specific PCR was negative because he had atypical mycobacterial infection. A genus-specific PCR would be required for the diagnosis in such patients.¹⁵

Prescription of short term quinolones without ascertaining the aetiological agent can have limitations. Our patient had received a short course of sparfloxacin in 1991 following which he improved symptomatically.

M. chelonae and *M. fortuitum* are known to be resistant to most antitubercular drugs. The isolate of *M. chelonae* in culture was sensitive *in vitro* to ciprofloxacin and to conventional non-tuberculous drugs such as amikacin, co-trimoxazole, erythromycin and linezolid. The response to ciprofloxacin and co-trimoxazole was dramatic. However, further follow up is required. Atypical mycobacterial infection should be suspected in patients with chronic arthritis and efforts should be made to obtain a bacteriological diagnosis.

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