

Case Report

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Nontuberculous Mycobacterial Pulmonary Disease Secondary to *Mycobacterium Szulgai*

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Introduction

Mycobacterium szulgai was first defined in 1972 as a non-pathogenic mycobacterium. It is a rarely seen nontuberculous mycobacterium (NTM or mycobacteria other than tuberculosis (MOTT)) and evidence of disease usually accompanies its isolation from clinical specimens [1]. The nontuberculous mycobacteria are accounted for significant morbidity in the immunocompetent and immunocompromised host. *Mycobacterium szulgai* is a slow growing mycobacterium which is rare in nature and humans. It has been isolated from environmental sources such as aquariums, swimming pools, and tropical fish. Therefore, *Mycobacterium szulgai* disease has to be distinguished from pseudoinfection because of contamination of samples in the laboratory. A few cases have been reported since *Mycobacterium szulgai* was defined. Several of these cases were the patients with acquired immunodeficiency syndrome (AIDS) [2,3]. Three-drug therapy which is used in vitro susceptibilities as a guide for 12 to 18 months increases the probability for success [4]. Although it involves lungs most commonly, organ involvement and disseminated disease may rarely occur. Herein, we present a case of *M. szulgai* pulmonary infection developed 24 years after an episode of pulmonary tuberculosis with a discussion of the difficulty for an accurate diagnosis and a review of the relevant literature.

Case Report

Our female patient at the age of 39 referred to our center in July 2011 and she had 2-week history of cough, fever, night sweats, malaise, and a weight loss of 5-kg during the last 4 weeks. She had been treated for pulmonary tuberculosis in 1997 and in 2001. Her prior two episodes have not been confirmed by sputum culture results. On her chest examination; inspiratory rhonchi, fine rales and a few expiratory wheezes were found in the right infraclavicular area. On the left side there were no breath sounds. On the chest radiography, a right upper lobe calcific infiltrate was identified. Left lung was completely involved with fibro-cavitary infiltrated from previous tuberculosis. There was a new cavitary lesion in the fibrotic left base. In 1991 and in 2001, she was administered isoniazid, ethambutol rifampicin, and pyrazinamide for assumed pulmonary tuberculosis for six months and later developed pulmonary sequelae. Subsequently, she had repeated lung infections with rapid recovery 1-2 weeks after initiation of broad-spectrum antibiotics. Acid-fast bacilli (AFB) studies of her sputum at presentation was smear positive for AFB. As a result of positive AFB and new pulmonary cavity the patient was diagnosed as probable recurrent tuberculosis and started on isoniazid, rifampicin, ethambutol, pyrazinamide and streptomycin. In the second month of treatment, cultures were positive for *Mycobacterium szulgai* and confirmed by dot blot hybridization. No immunological deficiency that may cause NTM infection identified. Rapid drug resistance testing resulted as sensitive for rifampicin, ethambutol and streptomycin and resistant for isoniazid. She continued to take isoniazid, ethambutol, rifampicin, pyrazinamide and streptomycin for 2 months. In November 2011 treatment was reorganized with isoniazid, rifampicin, and ethambutol. After the first

month of the treatment the patients sputum production had resolved and she couldn't provide sputum for evaluation. At the end of third month of treatment there was no radiographic improvement. The patient was treated with 12 months of isoniazid, rifampicin, and ethambutol. By the respiratory function test she has been diagnosed for asthma and started to take inhaling steroids. No radiographic improvement occurred with treatment and a small pulmoner paranchimal tissue observed within the left lung. Left pneumonectomy was recommended and declined by the patient and she is currently being medically managed.

Discussion

M. szulgai, *M. simiae* and *M. malmoeense* are some of the mycobacteria which are difficult to diagnose. These organisms may cause long-term cavitary disease of the lung, leading to significant morbidity. *M. szulgai* is a slow-growing, scotochromogenic NTM. It was first described in 1972 and is responsible for n<0.5% of all human isolates of NTM [5-7]. *Mycobacterium szulgai* was named after Dr. T. Szulga, who played a role in the development of the lipid analysis method that allowed its early distinction from other mycobacteria [8]. Although the natural reservoir of the mycobacterim is unknown, it has been isolated from snails and tropical fish [9,10] and in water, including drinking water and hospital water supply [11,12]. Microbiological diagnosis can be difficult. Long culture period, potential contamination with environmental mycobacteria may be some of the reasons. *M. szulgai* may be misidentified with other NTM [13,14]. It was initially misidentified as *M. gordonae* and *M. avium-intracellulare-scrofulaceum* respectively. *M. szulgai* produces rough or smooth pigmented colonies after two to four weeks [15]. Prolonged incubation of cultured material for at least four-week is necessary. The most common nontuberculous mycobacteria to cause disease of the lung result from *Mycobacterium avium* complex. Classically, it causes an indolent tuberculosis like disease in the patients with chronic lung disease; however, it may occur up to 30% of women with no obvious previous lung disease who have nodular infiltrates and bronchiectasis when seen [6,16]. Similar to other mycobacteria, *M. szulgai* also leads to a wide spectrum of disease. Besides the initial report that described oleacronon bursitis, cervical adenitis in a child and pulmonary infection [8] a subsequent review [17] reported osteomyelitis, tenosynovitis, cutaneous infection, and disseminated disease. Recently, meningitis due to *M. szulgai* was suggested by the isolation of this organism from the cerebrospinal fluid of a 6-year-old girl who had altered sensorium and low-grade fever [18]. *Mycobacterium szulgai* lung disease usually manifests

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with long-term weight loss and cough and, radiography reveals cavitory upper lobe infiltrates. Thus, the illness appears similar to the traditional pulmonary disease resulted from *M. avium* complex or other nontuberculous mycobacteria and recurrent tuberculosis [4]. Our patient reported in this case has had 2 previous lung disease episodes treated as pulmoner tuberculosis. We couldn't reach her sputum culture results for prior lung diseases in 1991 and 2001. It is clear that culture results are very important for a proper diagnosis and treatment. Previously healed or active tuberculosis is a well-defined predisposition to nontuberculous mycobacterial diseases of the lung. Therefore, it was not surprising that the patient developed *M. szulgai* infection 24 years after treatment for pulmonary tuberculosis. In two of the cases reported first in the literature, death was attributed to *M. szulgai* infection, despite chemotherapy [9], but no such deaths have been described since then. Seventeen of 18 patients with *M. szulgai* pulmonary infection whose outcome is described [2,17,19] responded to antituberculous chemotherapy. Most of the reported patients were treated with at least 2 and usually 3 anti tuberculous drugs. Therapy was continued for 9 months to 2 years in successfully treated patients. Limited clinical experience and insufficient cases for therapeutic trials were two challenges in defining the optimal duration of treatment of *M. szulgai* infection. Based on the described experience and knowledge about the more common nontuberculous mycobacteria, we agree with Maloney et al. [17] on that at least 9 to 12 months of three-drug therapy beyond culture conversion, will reduce the probability for failure of the treatment or relapse. Total treatment duration of up to 2 years is reasonable. So far there is no any standard recommendation for the treatment. Generally, administration of triple therapies are reported to warrant a low rate of relapses and to allow sterilization of cultures within a mean of 3 months [20]; however occasional relapses are reported even several years later. Isoniazid (85%) is the most commonly adopted drug followed by rifampin (77%) and ethambutol (73%). Despite the *in vitro* efficacy of the drugs used, in the case presented here a 12 months treatment was needed for the eradication of infection.

Choice of antibiotic regimens is critically important for treatment of MOTT diseases. The role of drug susceptibility testing remains uncertain and controversial because the correlation between in-vitro susceptibility and in-vivo effectiveness has not been demonstrated except for clarithromycin-based treatment of MAC and rifampin-based treatment of *M. kansasii* [20]. Rapid drug resistance test results may indicate resistance to any antibiotic. According to the obtained results, our patients were resistant to INH. Many laboratories report the results of drug resistance test for *M. tuberculosis* as either high or low. The difference can be seen when those results are compared with the therapeutic dose of the antibiotic. Therefore, we kept using INH and completed the treatment successfully.

Conclusion

Since nonpathogenic bacteria usually emerge in *patients with a previous history of pulmonary tuberculosis* and in immunosuppressed patients, the patients should undergo immunological evaluation. In treatment, drugs should be chosen based on drug resistance test results. In cases of pulmonary tuberculosis, the dose of drugs should not be reduced without observing clinical improvement and before drug resistance test results are obtained.

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