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LETTERS TO THE EDITOR

Dear Editor,

PULMONARY DISEASE DUE TO RIFAMPIN-RESISTANT *MYCOBACTERIUM KANSASII* IN AN ADOLESCENT

A 16-year-old boy presented with a 2-month history of coughing, sputum production, fatigue and weight loss. He had been vaccinated with bacilli Calmette–Guerin at 2 months of age, and there was no history of exposure and tuberculosis infection in his family. Two months before, he had completed a 3-month treatment of rifampicin and doxycycline for brucella sacroiliitis. The chest radiograph showed right hilar adenopathy. Chest computed tomography revealed a 34 × 26 mm cystic-necrotic right hilar adenopathy with multiple millimetric nodules in bilateral lung parenchyma (Fig. 1). His tuberculin skin test was 22 mm. Sputum for acid-fast bacilli smear was negative.

Treatment was commenced for suspected tuberculosis with isoniazid (300 mg/day), rifampicin (600 mg/day), pyrazinamide (35 mg/kg/day) and ethambutol (15 mg/kg/day). However, the child's symptoms continued to worsen with progressive cough and weight loss. Two months later, two sputum samples cultures on Middlebrook 7H11 agar were positive for nontuberculous mycobacteria that later was identified as *Mycobacterium kansasii* by a commercial nucleic acid probe (AccuProbe; Gen-Probe, San Diego, CA, USA). Antimicrobial susceptibility testing revealed resistance to rifampicin (minimum inhibitory concentration >1 μg/mL) but susceptibility to all other agents tested (isoniazid, ethambutol, clarithromycin, moxifloxacin, trimethoprim-sulfamethoxazole and amikacin). Based upon antimicrobial susceptibility results, the treatment regimen was subsequently modified as three-drug regimen consisting of clarithromycin (500 mg twice daily), ethambutol (15 mg/kg/ day) and moxifloxacin (400 mg once daily). Negative sputum culture conversion was achieved at 2 months, and the treatment continued until sputum cultures were consecutively negative for 12 months. After treatment the patient fully recovered.

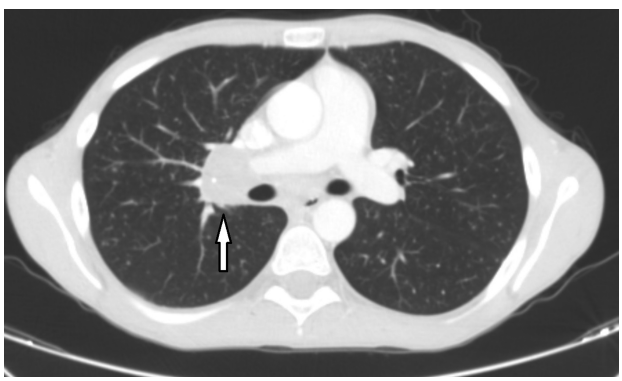


Fig. 1 Chest computed tomography showing a 34 × 26 mm cystic-necrotic right hilar adenopathy (arrow) with multiple milimetric nodules in both lungs.

During follow-up, all immunological investigations were found to be normal, including serum immunoglobulin levels, peripheral blood lymphocyte subsets, *in vitro* lymphoproliferative response to mitogens, dihydrorhodamine flow cytometry assay, flow cytometric analysis of interleukin-12Rβ1 cell surface expression on activated T cells, and Interferon-gamma cell surface expression on monocytes. Serological testing for human immunodeficiency virus was negative.

Although rare, *M. kansasii* can cause pulmonary disease that is clinically and radiologically indistinguishable from pulmonary tuberculosis in paediatric patients with no underlying lung disease or immunodeficiency.[1,2] It is usually sensitive to standard antituberculosis drugs expect pyrazinamide, and the recommend drug regimen is isoniazid, rifampin and ethambutol. Rifampin resistance has been associated with treatment failure, and should be considered in patients who had prior rifampin exposure,[1] as in our case. Consequently, species identification and antimicrobial susceptibility testing for mycobacteria is necessary for adequate treatment.

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