

Nasal *Mycobacterium kansasii* Infection in a Patient with Acquired Immunodeficiency Syndrome

Mycobacterium kansasii is one of the first species of mycobacteria other than tuberculosis whose role in human disease has been recognized (1, 2). It typically causes chronic lung disease closely resembling pulmonary tuberculosis, mainly in patients with underlying chronic obstructive lung diseases or various immunodeficiency disorders.

A 29-year-old male AIDS patient was referred to the Infectious Diseases Department of Careggi Hospital in Florence (Italy) in July 1991, complaining of persistent diarrhea and high fever. A former intravenous drug user, he had a history of chronic hepatitis B virus infection and had been HIV seropositive since 1988. His CD4+ count was below 10 cells/pl. Some months before he had developed interstitial pneumonia which resolved on treatment with trimethoprim-sulfamethoxazole. During hospitalization several sputa were positive for acid-fast bacilli (AFB). There was no radiographic or clinical evidence of chest infection, however examination of the nose and throat revealed the presence, in the right nasal cavity, of a granulomatous lesion covered with a mucopurulent exudate. Treatment with ethambutol, isoniazid and rifampin was undertaken and the patient was dismissed. In March 1992 the patient presented with diarrhea, high fever and a sinusitis-like pain, and was hospitalized again; the prescribed anti-mycobacterial therapy had been continued irregularly. The patient died in July 1992; an autopsy was not performed.

Rough colonies of a photochromogenic mycobacterium grew from all specimens (six sputa and two nasal swabs) on Lowenstein-Jensen medium. The organism was identified as *Mycobacterium kansasii* on the basis of the following findings (3): negative niacin test, nitrate reduction, positive heat-stable catalase test, positive semiquantitative catalase test, photochromogenicity, negative P-glucosidase test, Tween 80 hydrolysis, slow growth at 25°C and 37°C, no growth at 45°C, no growth on MacConkey agar, no tellurite reduction, negative arylsulfatase test, rough colonies, positive urease test, inhibition by NaCl (5 %), p-nitrobenzoate (500 µg/ml), thiacetazone (10 µg/ml), hydroxylamine (500 µg/ml), isoniazid (1 µg/ml) and oleate (250 µg/ml), and resistance to thiophene-2-carboxylic hydrazide (5 µg/ml). As a previously performed test for nucleic acid hybridization after culture (AccuProbe, GenProbe, USA) had scored negative for *Mycobacterium kansasii*, the strain was sent to the Unité de la Tuberculose et des Mycobactéries of the Institut

Pasteur, Paris (France), which confirmed the identification.

On testing in vitro by standard methods (4) the strain isolated during both the first and the second hospitalization was susceptible to ethambutol, rifampin, ethionamide and ofloxacin; moderately susceptible to amikacin, ciprofloxacin, isoniazid and streptomycin; and resistant to p-aminosalicylic acid, norfloxacin, kanamycin and pyrazinamide. Pyrazinamide susceptibility testing was performed with the Bactec system (Becton Dickinson, USA) using the procedure developed for *Mycobacterium tuberculosis*.

Several points are of interest in this case. In the pre-AIDS era, although frequently found in respiratory specimens, *Mycobacterium kansasii* was only rarely associated with extra-pulmonary infections, mostly cutaneous lesions with or without lymphonodal involvement (5-9). Almost all reported cases of *Mycobacterium kansasii* infection in HIV-infected patients have been characterized by pulmonary disease, with extra-pulmonary dissemination in about half (10-19); granulomatous lesions have been rare. Exclusively extra-pulmonary infections caused by *Mycobacterium kansasii* are extremely rare: there is one report each of a cutaneous abscess (20), osteomyelitis and dissemination without pulmonary involvement (17).

In our case a granulomatous tumefaction was present without evidence of planetary involvement or dissemination. Microscopic examinations and feces cultures on three occasions and the single blood culture were all negative. The mycobacteria isolated from sputum were probably of nasal origin, as suggested by the much higher concentration of AFB in the mucopurulent exudate over the lesion, which also yielded more colonies on culture.

The fact that the strain involved in the present case did not hybridize with the commercial probe prompted us to reconfirm its phenotypic identification as *Mycobacterium kansasii* according to the current definition. On the other hand, it cast doubts on the sensitivity of the probe. We are currently investigating several other probe-negative strains to test the hypothesis that they may be taxonomically distinct.

Several geographic areas are known to be endemic for *Mycobacterium kansasii* (21), and the number of HIV patients infected with it is rising (19). In the Florence area the isolation of *Mycobacterium kansasii* is unusual in comparison with various other mycobacterial species particularly *Mycobacterium xenopi* (22).

As shown in the review by Carpenter and Parks (23) there is a remarkable paucity of *Mycobacterium kansasii* infections in AIDS patients: even in an endemic area they found only nine cases compared with 160 cases of disseminated *Mycobacterium avium* complex infection and 50 cases of *Mycobacterium tuberculosis* infection. Thus, both the fact that *Mycobacterium kansasii* infection occurred in an AIDS patient in a nonendemic area and the site of infection are unusual aspects of this case.

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