Pulmonary Diseases due to *Mycobacterium Kansasi*ii in Japan

In Japan, the rate of pulmonary nontuberculous mycobacteriosis (NTM) (atypical mycobacterial diseases) in the total pulmonary mycobacteriosis is continuously increasing. In 1994 it reached 14.0% in newly admitted mycobacteriosis cases in 111 sanatoriums throughout Japan (1), and NTM became considered an important chest disease. This increase is mainly dependent upon the recent rapid decrease of tuberculosis in Japan, but partially on the substantial increase in NTM cases, especially those due to *Mycobacterium kansasii*. Nontuberculous mycobacteria include several species, and *M. kansasii* holds the second most important position after *Mycobacterium avium* complex in Japan.

The first Japanese case of *M. kansasii* was reported in 1963 by Kawabe et al. (2). Prior to 1980, the rate of this infection in total NTM was only 6% or 7%, and the cases were limited to the Tokyo and Kanagawa areas, but in 1980 the rate increased to over 20% and this infection began to appear in throughout Japan (3). Since 1980, the rate of *M. kansasii* cases has increased gradually; it reached 27.5% in 1985 and has maintained almost the same level since then (1, 4). The reason for the change in epidemiological status of infection of *M. kansasii* is unclear, but in part it depends on the ecological changes in Japan, and in part on the development of methods for identification of the species of mycobacteria.

The sex and age distribution of the cases of *M. kansasii* differ from those of *M. avium* complex; male/female ratio of the former is 37/1 and the average age is 46.9 years old, while they are 54/41 and 66.4 years old in the latter (4). This may indicate a more strong virulence of *M. kansasii* than *M. avium* complex. NTM frequently affects cases with previous lung disease, but *M. kansasii* affects more cases without previous diseases than *M. avium* complex and this also indicate a difference in virulence between these species.

The clinical course of the pulmonary disease due to *M. kansasii* was relatively poor, from the first report by Buhler and Pollak (5) in 1953 to the '60's report of Johanson and Nicholson (6), due to the low susceptibility of antituberculous drugs used for this bacteria at that time, but the development of rifampicin (RFP) and ethambutol (EB) changed the clinical course of this infection dramatically.

Ahn et al. (7) reported that in all 40 previously untreated pulmonary *M. kansasii* patients treated for 12 months with daily chemotherapy of isoniazid, RFP, EB and with streptomycin twice weekly for the first 3 months, their sputum converted to negative and relapse occurred in only one case 6 months after treatment. They considered that this regimen is sufficient for the treatment of this infection. The British Thoracic Society (8) recommended nine months of treatment with RFP and EB as an efficient regimen for this infection, by a large, prospective, multicenter study. These excellent results were dependent on the high susceptibility of this bacilli to RFP and EB, and the therapeutic effects of this regimen was ineffective in the resistant cases for RFP and EB, such as the case reported by Yamada et al (9), and the development of drugs for RFP- and EB-resistant cases is necessary, although quinolons and macrolides has been studied as possible drugs (10).

Fortunately, the rate of primary resistant cases for RFP is low in wild strains, Wallance et al (11) reported that the rate of RFP-resistant bacilli was only 4% of 464 isolates recovered in Texas between 1989 and 1992, and 90% of them had previously received RFP. In Japan, primary resistant cases for RFP are also rare (12). The possibility of the rapid decrease of the susceptibility of this bacilli for RFP or EB seemed low, since water was regarded as the most probable factor of transmission of *M. kansasii* (13), and the possibility of human to human infection seemed low even in patients with acquired immunodeficiency syndrome.

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References


