Pneumonitis Due to *Mycobacterium avium* Complex in Hot Tub Water

Infection or Hypersensitivity?

To the Editor:

We read with interest the paper by Embil et al (March 1997) because their first case resembled a patient we recently described in CHEST (January 1997). In our case, there was no doubt that we were dealing with an infection acquired from a hot tub. The patient’s condition deteriorated despite discontinuation of the use of the hot tub, and dramatically improved after appropriate antimycobacterial therapy. Cultures of the lung tissue yielded *Mycobacterium avium*, which was shown by restriction fragment length polymorphism testing to be identical to the organism isolated from the hot tub water.

Embil et al favor a diagnosis of hypersensitivity pneumonitis based on the history, lung biopsy histology, and spontaneous recovery. However, there was no serologic proof of an immunologic reaction to *M. avium*, and the microscopic features they describe and illustrate indicate mycobacterial infection rather than hypersensitivity pneumonitis. The pathologic diagnosis of hypersensitivity pneumonitis is based on the identification of patchy, nonspecific interstitial pneumonia with peribronchiolar accentuation, non-necrotizing granulomas and/or epithelioid histiocytes, and foci of bronchiolitis obliterans combined with the exclusion of infection. Figures 2 and 3 in their report show well-defined granulomas, whereas the lesions in hypersensitivity pneumonitis are described as “loosely formed and poorly circumscribed.” The identification of mycobacteria in histologic sections indicates invasion and multiplication of microorganisms in the tissue, which establishes this as an infection rather than an immunologic reaction. If nontuberculous mycobacteria could, in fact, cause hypersensitivity pneumonitis, it is strange not to have had previous examples, considering their ubiquitous presence.

The recovery, with little or no treatment, in this patient and the other cases reported by Embil et al, is unusual for *M. avium* infection. The normal immunity of the patients may have played a part, and the condition may be analogous to primary infection in tuberculosis, in which the door is shut on further progression by the development of cell-mediated immunity, exemplified by the presence of granulomas.

We agree with Embil et al that the cases we have described point to the need for caution in the use of hot tubs.

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References


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We acknowledge the points that Kahana and Kay make in their letter. However, we are unable to be as confident as to what was occurring in the patients we reported as they were in their case report; hence, our title “Hypersensitivity pneumonitis or infection?” Despite effort, we were unable to produce an adequate *Mycobacterium avium* complex (MAC) antigen for serologic testing and so we could not confirm immune reaction to MAC. On the other hand, lack of serology has not refuted the diagnosis of hypersensitivity pneumonitis in other reports. The editorial by two experienced workers in the field thought our suggestion of hypersensitivity pneumonitis to be plausible. Despite the different approaches and conclusions, each of these papers poses an intriguing aspect of the illness associated with MAC. Both are very different from the traditional disease described with MAC. We believe that our interpretation is most compatible with the discussion in the clinical pathologic exercise reported in 1996, which left the participants with the dilemma of infection or hypersensitivity. After all, the immune reactions of the lung which protect and those that may produce hypersensitivity are two faces of the same coin.

The basis for the suggestion of hypersensitivity pneumonitis is supported by the observation that cases 1 and 2 had symptoms over a number of months, with the former relapsing on further exposure to the hot tub and the latter acutely worsening on heavy exposure. This is very suggestive of a hypersensitivity reaction. In their letter, Kahana and Kay make much of the interpretation of the histology in our case—as they know it is as yet hard to be dogmatic in this area, since a range of findings have been reported in hypersensitivity pneumonitis. In Farmer’s Lung, organisms, admittedly spores, are seen in the biopsy—we are unable to judge multiplication of the MAC organism in the lung on our biopsy. If these cases were infections, presumably they were analogous to primary infection, and the widespread nature of the lesions was due to the dispersal of the inoculum. Or, alternatively, if an infection was smoldering in the lungs of cases 1 and 2, were the exacerbations of symptoms with further exposure to the hot tubs due to further infection or an immune response, hypersensitivity, to a new exposure?

We agree with Kahana and Kay that considering the ubiquitous presence of MAC, it is strange not to have previous examples of hypersensitivity pneumonitis, but this also applies to infections with *M. avium* such as they describe. In other studies of humidifier lung syndrome, there is little evidence that mycobacteria have been sought. It is unusual for such a case as described by Kahana and Kay to be so exceptionally responsive to treatment, particularly when the organism was resistant in vitro to the therapy that was initially selected, in an infection that had progressed. Was an immune reaction contributing to the disorder and symptoms? Clearly, the two papers raise more questions than answers. Could the responses that were observed be due to differing virulence of the MAC isolated, the magnitude of the