

## INCREASED SUPPRESSOR CELL ACTIVITY IN A PATIENT WITH *MYCOBACTERIUM AVIUM-INTRACELLULARE* PULMONARY DISEASE AND HYPOGAMMAGLOBULINEMIA

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THE SO-CALLED ATYPICAL mycobacteria are saprophytes under most conditions and generally can be found in the environment. Unlike *Mycobacterium tuberculosis*, most people are exposed to atypical mycobacteria but only a few develop diseases.<sup>1</sup> Host factors seem to play an important role in the development of disease with atypical mycobacteria. Most of the cases of *M. kansasii* and *M. avium-intracellulare* disease occur in patients with underlying pulmonary disease such as pneumoconiosis, previous tuberculosis, chronic obstructive lung disease, chronic aspiration and bronchiectasis.<sup>2</sup> Immunodeficiency has also been implicated, particularly cellular immunity impairment, as an underlying host factor predisposing to atypical mycobacterial infection.

Increased suppressor cell activity has been found in humans with tuberculosis<sup>3,4</sup> as well as a variety of other diseases including leprosy,<sup>5</sup> disseminated fungal infection,<sup>6</sup> solid tumors,<sup>7</sup> Hodgkin's Disease,<sup>8</sup> sarcoidosis,<sup>9</sup> aplastic anemia,<sup>10</sup> multiple myeloma and common variable hypogammaglobulinemia.<sup>11</sup> It is therefore tempting to speculate that an immunoregulatory imbalance may be the basis of a host factor predisposing to mycobacterial diseases such as *M. tuberculosis* and *M. leprae* and possibly also the atypical mycobacteria. The results of an investigation of the suppressor cell activity in a patient with *Mycobacterium avium-intracellulare* pulmonary disease associated with common variable hypogammaglobulinemia and bronchiectasis are reported here.

### Case Report

The patient is a 63-year-old, non-smoking woman who was in good health until 30 years ago when she developed frequent respiratory infections, chronic cough and sputum production. Bronchiectasis was diagnosed in 1968 and the patient underwent right upper and middle lobe

resection. Hemoptysis was noted in 1977 and subsequently pulmonary disease with *Mycobacterium avium-intracellulare* was diagnosed. The patient was treated initially with a three-drug regimen of isoniazid, ethambutol and rifampin and was referred to National Jewish Hospital and Research Center, Denver, Colorado, for further evaluation. The patient's symptoms included intermittent chronic productive cough with yellowish sputum averaging one-half cup per day. The family history indicated that her father died at the age of 34 of pneumonia and her mother died of cancer of the liver. Her two sons are healthy. The physical examination revealed a thin 63-year-old deaf woman with tracheal deviation to the right, decreased breath sounds on the right and scattered rhonchi bilaterally. The rest of the examination was unremarkable.

A chest X-ray revealed thoracic kyphoscoliosis, diffuse bilateral parenchymal disease with large cavities in the right upper lung field with pleural thickening. The right lower lung field and left mid lung field showed interstitial disease. The mediastinum was shifted from the left to the right (Figure 1).

Pulmonary function testing indicated a mild restrictive pattern with air flow obstruction. The routine blood count and blood chemistries were unremarkable. An alpha I antitrypsin level was elevated at 410 mg% (N: 196 ± 41 mg%).

The sputum culture was repeatedly positive for large numbers of *M. avium-intracellulare* organisms. Despite six months' intensive treatment with a six-drug regimen (isoniazid, ethambutol, rifampin, cycloserine, ethionamide and capreomycin) the patient remained culture positive.

### Materials and Methods

Serum immunoglobulins (IgG, IgA, IgM) were determined by nephelometry using monospecific antisera.<sup>12</sup> IgE was determined by Prist Test and IgD by radioimmunoassay.

Enumeration of B lymphocytes was done with an

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