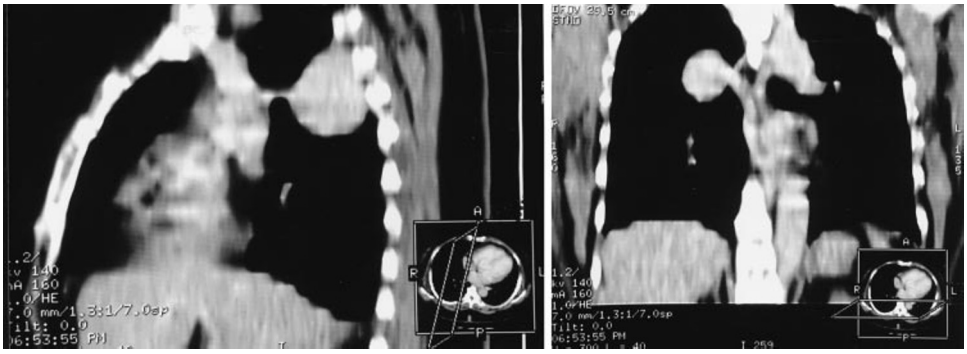

FIGURE 5. Reconstructive two-dimensional CT, sagittal (*left*) and coronal (*right*), views showing the mass connected the azygos vein and the superior vena cava (arrow).

variceal bleeding in patients with liver cirrhosis. It is known that the size and pressure of esophageal varices, which correlate with risk of hemorrhage, parallel azygos blood flow.[4] The volume of a compliant vessel increases more easily, according to the formula of elastic pressure: $C = \Delta V/\Delta P$, where C = compliance, ΔV = volume change, and Δp = pressure change. According to the Hagen-Poisseulle equation in hydrokinetics ($Q = \Delta P\ \pi r^4/8\ \mu L$, where Q = flow velocity, r = the radius of a tube, Δp = the pressure gradient within the tube, and L = the length of the tube), when there is constant pressure in the portoesophageoazygos venous system, an increase in the cross-sectional area contributed by the azygos vein dilation decreases flow within the system. This in turn reduces the risk of variceal bleeding.

In addition to reducing the flow in the system overall, another factor may also be significant in preventing bleeding. In general, portal flow varies diurnally, with peak flow occurring at midnight.[4] The azygos varix increases in size when the patient is recumbent, further reducing flow in the system. This phenomenon, as with the administration of propranolol at night, may play an important role in preventing variceal bleeding. The azygos varix thus may be present on chest radiograph before varices are diagnosed. In our patient, the mediastinal mass was seen 2 years before her first episode of variceal bleeding.

In summary, a mediastinal mass in a patient with cirrhosis of the liver may be a giant azygos vein varix. An awareness of this possible diagnosis should perform noninvasive imaging procedures prior to invasive tests. A comparison between supine and standing chest radiographs is useful in differential diagnosis. However, a chest CT if necessary may be adequate to make the diagnosis.

ACKNOWLEDGMENT: We thank Dr. Mary Jeanne Buttrey, Consulting Physician, Department of Internal Medicine, Mackay Memorial Hospital, for review and revision of the manuscript.

REFERENCES

1 Miguel G, Rosa MM, Albert M, et al. Idiopathic azygos vein aneurysm: a rare cause of mediastinal mass. Thorax 1999; 54:653–655
2 Strollo DC, Rosado-de-Christenson ML, Jett JR. Primary mediastinal tumors: part II: Tumors of the middle and posterior mediastinum. Chest 1997; 112:1344–1357
3 Dunn V, Miller FJ Jr. Mediastinal mass in a patient with liver disease. JAMA 1982; 247:1873–1874
4 Shigeo S, Kunihiro Y, Kayoko T, et al. Daily variation of azygos and portal blood flow and the effect of propranolol administration once evening in cirrhotics. J Hepatol 2001; 34:26–31

Hypersensitivity Pneumonitis Reaction to *Mycobacterium avium* in Household Water*

Theodore K. Marras, MD; Richard J. Wallace, Jr., MD, FCCP; Laura L. Koth, MD; Michael S. Stulbarg, MD;† Clayton T. Cowl, MD, FCCP; and Charles L. Daley, MD

Background: Hypersensitivity pneumonitis has been described with exposure to aerosolized *Mycobacterium avium complex* (MAC) in indoor hot tubs (hot tub lung).
***Objectives:* To describe a case of MAC-associated hypersensitivity pneumonitis-like reaction possibly from showering and review previous hot tub lung reports.**
***Methods:* For the case report, we investigated a patient with histologically diagnosed hypersensitivity pneumonitis and MAC-positive sputum culture findings. Mycobacterial cultures were obtained from his home and workplace. Isolates were typed using pulsed-field gel electrophoresis. For the review, MEDLINE and EMBASE were searched for hot tub lung reports, which were reviewed and summarized.**
***Results:* A 50-year-old man had progressive dyspnea and episodic fever and myalgias. Pulmonary function testing results revealed obstruction and impaired diffusion; a chest CT scan found diffuse, centrilobular, ground-glass nodules, and air trapping, and a lymphocytic alveolitis with an elevated CD4/CD8 ratio. Transbronchial biopsy showed multiple well-formed nonnecrotizing granulomas. Multiple respiratory samples and shower and bathtub specimens**

grew MAC, with matching pulsed-field gel electrophoresis *patterns. The patient changed from showering to tub bathing. Prednisone and antimycobacterial drugs were administered for approximately 1 year. His symptoms, pulmonary function abnormalities, and CT scan findings resolved. The literature review yielded 36 cases of hot tub lung. Clinical features included dyspnea (97%), cough (78%), and fever (58%). Pulmonary function testing showed obstruction (67%), restriction (55%), and impaired diffusion (75%). A chest CT scan showed ground-glass opacification (95%) and nodules (67%). Granulomas were well-formed in 95%. Treatments included discontinuation of hot tub use and prednisone, antimycobacterial drugs, or both. Outcomes were favorable.*

Conclusions: **A hypersensitivity pneumonitis-like reaction to mycobacteria can occur from exposures other than hot tubs. There are key differences between classic hypersensitivity pneumonitis and MAC-associated hypersensitivity pneumonitis. Antimycobacterial therapy may be required. The possibility of MAC hypersensitivity pneumonitis from showering raises potential implications in the investigation of patients with hypersensitivity pneumonitis.**

(CHEST 2005; 127:664–671)

Key words: alveolitis, extrinsic allergic; *Mycobacterium avium-intracellulare* infection; Mycobacterium infections, atypical; water microbiology

Abbreviations: BCG = bacille Calmette-Guérin; MAC = *Mycobacterium avium complex*; NTM = nontuberculous mycobacteria

Pulmonary nontuberculous mycobacteria (NTM) infection is an increasingly common problem,[1] usually associated with structural lung disease such as bronchiectasis or cystic fibrosis, but often also in otherwise apparently normal hosts. The classic response to pulmonary NTM includes both signs and symptoms of infection and necrotizing granulomas with acid-fast bacilli on histopathologic examination.[2,3] However, NTM can incite a range of histopathologic changes in the lung.[4] In particular, a hypersensitivity pneumonitis-like syndrome in patients exposed primarily to aerosolized *Mycobacterium avium complex* (MAC) has recently been described by a number of investigators.[5–14] The term *hot tub lung* has been used to describe these hypersensitivity pneumonitis-like cases because they have generally been associated with hot tub use, a situation thought to lead to particularly high levels of infectious aerosols containing organisms found in the water.[6] Whether this pulmonary response to MAC represents true infection or "classic" hypersensitivity pneumonitis is controversial, as some patients improve only after antimycobacterial drug therapy, while other patients improve after corticosteroids or the removal of the exposure. The classification of this pulmonary response to MAC is further complicated by the increasing observations of histopathologic and bronchoscopic differences between "classic" hypersensitivity pneumonitis and MAC-associated hypersensitivity pneumonitis.[6,9,14]

We present the first reported case of MAC hypersensitivity pneumonitis associated with exposure to MAC in the routine use of household water. Water samples from the patient's shower and bathtub faucets and his sputum all yielded the same strain of MAC, as defined by pulsed-field gel electrophoresis. A review of the details of this case and other cases of MAC- and NTM-associated hypersensitivity pneumonitis highlights the clinical differences between "classic" hypersensitivity pneumonitis and MAC hypersensitivity pneumonitis, which we propose may have implications for classification and treatment. Further, our report stresses the clinical importance of alternative sources of aerosolized MAC that can lead to MAC hypersensitivity pneumonitis.

Case Report

A 50-year-old man had progressive exertional dyspnea and chest tightness for 3 months. He noted two or three previous discrete self-limited episodes characterized by malaise, subjective fever, and myalgias, each lasting 24 to 72 h, the first of which predated his pulmonary symptoms by 1 week. His medical history was noncontributory, he took no medications, never smoked, used no illicit substances, and had no significant risk factors for HIV infection. He drank one to two glasses of wine on weekends. He had always been extremely active and regularly completed a demanding mountain bicycling course until pulmonary limitations developed. He had worked as a dentist for 23 years, performing routine examinations and procedures and producing oral appliances with dimethacrylates, but there was no history of exposure to beryllium. He wore a paper ear-loop mask when fabricating appliances and working with patients. His symptoms were not clearly associated with work, and there was no change on the weekends. He lived with his wife in the same house for 16 years. The structure was 65 years old, and had been renovated before they moved in. Minor remodeling, involving removal of some drywall and subsequent plastering, was performed around the time his symptoms began. He intermittently used his outdoor hot tub, which filtered through an adjacent swimming pool, but not for several weeks before symptom onset. He kept no pets.

Physical examination was unremarkable. The chest examina-

*From the Department of Medicine (Respirology) [Dr. Marras], University of Toronto, Toronto, Canada; Department of Microbiology (Dr. Wallace), University of Texas Health Center at Tyler; Department of Medicine (Pulmonary and Critical Care) [Drs. Koth, Daley, and Stulbarg], University of California, San Francisco; and Department of Internal Medicine (Preventive and Occupational Medicine) [Dr. Cowl], Mayo Clinic, Rochester, MN.

†Deceased.

This work was performed in the Department of Medicine (Pulmonary and Critical Care), at the University of California, San Francisco.

Dr. Marras was supported by a postdoctoral fellowship from the Canadian Institutes of Health Research and Canadian Thoracic Society.

Manuscript received March 8, 2004; revision accepted August 10, 2004.

Correspondence to: Theodore K. Marras, MD, Division of Respirology, Toronto Western Hospital, Edith Cavell 4–022, 399 Bathurst St, Toronto, ON, Canada M5T 2S8; e-mail: ted.marras@utoronto.ca