Communications to the Editor

Absence of Lymphocytic Alveolitis in Patients with Multiple Sclerosis

To the Editor:

The differentiation between multiple sclerosis (MS) and human T-cell lymphotropic virus type I (HTLV-I)-associated paraplegia (HAP) is difficult. Clinical guidelines for separating the two entities have been suggested.1 Because lymphocytic alveolitis is a frequent finding in HAP patients, an increased number of lymphocytes in bronchoalveolar washings may be suggestive of HAP rather than MS.1,2 The aim of this study was to determine whether MS patients have lymphocytic alveolitis.

We studied six patients (five white, one black; three male, three female). The black patient was a 36-year-old woman from French Guiana. None of the patients had abused drugs. None had a previous history of blood transfusion. All male patients were heterosexual. All patients fulfilled the diagnostic criteria for definite MS.1 No patient presented with paraplegia. In five cases, MS was in remission when bronchoalveolar lavage was performed. One month before study, the black female patient had had left arm and leg weakness, which improved with corticosteroid therapy. All patients were seronegative for human immunodeficiency virus and HTLV-I in enzyme-linked immunosorbent and Western blot assays. Informed consent was obtained from all patients.

Bronchoalveolar lavage was performed as previously reported.2 Patients 1, 2, and 4 were smokers. No patient had pulmonary signs or symptoms, and all patients had normal chest roentgenograms. The characteristics of cells recovered by bronchoalveolar lavage are summarized in Table 1. Absolute numbers and proportions of cell yields by bronchoalveolar lavage were normal.

We have shown an absence of lymphocytic alveolitis in all MS patients tested. These findings contrast sharply with the high frequency of lymphocytic alveolitis observed in HAP patients.1,2 We agree with the hypothesis of Poser et al3 that MS patients have no lymphocytic alveolitis. In endemic areas for HTLV-I infection, determination of the presence of lymphocytic alveolitis may differentiate MS patients from patients with HAP.

Louis-Jean Couderc, M.D.,
Gérard Said, M.D.,
Jean-Luc Truelle, M.D.,
Dominique Israel-Biet, M.D., and
Bernard Epardeau, M.D.,
Paris, France

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Mycoplasma pneumoniae in the Immunocompromised Host

To the Editor:

In the article by Perez and Leigh,1 which appeared in the September 1991 issue of Chest, the authors propose a greater risk for Mycoplasma pneumoniae infection in respiratory tract infections of immunocompromised hosts than is generally recognized. As an extension of their proposal, it is reasonable to project that the common community-acquired etiologic agents of respiratory disease in immunocompetent patients will also be those that are common for immunocompromised patients in the community. The major challenge in this regard for diagnostic laboratories will be to confirm the presence of such common agents before more detailed, invasive, and/or costly investigations are required to subsequently diagnose the opportunistic infections. I report the cases of two immunocompromised patients whose diagnoses of M pneumoniae infection were established by the use of a rapid 4-h serologic assay.2

Case 1: A 13-year-old girl, who was actively receiving chemotherapy for acute myelocytic leukemia, was admitted to the hospital...
with a seven-day history of rhinorrhea and cough. On admission, the patient was febrile, productive of sputum, and tachypneic. A chest x-ray film revealed lower lobe pneumonia. The white blood cell count was 1.9 x 10^9/L, which included 10 percent blast forms. A rapid immunoglobulin M (IgM) immunoblotting assay was positive. The patient responded to oral erythromycin. The complement fixation (CF) titer on the single serum specimen was subsequently determined to be 1/128.

Case 2: An 11-year-old girl, who had completed chemotherapy for acute lymphocytic leukemia six months previously, was admitted to the hospital with fever and a cough of three weeks' duration. The respiratory disease did not respond to either amoxicillin or cefaclor. A chest x-ray film revealed left perilobar infiltrates. Peripherally white blood cell count was 8.0 x 10^9/L, and there was no evidence of the leukemia, although further investigations uncovered a relapse of central nervous system involvement. The rapid IgM test was positive, and the CF titer was subsequently found to be 1/256.

These two case studies illustrate that it is possible to establish a rapid serologic diagnosis of M pneumoniae infection in some immunocompromised patients. This approach has the potential to establish a "same-day" diagnosis, in contrast to the delayed results from culture and conventional CF serologic study. The results of any serologic assay should be considered, of course, in the context of the degree of immunosuppression and the transfusion history.

Nevio Cimolai, M.D.,
Division of Medical Microbiology,
University of British Columbia,
Vancouver, Canada

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Nonsurgical Management of Bleeding Secondary to Tube Thoracostomy

A Case Report

To the Editor:

Bleeding secondary to tube thoracostomy frequently originates from an injured intercostal vessel.1 A case of significant bleeding (>1.4 L over 2 h) after tube thoracostomy was recently encountered in a 51-year-old woman with an exudative right pleural effusion, overwhelming sepsis, disseminated intravascular coagulopathy, and multisystem organ failure. The tube was steriley replaced with a 30-mL balloon-tipped 24F Foley catheter connected to 20 cm H2O suction drainage and placed on 1 lb of tension. The wound site was tightly packed. Hemorrhage was immediately tamponaded, and subsequently the catheter was removed without recurrent bleeding.

Hemorrhage occurred although standard techniques for tube thoracostomy were employed, including (1) acquisition of a preprocedure chest radiograph; (2) high lateral insertion to prevent subdiaphragmatic placement; (3) dissection over the cephalad surface of the lower rib of the interspace to avoid injury to the intercostal vessels; (4) "blunt technique" insertion, rather than use of a trocar chest tube, which can more readily injure chest wall and visceral structures; and (5) digital palpation of the pleural cavity to ensure nonadherence of the lung locally.

In conclusion, bleeding secondary to tube thoracostomy that is suspected to originate from the chest wall (ie, no air leak present)