Hepatitis A Vaccine and Travel Departure

Sir—In his excellent state-of-the-art clinical article on protection of travelers, Wolfe [1] states that hepatitis A vaccine may not provide protective immunity until 4 weeks after immunization.

Hepatitis A is the most common vaccine-preventable illness among travelers [2]. The prescribing information for Havrix (Hepatitis A Vaccine, Inactivated; SmithKline Beecham Pharmaceuticals, Philadelphia) states that primary immunization should be completed at least 2 weeks before anticipated exposure to hepatitis A virus, although Wolfe’s recommendations are concordant with those of the Advisory Committee on Immunization Practices [3].

Most vaccinees respond with detectable antibodies to hepatitis A virus by 15 days after vaccination [4]. Given the incubation period of hepatitis A virus (average period, 28 days) [3], it may be reasonable to give the first dose of vaccine at any time >2 weeks before departure.

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References

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Reply

Sir—Sensitive immunoassays indicate that most persons have detectable antibody to hepatitis A virus 2 weeks after hepatitis A vaccination. However, studies have shown that not all vaccinees have detectable neutralizing antibody at this time: 54%–62% developed neutralizing antibodies 2 weeks after vaccination (nearly all persons have neutralizing antibodies 1 month after vaccination) [1]. Since early after vaccination the presence of neutralizing antibodies is probably a better indicator of protection than are antibodies to hepatitis A virus, the safest approach is to administer hepatitis A vaccine 4 weeks before travel to an area where hepatitis A is endemic. If this cannot be done, we believe that as a conservative measure to better ensure protection during this 4-week period of potential vulnerability, immunoglobulin (if available) should also be administered at the time of hepatitis A vaccination.

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Reference

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Pneumocystis carinii Pneumonia in Patients with Breast Cancer: Are There Contributing Local Factors?

Sir—Kulke and Vance [1] recently described two patients with metastatic breast cancer who developed Pneumocystis carinii pneumonia (PCP) after receiving therapy with high doses of cyclophosphamide with peripheral blood stem cell support. Both patients had severe CD4 T cell lymphocytopenia. Walzer [2], in an editorial response, suggested that CD4 cell counts might serve as a crude measure of the extent of immunosuppression in this setting. We describe a patient with breast cancer who developed PCP after a single course of low-dose, nonmyeloablative chemotherapy; she did not have severe CD4 cell depletion.

A 61-year-old woman was hospitalized for evaluation of dyspnea, cough, and fever 1 month after she received a first course of chemotherapy (cyclophosphamide [400 mg/ (m2 · d)] and pirarubicin [20 mg/(m2 · d)] for 3 days) for local relapse of breast carcinoma. She had undergone lumpectomy and local radiotherapy (45 Gy) for invasive node-negative adenocarcinoma of the breast 5 years before the current admission. Administration of chemotherapy resulted in grade 4 neutropenia (World Health Organization classification) with spontaneous recovery 5 days later. She had not received any other immunodepressive medications, including corticosteroids.

On admission to the hospital, her temperature was 38°C, and her respiration rate was 26; there was no hemodynamic disturbance. A chest radiograph revealed bilateral diffuse infiltrates. The P02 was 78 mm Hg while the patient was breathing room air, the PC02 was 38 mm Hg, and the pH was 7.43. Examination of bronchoalveolar lavage (BAL) fluid obtained by fiberoptic bronchoscopy revealed