**Pneumocystis carinii**. A serological test for HIV was negative. The WBC count was 6,750/μL, and the CD4 cell count was 340/μL, with a CD4/CD8 cell ratio of 1.4. Therapy with trimethoprim-sulfamethoxazole (TMP-SMZ) (100 mg/[kg·d]) was initiated, and she deferred over the course of 72 hours. After she had received this regimen for 21 days, a continuous prophylactic regimen for PCP (TMP-SMZ, one double strength tablet/d) was prescribed, and a second cycle of chemotherapy was administered as previously.

Twenty days later, the patient’s cough and shortness of breath relapsed, without fever. Examination of specimens, including BAL fluid obtained by fiberoptic bronchoscopy, did not reveal *P. carinii*, but histological studies of a transbronchial biopsy specimen revealed lymphangitic carcinomatosis of the lung. She subsequently received eight cycles of high-dose chemotherapy, which included cyclophosphamide, doxorubicin, 5-fluorouracil, and vindesine, and her respiratory symptoms resolved. After six cycles, the results of several studies (including fiberoptic bronchoscopy with transbronchial biopsies) were completely negative, and the patient was considered to be in complete remission. No relapse of PCP has been observed while PCP prophylaxis was continued (as of a 16-month follow-up examination).

Our HIV-negative patient, who had not had previous exposure to corticosteroids, developed documented PCP after a single cycle of low-dose cytotoxic chemotherapy for local relapse of breast cancer. In their review of PCP in patients with breast cancer (24 cases), Kulke and Vance [1] stressed that most of the patients had received prolonged corticosteroid therapy. The two patients they described had not received steroids, but these patients were receiving high-dose chemotherapy and had severe CD4 T cell lymphocytopenia at the time PCP was diagnosed.

According to Walzer [2], it is not the nature of the underlying disorder that predisposes to the development of PCP in patients with cancer, but rather the type and the intensity of the cytotoxic or immunosuppressive therapy used to treat the cancer. However, other potential contributing factors deserve to be studied. Sepkowitz et al. [3] suggested that patients who had received previous radiation therapy to the thorax might be at higher risk of developing PCP. Among the 24 cases of PCP in patients with breast cancer reviewed by Kulke and Vance [1], the only patient who clearly did not receive steroid therapy before infection developed had concurrent lymphangitic carcinomatosis. In the case reported herein, neither the single cycle of low-dose chemotherapy nor the CD4 cell count would be sufficient to explain the development of PCP. We suggest that local factors such as previous irradiation or the presence of lymphangitic carcinomatosis could be additional risk factors for PCP in patients with breast cancer.

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In summary, studies have demonstrated that *P. carinii* may not have been sufficiently considered for patients whose common feature is immunosuppression due to a variety of reasons, including therapy for malignancy and organ transplantation. Although the prevalence of PCP among HIV-1–positive individuals has received more attention, it is clear that heightened awareness of the possibility of PCP in any significantly immunosuppressed patient would be prudent. Without question, further research will be essential to provide more in-depth fundamental information about the nature of this intriguing opportunist.

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**Correcting the Historical Record: Diagnosis and Management of Group A Streptococcal Pharyngitis**

**Stir**—In their guidelines for diagnosis and management of group A streptococcal infections [1], Bisno et al. err in stating that the only antibiotic therapy actually examined in controlled studies and shown to prevent rheumatic fever is penicillin. Chlortetracycline [2] and oxytetracycline [3] were evaluated in clinical trials of sufficient size to determine their effectiveness in preventing rheumatic fever. A significant reduction in the number of cases of rheumatic fever was demonstrated with chlortetracycline treatment, while the reduction with oxytetracycline treatment was borderline ($P = .07$).

Perhaps the authors meant to say that of currently recommended antibiotics, penicillin is the only one that has been evaluated in clinical trials. The criterion for choosing other antibiotics is a high rate of eradication of the infecting organism. My colleagues and I observed in our paper on chlortetracycline that eradication of the infecting organism is necessary to prevent subsequent episodes of rheumatic fever.

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**Reply**

**Sir**—Houser’s critique is well taken. We erred in not mentioning the tetracycline data [1]. As he correctly surmised, however, our guideline was directed toward currently recommended therapy for acute streptococcal pharyngitis. As Houser et al. pointed out in their 1953 report [2], penicillin was more effective than aureomycin in eradicating streptococci, decreasing antistreptolysin formation, and preventing rheumatic fever. Moreover, ~10% of group A streptococci in the United States are currently resistant to tetracycline [3], while these organisms remain uniformly susceptible to penicillin. Then, as now, penicillin remains the therapy of choice for streptococcal pharyngitis.

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