A 39-year-old man with AIDS suffered from fever for 1 week. Chest posteroanterior X-ray showed a ground glass appearance in the bilateral hilar areas. He died of respiratory failure. An autopsy proved a diagnosis of *Pneumocystis carinii* pneumonia characterized by foamy exudates in the alveolar spaces accompanied by lymphoplasma cell infiltration in the interstitial areas (Fig. 1A) and a cluster of pneumocystic cysts demonstrated with Grocott’s methenamine–silver stain (Fig. 1B). *P. carinii* causes progressive, often fatal, pneumonias in persons with severely impaired cell-mediated immunity and is the most common serious opportunistic pathogen in persons with AIDS, such as our patient. *P. carinii* reproduces in intimate association with alveolar type 1 lining cells and active disease is confined to the lungs. Infection begins with attachment of the *Pneumocystis* trophozoite to the alveolar lining cell. The trophozoite feeds on the host cell, enlarges and transforms into cyst form, which contains daughter organisms. The cysts then rupture to release new trophozoites, which, in turn, attach to additional alveolar lining cells. If the process is not checked by the host immune system or antibiotic therapy, the infected alveoli eventually fill with organisms and exudates.

Fig. 1 — (A) Histopathology shows foamy pinkish exudates in the alveolar spaces with lymphoplasma cell infiltration in the interstitial areas (hematoxylin & eosin, 200×). (B) Grocott’s methenamine–silver stain shows a cluster of *Pneumocystis carinii* cysts.
proteinaceous fluid. The progressive filling of the alveoli prevents adequate gas exchange and the patient slowly suffocates. The clinical presentation of pneumocystic pneumonia is variable. At one extreme, symptoms are minimal, whereas at the other, there is rapidly progressive respiratory failure. The disease is treated with trimethoprim-sulfamethoxazole.

References