in. While receiving appropriate antibiotic therapy, two patients had fatal outcomes (due to concomitant opportunistic infection) and four patients recovered. As Cunha correctly suggested, prior antibiotic therapy probably selected out *P. aeruginosa* and *P. aeruginosa* colonized the patients' lower respiratory tracts. In our study, hospital-acquired pneumonia was defined as bacterial pneumonia occurring ≥ 2 days after hospitalization without signs of pneumonia at the time of admission [3]. That was not the case for the six patients with pneumonia due to *P. aeruginosa*. However, we agree with Cunha that the definition of hospital-acquired pneumonia may not be appropriate for highly immunocompromised HIV-infected patients with a history of frequent hospitalization.

Our study was undertaken before the start of new multidrug therapy containing protease inhibitors, in 1994. We focused on 33 cases of pyogenic bacterial pneumonia in hospitalized HIV-infected patients, with a resulting incidence of 12.5 cases per 100 inpatients (95% CI, 8.8–17.2). We now report the results of a study we conducted in 1998. The number of HIV-infected persons followed in the Department of Infectious Diseases at the Pitié-Salpêtrière Hospital (Paris) was 2,224 in January 1998. Again, as in 1994, we prospectively included all cases of pyogenic bacterial pneumonia in 293 consecutive HIV-infected inpatients over the same 6-month study period. The case definition of bacterial pneumonia and the standardized questionnaire were the same as in the first study. From February through July 1998, 22 inpatients with 27 episodes of bacterial pneumonia were included (e.g., 7.5 cases per 100 inpatients [95% CI, 4.7–11.1]). Three patients had two consecutive episodes and two patients had three consecutive episodes during the study period. Thirteen patients (59.1%) had a previous AIDS clinical status, with a mean duration of AIDS of 3.5 years (SD ± 2.3 years). Three patients (13.6%) were considered HIV-infected at the time of their bacterial pneumonia. The median CD4+ cell count was 196/µL (interquartile, 27–405/µL); CD4+ lymphocyte count was <50/µL, 50–200/µL, >200/µL in 8, 3, and 11 episodes, respectively. The median viral load was 3.95 log_{10} copies/mL (interquartile: 3.35–5.19 log_{10} copies/mL). Eighteen patients (82.0%) received antiretroviral therapy for a median duration of 65 months (interquartile, 3.6–119 mo) including 14 patients (64%) who received multidrug therapy containing a protease inhibitor for a median duration of 19 months (interquartile, 11.4–21 mo).

The two major risk factors for bacterial pneumonia in HIV-infected inpatients identified in our previous study were still evident. Thirteen patients (48.1%) had had sinusitis within 1 month before admission and twelve (44.4%) had had bacterial infection of the lower respiratory tract during the 6 months before admission. None of the patients were vaccinated against *Streptococcus pneumoniae* or *Haemophilus influenzae*. Microbiological etiologies were obtained in 12 (44.4%) of the 27 episodes of bacterial pneumonia. Cultures of PSB specimens (10 episodes) and blood (4) yielded positive results: *S. pneumoniae* (7), *H. influenzae* (1), *P. aeruginosa* (1), *Escherichia coli* (2), *Moraxella catarrhalis* (1), and *Bordetella bronchiseptica* (1). In one PSB specimen, two bacterial species were identified by culture. Six episodes were due to *S. pneumoniae* with decreased susceptibility to penicillin, including five low-level penicillin-resistant strains and one high-level penicillin-resistant strain (MIC, 3 mg/L).

In conclusion, new multidrug therapy containing protease inhibitors decreases the incidence of severe bacterial pneumonia in HIV-infected patients. Cases of *P. aeruginosa* pneumonia have probably decreased because of the improvement of system restoration induced by new antiretroviral therapy. *S. pneumoniae* with decreased susceptibility to penicillin still remains a major pathogen for severe bacterial pneumonia in HIV-infected patients.

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References

Varicella-Zoster Virus Infection Associated with Acute Liver Failure

SIR—Dits and colleagues recently described a renal transplant recipient with varicella-zoster virus infection and hepatic failure [1]. I would like to present a similar case that occurred in a previously healthy man.

A 20-year-old man was admitted to the hospital with a 2-day history of progressively worsening back and abdominal pain accompanied by a rash. He was a known asthmatic and used theophylline and bronchodilator inhalants as needed. He had not recently taken steroids. He consumed alcohol only socially, and he was a nonsmoker.

Physical examination revealed an anxious man with a temperature of 100°F, thrashing about on the bed and crying with pain. The abdomen was soft, with audible bowel sounds and no organomegaly. There was no rebound tenderness, but the lower abdomen was mildly tender to palpation. The patient noted mild abdominal pain accompanied by a rash. He was a known asthmatic and used theophylline and bronchodilator inhalants as needed. He had not recently taken steroids. He consumed alcohol only socially, and he was a nonsmoker.

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of the liver. Despite initiation of therapy with intravenous acyclovir, the patient’s condition deteriorated; he developed respiratory insufficiency requiring intubation, acute renal failure with massive metabolic acidosis, disseminated intravascular coagulation, and fatal gastrointestinal hemorrhage. Permission for an autopsy was refused.

Although no viral cultures were obtained, given the appearance of the rash and the pattern of spread, there seemed little doubt that this was acute varicella-zoster virus infection. The degree of abdominal and back pain was somewhat unusual, but has been described in the literature. Rowland et al. [2] reported their experience with varicella-zoster virus complicating acute lymphoblastic leukemia at the Children’s Hospital of Pittsburgh [2]. In 13 (30%) of their 42 cases, they noted extracutaneous involvement; 5 of these children died. Of note, severe abdominal and/or back pain was a prominent feature in 10 of the 13 patients who had visceral involvement and in 4 of the 5 who died.

In summary, a previously healthy man developed varicella zoster virus infection with hepatic dysfunction, progressing to multi-organ system involvement and death. Abdominal or back pain is probably an underappreciated feature of varicella-zoster virus infection and may portend a poor prognosis.

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