Gram staining of the fluid did not demonstrate any organisms. Culture of the fluid yielded *Salmonella typhimurium*. He was treated with intravenous fluids, thiamine, diuretics, and amoxicillin/clavulanate but died on day 11.

We reviewed the medical literature (irrespective of language) from 1983 to 1997 with use of MEDLINE for cases of PBP and extended the search to other referenced articles. Eligible cases had both culture of peritoneal fluid that yielded nonenteric salmonella and a clinical diagnosis of PBP with no evidence for a perforated viscus.

We found 17 cases of PBP due to nonenteric salmonellosae in addition to our two cases [3–10] (table 1). Most patients had preexisting conditions, often associated with ascites, and presented with nonspecific symptoms. On the other hand, three of the patients had no known antecedent health problems: patient 1 in this report and the patients described by Laurens et al. [7] and Oliver et al. [10]. All three of these patients presented with acute abdominal emergencies, had preoperative diagnoses of acute appendicitis, and had a correct diagnosis of PBP only when pus was noted, and a perforated viscus was excluded, during laparotomy.

The six deaths occurred in patients with disseminated gastric carcinoma, congestive heart failure with alcoholism, chronic active hepatitis with non-insulin-dependent diabetes mellitus, systemic lupus erythematosus, and alcoholic cirrhosis (two cases). The age range for these patients was 27–71 years (mean, 52 years; median, 57 years). Two of them were infected with *Salmonella arizonae*. There were no reported systematic differences in the management of fatal cases. Some of the patients who died did not undergo early diagnostic paracentesis despite presenting with ascites, abdominal pain, or constitutional symptoms. None of the cases in this review occurred as part of recognized outbreaks of salmonella infection.

Both presentation and outcome of the three cases of salmonella PBP in previously healthy people were different. These patients presented with symptoms indicative of an acute inflammatory process in the abdomen. They underwent early surgery, and their diagnoses were rapid. All three patients survived.

**Invasive Fungal Sinusitis Due to *Scedosporium apiospermum* in a Patient with AIDS**

Invasive disease caused by filamentous fungi is an uncommon but increasingly reported complication of AIDS. To our knowledge, we describe the first case of invasive disease due to *Scedosporium apiospermum* in a patient with AIDS.

A 21-year-old male with hemophilia and AIDS was hospitalized because of left mastoid pain and swelling. His previous opportunistic infections included recurrent bacterial pneumonia, chronic sinusitis, and pulmonary aspergillosis, the latter for which the patient was receiving the 18th week of treatment with amphotericin B lipid complex (5 mg/[kg patient] day). His antiretroviral regimen included lamivudine, stavudine, and indinavir. The CD4 cell count 1 month before was 60/mm³, and the HIV type 1 RNA level was 1.5 × 10⁵ copies/mL. Physical examination revealed swelling and erythema of the left mastoid process.

Acute mastoiditis was diagnosed (figure 1), and therapy with imipenem and tobramycin was started. Transmastoid antec- tomy was performed, and cultures of the purulent material from the mastoid antrum yielded *S. apiospermum*. Histopathologic examination of antral tissue with Grocott-Gomori methenamine—silver nitrate staining revealed septated fungal hyphae. Itraconazole treatment was discontinued, and therapy with oral ketoconazole (400 mg b.i.d.) was empirically instituted. Susceptibility testing for the fungal isolate revealed the following MICs: amphotericin B, 6.0 μg/mL; fluconazole, 25 μg/mL; itraconazole, >12.5 μg/mL; ketoconazole, 6.25 μg/mL; and miconazole, <0.39 μg/mL. Mastoiditis worsened despite redebridement and replacement of ketoconazole with intravenous miconazole (600–1,000 mg i.d.). A fungal synergy study verified that the isolate remained susceptible to miconazole in the presence of amphotericin B; 1 week later, the patient died of worsening pulmonary disease.

Fungal sinusitis is a rare complication of HIV infection and is associated with late-stage AIDS and low CD4 cell counts (<150/mm³) [1]. A MEDLINE search of the literature identified a total of 24 cases of fungal sinusitis in patients with AIDS, 19 of which were due to *Aspergillus* species [2, 3]. The remaining five cases of fungal sinusitis were caused by *Pseudallescheria boydii,*
Primary Meningococcal Pericarditis

Primary meningococcal pericarditis is purulent pericarditis without clinical evidence of meningococcemia, meningitis, or other foci of meningococcal infection. It is extremely rare, elusive at initial presentation, and often associated with rapid progression of complications [1]. We report a case of primary meningococcal pericarditis.

An 18-year-old previously healthy male developed an abrupt onset of severe pleuritic left sided chest pain without other associated symptoms. At the time of physical examination, his vital signs were as follows: blood pressure, 140/110 mm Hg; pulse rate, 98; respiratory rate, 26; oral temperature, 37.3°C; and O2 saturation while the patient was breathing room air, 98%. The patient was muscular, sitting upright in obvious discomfort, and taking shallow breaths. The neck was supple without distention of the jugular veins. Heart sounds were normal, lungs were clear, and there was no chest wall tenderness. The skin was slightly diaphoretic without a rash.

Results of electrocardiography and laboratory studies were all

Treatment of invasive S. apiospermum infection requires prompt surgical excision along with antifungal therapy. This organism, however, is rarely susceptible to amphotericin B. Susceptibility to the imidazoles has been reported, and these agents are considered the treatment of choice [4, 6]. It is unclear if P. boydii susceptibilities can be generalized to S. apiospermum, as the sexual state may have different patterns of resistance to amphotericin B [7]. It is also unknown if prolonged use of amphotericin B has a selection effect on such organisms. Unfortunately, susceptibility testing for filamentous fungi is not standardized to date, and results of such testing correlate poorly with individual responses to therapy. In the future, an optimal testing method for molds and interpretive guidelines will need to be established [8].

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References