an exudate. Third, culture of the pleural fluid yielded *E. coli*, which is also a common infectious agent in spontaneous bacterial peritonitis [4].

Although 10% of cirrhotic patients with ascites have an associated pleural effusion [2], SBE is rare; it has been reported previously in only 21 patients [5], most of whom presented concomitantly with spontaneous bacterial peritonitis. SBE occurred in association with sterile ascitic fluid in only three cases [5].

The pathogenesis of SBE is unclear. Infected ascitic fluid may penetrate into the pleural cavity through the diaphragm, which would explain the simultaneous infection of both ascitic fluid and pleural fluid. In our case it appears that bacteremia, which commonly occurs in portal hypertension, caused seeding of the *E. coli* in the pleural fluid. However, it is unclear why bacterial seeding occurs in the pleural fluid but not in the ascitic fluid.

Since spontaneous bacterial peritonitis can cause clinical deterioration in cirrhotic patients who do not have signs of peritonitis, patients in whom clinical deterioration of unknown cause occurs should be evaluated (by pleurocentesis) for the presence of SBE even when examination of ascitic fluid does not reveal infection.

Prolonged Fecal Shedding of *Escherichia coli* O157:H7 During an Outbreak at a Day Care Center

It has been reported that *Escherichia coli* O157:H7 is cleared from the gastrointestinal tract rapidly after the onset of hemorrhagic colitis [1]; recovery of the organism in stool is unlikely >1 week after the onset of symptoms [2, 3]. However, the duration of excretion of *E. coli* O157:H7 has been found to vary inversely with age [4], Pai et al. [5] found that 53% of children <5 years of age had stool cultures positive for *E. coli* O157:H7 3 weeks after the onset of symptoms. In a study of children <5 years of age in Minnesota child care centers, the median duration of shedding of *E. coli* O157:H7 in a cohort of culture-positive children was estimated to be 17 days, with a range of 2–62 days; 38% of these children continued to shed the organisms for ≥3 weeks [6]. The 16th edition of the *Control of Communicable Diseases Manual* reported a duration of excretion of up to 1 week in adults and up to 3 weeks in one-third of children [7].

An outbreak of hemorrhagic colitis due to *E. coli* O157:H7 in a Colorado child care center in June 1995 provided the opportunity to study the duration of shedding of the organism. A case was defined as any child within the child care center who had a stool culture positive for *E. coli* O157:H7 or who had diarrhea for ≥2 days. Of the 141 children in the center, 24 met the case definition.

As this case demonstrates, the finding of sterile ascitic fluid does not exclude the presence of SBE in cirrhotic patients.

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References

Concurrent Mucosal Leishmaniasis and Pulmonary Tuberculosis

We observed a case in which clinical resolution of both mucocutaneous leishmaniasis and pulmonary tuberculosis occurred following treatment with only antituberculosis medications. This case raises three issues: the lack of guidelines for simultaneous treatment of these two infections, the potential efficacy of antituberculosis medications in the treatment of leishmaniasis, and the possible interaction of these two diseases.

A 50-year-old male agricultural worker from the department of Valle, Colombia, presented with mucocutaneous leishmaniasis of 7 months’ duration. We observed a 3 × 2-cm ulcer on his filtrum that extended to his lower nares; there was complete destruction of his septum and middle and inferior turbinates and a granulomatous infiltrate of the soft palate with destruction of the uvula, pillars, and tonsils. The pharynx was edematous; the larynx was examined, but the patient’s voice was hoarse. A hyperpigmented scar on the right ear lobe, typical of prior cutaneous leishmaniasis, had healed 25 years earlier. The patient also reported a family history of tuberculosis; a 1-year history of weight loss, anorexia, fatigue, and nocturnal fevers; and a productive cough with sputum that had been streaked with blood for 1 month. Findings of the physical examination were otherwise normal.

Cultures of two mucosal biopsy specimens yielded Leishmania species. Leishmania viannia braziliensis or Leishmania viannia panamensis was the most likely pathogen based on the patient’s place of residence. The leishmanin skin test reaction was 9 × 6 mm, and a PPD test with 5 TU produced an induration of 29 mm. Sputum smears were positive for acid-fast bacilli, and cultures of sputum yielded Mycobacterium tuberculosis. A chest roentgenogram showed reticular infiltrates in the upper-right and lower-left lobes. A Venereal Disease Research Laboratory test was negative, as were serologies for HIV and human T-cell lymphotropic virus type I.

The patient received supervised outpatient antituberculosis therapy; im streptomycin (0.75 g/d), oral rifampin (600 mg/d), isoniazid (300 mg/d), and pyrazinamide (1,500 mg/d) were administered for 8 weeks, followed by oral rifampin (600 mg) and isoniazid (800 mg) twice weekly for 7 months. His sputum smear converted to negative after 2 months and continued to be negative. He failed to return for his deferred antimonial therapy. Three months after completing his antituberculosis medications, he had gained 4 kg, the cutaneous lesion of the filtrum was scarred, and fibrosis of the mucosal lesions was observed. Cultures of biopsy specimens from the right vestibulum were negative for *Leishmania* species.

We deferred antimonial treatment because the manufacturer’s product information in 1991 stated that meglumine antimoniate (Glucantime, Specia, Paris) was contraindicated in patients with tuberculosis and because findings in previously reported cases had raised similar concerns. Two patients with both tuberculosis and visceral leishmaniasis died, and their deaths were apparently accelerated by the initiation of antimonials [1]. A 60-year-old male with pleural tuberculosis and mucosal leishmaniasis died of acute renal failure shortly after initiation of therapy with sodium stibogluconate (Pentostam; Wellcome Foundation, London, UK) [2].

The sequence of events, the fact that pleural tuberculosis is rarely fatal, and the fact that antimonials are associated with renal toxicity support antimonials as the most plausible—but not certain—cause of this patient’s death.

In contrast, eight patients with both kala-azar and pulmonary tuberculosis were treated concurrently with antimonials and antituberculosis drugs, and the conditions of these patients improved [3]. In a trial of antimonial treatment for visceral leishmaniasis, children with concomitant tuberculosis were less responsive to antimonials than were children without tuberculosis [4]. This latter observation indicates that antimonials may be less effective when administered to patients with concomitant tuberculosis but would not preclude their use in patients with active tuberculosis. Because tuberculosis is highly prevalent in areas that are endemic for leishmaniasis, their simultaneous occurrence is likely. Clinicians should therefore use antimonials