an exudate. Third, culture of the pleural fluid yielded \textit{E. coli}, which is also a common infectious agent in spontaneous bacterial peritonitis \cite{4}.

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

\textbf{Prolonged Fecal Shedding of \textit{Escherichia coli} O157:H7 During an Outbreak at a Day Care Center}

It has been reported that \textit{Escherichia coli} O157:H7 is cleared from the gastrointestinal tract rapidly after the onset of hemorrhagic colitis \cite{1}; recovery of the organism in stool is unlikely \(>1\) week after the onset of symptoms \cite{2, 3}. However, the duration of excretion of \textit{E. coli} O157:H7 has been found to vary inversely with age \cite{4}. Pai et al. \cite{5} found that 53\% of children \(<5\) years of age had stool cultures positive for \textit{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 \textit{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 \cite{6}. The 16th edition of the \textit{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 \cite{7}.

An outbreak of hemorrhagic colitis due to \textit{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 \textit{E. coli} O157:H7 or who had diarrhea for \(>2\) days. Of the 141 children in the center, 24 met the case definition.

Twelve of the 24 cases had stool cultures positive for \textit{E. coli} O157:H7. The average time between the onset of symptoms and the first positive stool culture was 10.5 days for the 12 culture-positive children, whereas the average time between the onset of symptoms and the first negative stool culture was 22.5 days for the 12 culture-negative children.

Children with stool cultures positive for \textit{E. coli} O157:H7 were included with other cases at the center or excluded until they had two consecutive stool cultures negative for \textit{E. coli} O157:H7. Thus, the duration of fecal shedding for \textit{E. coli} O157:H7 for symptomatic, culture-positive children could be estimated from the results of serial stool cultures. The duration of fecal shedding was defined as the interval from the onset of diarrhea to the first of two consecutive negative stool cultures. The median duration of shedding was 29 days, with a range of 11–57 days. The duration of shedding was \(>3\) weeks for 92\% of the children. Three culture-positive cases were treated with antibiotics and had a mean (± SD) duration of excretion of 35.7 days (± 12.4) versus 30.1 days (± 13.0) for the culture-positive cases not treated with antibiotics.

The present study may overestimate the duration of excretion, since children who shed \textit{E. coli} O157:H7 for shorter periods were more likely to be culture-negative when they were first tested. We accounted for this possibility by arbitrarily assuming a conservative shedding period of 7 days for the 12 culture-negative cases. The mean duration of shedding was then recalculated for all 24 cases. Even with this conservative estimate, the mean duration of shedding was 19.3 days.

In summary, the duration of excretion of \textit{E. coli} O157:H7 among children in this outbreak was longer than has been previously reported. The prolonged shedding period had a significant impact on day care staff and parents because of the inconvenience of cohorting and/or excluding children (i.e., keeping them at home or in separate rooms) until stool cultures demonstrated that fecal shedding was no longer present. The results of this study are
consistent with the observation that the sooner stool cultures are performed after the onset of symptoms, the greater the likelihood they will be positive for *E. coli* O157:H7 [2, 4].

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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 x 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 not 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 x 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 1.

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 (pentostam; Wellcome Foundation, London, UK) [2].

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