the 14 cases involving *E. coli*, results of evaluation for stx1, stx2, and eae genes were positive for 6 of 6, 3 of 5, and 2 of 3, respectively. Of the 6 cases of *E. coli* with known serotypes, 5 were non-O157. Thus, the phenotypic and genetic profiles of our strain (not serotypable; stx2 positive; stx1 and eae negative) seem to be atypical. The lack of diarrhea in the patient we described might be related to the absence of the eae gene in our strain, given that this gene encodes for intimin, an adhesive protein that facilitates colonization of intestinal epithelium and subsequent disease [4]. The transient increase of serum creatine levels following antibiotic therapy for UTI does not allow us to conclude with certainty that ofloxacin was responsible. However, this observation is in agreement with results of a recent in vitro study showing that quinolones may induce the stx2 gene [5]. The presence of the stx2 gene in our strain might also explain the severity of the HUS, given that the association of stx2 with severity of the disease has been previously reported [6].

The case described herein of a STEC UTI associated with HUS underscores the importance of direct detection of Shiga toxins in urinary *E. coli* strains and suggests that quinolones should perhaps be avoided as therapy for UTI in this context. Many authors attributed the adverse effect of antimicrobial agents in HUS with diarrhea to the release of Shiga toxins. Our observation seems to support this position.

**References**


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**Streptococcus intermedius: A Cause of Lobar Pneumonia with Meningitis and Brain Abscesses**

Members of the *Streptococcus milleri* group—*Streptococcus intermedius, Streptococcus anginosus, and Streptococcus constellatus*—are known for their propensity to cause suppurative infections [1, 2]. The lungs or the brain are commonly affected in these infections [3–5]. Infrathoracic infections most commonly present as empyemas [3, 4]. Although pneumonia is a well-known presentation of infections due to the *S. milleri* group, lobar pneumonia has not been described [3, 4]. Furthermore, even though brain abscesses caused by this group of organisms have been reported [5], the association of pneumonia, meningitis, and brain abscesses has not been recognized. We describe a patient with *S. intermedius* infection to illustrate that this association might be characteristic of infection with these organisms.

A 55-year-old man with alcoholic cirrhosis presented with a 10-day history of fever, cough, and hemoptysis. He then began to have headache followed by a rapid onset of lethargy and confusion. Physical examination disclosed fever (temperature, 38.5°C), stiff neck, and rales in the right upper-lung field. A chest radiogram demonstrated right upper-lobe consolidation. MRI of the brain disclosed bilateral ring-enhancing lesions consistent with multiple abscesses. CSF analysis revealed the following values: WBCs, 917/mL (67% polymorphonuclear neutrophils); glucose, 26 mg/dL; and protein, 238 mg/dL. Gram staining of CSF was negative.

Therapy with ceftriaxone and ampicillin was started; however, his condition rapidly deteriorated, and he required mechanical ventilation. Gram staining of sputum showed mixed bacteria. Video-assisted thorascopic lung biopsy was performed, but the patient had cardiac arrest and died. Sputum culture yielded normal flora, and blood and CSF cultures remained negative; however, examination of the lung biopsy specimen showed necrotizing pneumonia with gram-positive cocci in chains, and culture of the specimen yielded pure growth of *S. intermedius*. Autopsy demonstrated lobar pneumonia with a single lung abscess, meningitis, and multiple brain abscesses with numerous gram-positive cocci; brain culture was not performed.

This case illustrates that *S. intermedius* may cause lobar pneumonia. Previously reported thoracic infections with this organism, and other species that belong to the *S. milleri* group, include empyema, lung abscess, and bronchopneumonia [3, 4]. Lobar pneumonia has not been described. Whether lobar pneu-
monia is a rare presentation or a more common but unrecognized manifestation because of failure to recover the organism from sputum is uncertain. Furthermore, our patient was cirrhotic. It is possible that cirrhosis predisposed him to have more severe pneumonia. In comparison with infections caused by Streptococcus pneumoniae, those due to the S. milleri group affect hosts with underlying conditions and present with a more protracted course [4]. Extrathoracic manifestations of infections caused by the S. milleri group include dental abscess, endocarditis, and visceral abscesses [2, 3, 5, 6].

Our patient presented with a triad: pneumonia, meningitis, and brain abscesses. Although S. pneumoniae, Haemophilus influenzae, and Neisseria meningitidis are known to cause simultaneous lung and brain infections, CNS involvement is typically associated with meningitis and not with brain abscess. The characteristics of our patient suggest some unique features: subacute presentation, lack of bacteremia, and difficulty in recovering the organism from sputum and CSF. All these features are recognized characteristics of infection with the S. milleri group [4]. Therefore, we believe that S. intermedius infection should be considered in the differential diagnosis for patients with concomitant lung and brain infections, especially those with brain abscesses. Although S. pneumoniae more commonly causes associated lung and CNS infections, the subacute presentation, lack of bacteremia, and development of brain abscesses are perhaps more characteristic of infection with S. intermedius and possibly other species within the S. milleri group.

Central Nervous System Pneumocystosis in AIDS: Antemortem Diagnosis and Successful Treatment

CNS pneumocystosis is an extremely rare event in patients with AIDS. In a recent article published in Clinical Infectious Diseases, Bartlett and Hulette reported 1 case and reviewed 6 additional cases published in the English-language literature [1]. A striking feature is that all 7 cases were diagnosed after death, during necropsy. Recently we diagnosed CNS pneumocystosis in a patient with AIDS, which was treated successfully.

A 38-year-old homosexual man was admitted because of cephalalgia and fever, which had started 1 week previously. His medical history was unremarkable. At the physical examination the patient appeared to be severely ill. The axillary temperature was 37.8°C, the blood pressure was 122/78 mm Hg, and the heart rate was 78 beats per minute. He had some degree of confusion and mild neck rigidity.

Chest radiographic findings were normal. A cranial CT scan indicated mild cerebral atrophy. A lumbar puncture yielded clear CSF with a protein content of 2.4 mmol/L (glycemia, 6.1 mmol/L), and 42 leukocytes/mm³ (90% lymphocytes). Pathological examination revealed cryptococci and Pneumocystis carinii (figure 1), and culture of the CSF yielded Cryptococcus neoformans.

The titer of cryptococcal antigen in CSF was positive, at 1:2048. Serological tests for HIV (EIA and Western blotting) were positive. The CD4⁺ cell count was 40/mm³, and the HIV-1 viral load was 73,240 copies/mL. The patient was treated with iv amphotericin B at doses ≤0.1 mg/kg/d for 21 days and with cotrimoxazole (800/160 mg q.i.d.) administered intravenously for 15 days and then orally for 28 days, resulting in marked improvement. Later, he started secondary prophylaxis with oral fluconazole (400 mg/d) and cotrimoxazole (800/160 mg 3 times a week), plus antiretroviral therapy with lamivudine, stavudine, and indinavir, at current standard doses.

Six weeks later, biochemical parameters of the CSF were near normal, and no cryptococci or P. carinii were detectable pathologically. When the CSF was examined 6 months later, it was entirely normal. At the time of this writing (>1 year later), the patient was asymptomatic, and his condition was evolving well. His current viral load is <20 copies/mL, and the CD4⁺ cell count is 280/mm³. No relapses of CNS pneumocystosis have been detected.

References