triangle. The patient was apyrexial, findings on a chest examination were normal, and there was no hepatosplenomegaly or rash. A chest radiograph was obtained that showed widespread miliary shadowing. He was admitted to the hospital for further investigation.

Biopsy of the lymph node was performed; histopathologic examination showed caseating giant cell granulomas containing moderate numbers of acid-fast bacilli. Cultures of lymph-node biopsy material and early morning urine yielded Mycobacterium tuberculosis, which was fully susceptible to first-line antituberculous agents.

The patient started receiving therapy with rifampin, isoniazid, ethambutol, pyrazinamide, and pyridoxine. Within 4 days he developed a low-grade fever, which resolved over the subsequent 2 weeks. He was discharged to his home.

Results of outpatient laboratory studies revealed an increased blood CD4 T cell count, and the HIV viral load remained <400 copies/mL. The cervical lymph nodes expanded and overlying skin broke down.

Five months after starting antituberculous therapy, the patient had a grand mal seizure. A CT scan of the brain was obtained, followed by an MRI. The MRI showed enhancing lesions with surrounding vasogenic edema, compatible with tuberculoma. The diagnosis of tuberculoma was confirmed by stereotactic biopsy, which demonstrated no viable mycobacteria. A course of oral prednisolone was started. The patient continued to receive this therapeutic regimen, and abatement of the intracranial lesions was monitored radiologically.

It has been suggested that the growth of tuberculomas during treatment may be related to an immunologic process involving altered cell-mediated responsiveness in the context of mycobacterial killing during chemotherapy [1, 3]. Certainly, clinicians have long recognized that enlargement of regional lymph nodes occurs during chemotherapy [4].

The recent advent of widespread use of HAART in HIV disease has produced encouraging results in terms of reduction in HIV viral loads and some degree of immune reconstitution reflected by rising CD4 T lymphocyte counts and other indices [5]. Various speculations have been made about the clinical effects of such immunologic changes [6].

In our case, the expansion of intracranial tuberculomas in the context of HIV infection during quadruple primary therapy for miliary tuberculosis caused by a fully susceptible organism occurred after the introduction of HAART. We postulate that immune reconstitution may have contributed to the paradoxical expansion in this case, particularly as this paradoxical expansion is reported so rarely in association with HIV infection. Neurological features in patients with miliary tuberculosis who are receiving therapy should alert clinicians to the possibility of expansion of tuberculomas seeding to the meninges or CNS. The role of primary prophylactic corticosteroids is well established in treatment of tuberculomas meningitis, but data are lacking on the appropriateness of such treatment in the setting of miliary tuberculosis, where, by definition, involvement of the CNS is possible. We expect that others may notice changes in the clinical courses of their patients who are coinfected with tuberculosis and HIV and who are concurrently receiving HAART.

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References
Biliary Tract Infection Due to Bile-Soluble Bacteria: An Intriguing Paradox

Streptococcus pneumoniae and Haemophilus parainfluenzae exhibit autolysis in bile; thus, their isolation from bile is unexpected.

Nevertheless, both organisms have been linked to biliary infections [1–3]. In addition, Haemophilus parahaemolyticus has been implicated in empyema of the gallbladder [4]. In this report, we present five cases of biliary tract infection due to bile-soluble bacteria and review the literature.

Patient 1. A 1-year-old female with congenital biliary atresia managed by portoenterostomy and subsequent living-related-donor liver transplantation developed repeated episodes of fever and chills and had impaired liver function 2 months after transplantation. Serial ultrasonograms showed progressive dilation of intrahepatic bile ducts, which required repeated percutaneous transhepatic cholangiography with balloon dilation, percutaneous transhepatic biliary drainage (PTBD), and iv antibiotics. Micro-