**Extragenital Mycoplasma hominis Infection in Two Liver Transplant Recipients**

*Mycoplasma hominis* has been recognized as an extragenital pathogen in immunocompromised hosts, including kidney and heart transplant recipients; patients with systemic autoimmune diseases, lymphomas, or leukemia; and patients who have undergone cardiac surgery or who have experienced trauma [1, 2]. Liver transplantation has rarely been reported to increase host susceptibility to intraabdominal *M. hominis* infection [1, 3, 4].

We describe two liver transplant recipients with extragenital *M. hominis* infection, and we emphasize the need for physicians to be aware of the pathogenicity of *M. hominis* for this group of patients. The isolation of *M. hominis* from the pleural space and the abdominal surgical wound in the first case and from an intrahepatic abscess in the second one indicates that *M. hominis* infection in sites other than the peritoneal cavity should be considered. We furthermore propose the use of sequence determination of the *tuf* gene [5–7] for rapid species diagnosis to accelerate clinical decisions.

A 48-year-old male patient underwent orthotopic liver transplantation for treatment of hepatocellular carcinoma and liver cirrhosis associated with hepatitis B virus. The patient developed fever, pleural effusions, and signs of abdominal wound infection in the fifth week after transplantation. The results of standard bacteriologic examination of the pleural fluid and of wound swabs were unremarkable; after 3–4 days, translucent pinpoint colonies appeared on Columbia agar and on chocolate agar. Subculture on supplemented mycoplasma agar (Oxoid, Basingstoke, UK) revealed colonies with the “fried-egg” appearance that is typical for mycoplasmas after 2 days.

Before specific antibiotic therapy could be initiated, the patient developed fulminant and fatal *Klebsiella oxytoca* pneumonia that did not respond to initial treatment with piperacillin and tobramycin. The mycoplasma isolates were nonglycolytic and hydrolyzed arginine. The isolates were confirmed to be *M. hominis* by amplification of the gene encoding the elongation factor Tu (*tuf*) and by direct sequence determination of the PCR product as described previously [5–7].

Although the clinical relevance of the pleural and wound infection remained unclear in this patient's case, our experience prompted us to culture relevant extragenital specimens obtained from liver transplant recipients in mycoplasma agar (this agar was used in addition to standard diagnostic culture media). The value of using this agar was confirmed since 2 months later *M. hominis* was isolated from swab specimens from the peritoneal cavity of a female 29-year-old drug addict who had hepatitis B virus–induced acute liver failure that had been treated by liver transplantation. Peritonitis and fever had developed 4 weeks after transplantation due to partial necrosis of the bile duct, which was treated surgically by a hepatojejunostomy.

**Figure 1.** CT scan demonstrates liver abscess formation (arrow) due to *Mycoplasma hominis* in a 29-year-old liver transplant recipient.

*M. hominis* as well as coagulate-negative staphylococci and *Enterococcus faecium* were isolated from the swabs. Since the *tuf* gene was amplified from suspicious primary colonies and since subsequent sequence determination allowed *M. hominis* to be identified within 24 hours, treatment with ciprofloxacin [8, 9] was started immediately and continued for 12 days. Ciprofloxacin was administered with vancomycin for treatment of infection due to gram-positive cocci.

Despite initial improvement in the patient's condition, a CT scan revealed a subcapsular mass of the sixth hepatic segment 15 days after completion of the course of ciprofloxacin therapy (figure 1). Drainage of the abscess yielded 25 mL of pus. Bacteriologic analysis of the aspirate by initial plating on mycoplasma agar and sequencing of the *tuf* gene PCR product from suspicious colonies revealed intrahepatic *M. hominis* abscess formation. Aerobic and anaerobic culture of the abscess did not yield any other bacteria. The patient was then successfully treated with combination antibiotic therapy that included clindamycin and doxycycline [10] for 18 days and by irrigation of the abscess with sodium chloride solution for 10 days. She was discharged to her home 5 weeks after completion of the combination therapy, and a control CT scan was not performed.

Since there are only a few reports on extragenital *M. hominis* infections in liver transplant recipients, our two cases serve to emphasize the pathogenic potential of *M. hominis* for these patients. Bacteriologic laboratories that perform tests for liver transplant recipients should include mycoplasma agar in their routine diagnostic program to accelerate the diagnosis of *M. hominis* infection. *Tuf* gene amplification [5–7] directly from suspicious colonies followed by sequence determination of the PCR product allows diagnosis of the species within 24 hours and provides the diagnostic basis for rapid specific antibiotic therapy. Despite initial therapy with ciprofloxacin, a liver abscess developed in one patient; this abscess was then successfully treated by drainage and irrigation and with systemic antibiotic therapy (i.e., clindamycin combined with doxycycline).

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Exogenous Reinfection with Multidrug-Resistant Mycobacterium tuberculosis

Active tuberculosis in patients with prior tuberculous infection can occur after endogenous reactivation or exogenous reinfection. Endogenous reactivation results from reactivation of viable tubercle bacilli in dormant foci that persist after primary infection. Exogenous reinfection results from inhalation of new bacilli, causing active disease. Since the publication of Stead’s article in 1967 [1], exogenous reinfection has not been considered a significant cause of tuberculosis in individuals known to have had previous tuberculin reaction in the United States and other developed countries.

However, recent reports from the United States have documented exogenous reinfection with resistant Mycobacterium tuberculosis in patients with advanced HIV infection, alcoholism, and malnutrition [2–5]. We describe a diabetic patient with a history of tuberculosis who is receiving treatment for multidrug-resistant tuberculosis (MDRTB) apparently acquired through exogenous reinfection.

A 60-year-old Peruvian man was treated for pulmonary tuberculosis in 1951 with collapse therapy and intramuscular streptomycin. He moved to New York in 1953 and has worked in our hospital’s housekeeping department since 1971. In 1974, he underwent a tine test done at the employee health service, which was positive. Other than adult-onset diabetes mellitus, well controlled with oral hypoglycemic agents, he had no other illnesses and took no other medications.

The patient was well until September 1994, when he developed a persistent productive cough and lost 20 pounds over the ensuing 2 months. In November 1994 a chest radiograph revealed patchy nodular infiltrates of the right lung and left-sided pleural thickening. No previous radiograph was available for review. He denied all risk factors for HIV infection and tested negative for antibodies to the virus. Multiple sputum specimens were smear positive for acid-fast bacilli, and cultures yielded M. tuberculosis that was resistant to seven antitubercular drugs, including all five first-line agents. Restriction fragment length polymorphism (RFLP) analysis revealed that this strain’s pattern was identical to that of a strain responsible for many MDRTB cases in New York City (Public Health Research Institute Tuberculosis Center [New York City] strain W) [6].

In January 1997 the patient was completing his 24th month of directly observed therapy, which consisted of daily oral ofloxacin, cycloserine, p-aminosalicylic acid, and pyridoxine; intramuscular capreomycin was included in this regimen during the first 6 months. His compliance with this regimen has exceeded 90%. After he started receiving this therapy, his sputum smears and cultures converted to negative within 2 months, and he had an excellent clinical and radiographic response.

The findings in this case illustrate probable exogenous reinfection of a diabetic man with MDRTB. Despite lack of culture confirmation of our patient’s original episode of tuberculosis, much evidence supports prior infection, including his description of a clinical picture consistent with tuberculosis, previous surgical and medical treatment for tuberculosis, and a positive tine test in 1974. In 1994 he had culture-confirmed tuberculosis, probably caused by a strain of M. tuberculosis different from that which caused his initial infection. His job involved cleaning respiratory isolation rooms of patients with suspected and known tuberculosis, including some patients with MDRTB whose isolates had a strain W pattern. Strain W, which apparently dates back to the 1980s, has been recovered primarily in New York City and has caused many cases of MDRTB, including nosocomial outbreaks [6]. This strain first appeared at our institution in November 1992. The RFLP pattern of the isolate from the patient described herein matched the pattern of isolates from other patients seen at our hospital.

This case, as well as those previously reported [2–5], demonstrates that prior tuberculous infection or disease does not provide all patients with adequate protective immunity to reinfection with a different strain of M. tuberculosis. These patients were all in groups considered to be at high risk of developing active disease after tuberculous infection [7]. The factors that make such patients more likely to develop disease when they are infected may also put them at greater risk of reinfection when exposed again to M. tuberculosis.

Exogenous reinfection is believed to be a significant cause of tuberculosis in developing countries and other areas where