Donor-to-Host Transmission of *Mycoplasma hominis* in Lung Allograft Recipients

Rebekah Gass, James Fisher,* David Badesch, Martin Zamora, Adriana Weinberg, Heather Melsness, Frederick Grover, Joseph G. Tully,† and Ferric C. Fang

From the Departments of Medicine, Pediatrics, Pathology, Cardiothoracic Surgery, and Microbiology, University of Colorado Health Sciences Center, Denver, Colorado

***Mycoplasma hominis* is a significant pathogen in immunocompromised hosts, particularly organ transplant recipients. We describe two recipients of lung allografts from the same donor who had *M. hominis* pleuropulmonary infection during the immediate postoperative period. The most likely source of infection in these cases was the donor's respiratory tract. The slow-growing pinpoint colonies formed by *M. hominis* on routine bacterial culture medium may be easily overlooked and should be subcultured to mycoplasmal medium for definitive identification. The recommended management of this infection consists of drainage and antimicrobial therapy with tetracycline, clindamycin, or a fluoroquinolone. This report highlights the potential for *M. hominis* to be transmitted from donor to recipient during organ transplantation.**

Mycoplasma hominis is a resident of the genital and respiratory tracts of healthy adults [1, 2] and has long been recognized as a urogenital and peripartum pathogen. Its role as an extragenital pathogen in immunocompromised hosts has more recently been appreciated [3]. We present two cases of *M. hominis* pulmonary infection in recipients of lung allografts from the same donor. These cases suggest the potential for transmission of *M. hominis* from donor to host during solid organ transplantation.

Case Reports

Case 1

A 65-year-old man received a right-sided lung transplant because of respiratory insufficiency related to α_1-antitrypsin deficiency. The donor, a 20-year-old man who had received a gunshot wound to the head, had been intubated for 24 hours; his chest radiograph before organ donation was unremarkable. The recipient received therapy with azathioprine, cyclosporin A, prednisone, ceftazidime, vancomycin, and trimethoprim-sulfamethoxazole postoperatively. His clinical course was complicated by hemorrhagic pleural effusion and persistent pneumothorax. On postoperative day 7 fever without respiratory deterioration developed. Physical examination revealed diffuse coarse rhonchi and a pleural friction rub. The peripheral WBC count was 21.1 $\times$ 10^9/L (95% neutrophils). Chest radiography revealed bilateral ill-defined infiltrates. Bacterial cultures of preoperative bronchial specimens from the donor were significant for only rare *Staphylococcus aureus*. Postoperative specimens of pleural fluid and bronchoalveolar lavage fluid contained moderate neutrophils without visible organisms. Aerobic culture yielded tiny punctate colonies on chocolate and blood agar after 48 hours. Gram staining of these colonies was negative, but organisms were visualized with use of acridine orange.

Case 2

A 50-year-old man with end-stage emphysema received the left lung from the same donor as in case 1. His postoperative course was uncomplicated, and his medications at discharge were cyclosporin A, azathioprine, prednisone, itraconazole, and trimethoprim-sulfamethoxazole. He returned to the clinic 5 days later with complaints of dyspnea and left-shoulder pain. Oxygen saturation was 90% while the patient was breathing 2 L of oxygen. His temperature was 38.3°C. Physical examination revealed left-sided crackles. The WBC count was 27.1 $\times$ 10^9/L. Chest roentgenography demonstrated a large left pleural effusion. The patient was admitted to the hospital, and empirical therapy with vancomycin and ceftazidime was started. Pleural fluid analysis showed the following: RBC count, 18,400/mm^3; WBC count, 6,800/mm^3 (85% neutrophils); glucose level, 88 mg/dL; protein level, 2.3 g/dL; lactate dehydrogenase level, 3,982 U/L; and pH, 7.39. Gram staining of the pleural fluid revealed moderate neutrophils but no organisms. Culture of the fluid demonstrated colonies similar to those in case 1. Review of records of the patient's earlier admission revealed that culture of postoperative bronchoalveolar lavage fluid yielded similar colonies.

Organisms from both patients were cultivated on A7 medium, and *M. hominis* was presumptively identified by the metabolism of arginine. At the time of identification, antibiotic therapy for both patients was changed to ciprofloxacin (750 mg twice a day). Identification was confirmed by fluorescent antibody testing at the National Institute of Allergy and Infectious Diseases. Both patients underwent chest tube drainage and received 4 weeks of ciprofloxacin therapy with abatement of symptoms.

Received 18 July 1995; revised 11 October 1995.

* Present affiliation: Wayne State University School of Medicine, Detroit.

† Present affiliation: National Institute of Allergy and Infectious Diseases, Frederick, Maryland.

Reprints or correspondence: Dr. Ferric C. Fang, Department of Medicine, University of Colorado Health Sciences Center, 4200 East Ninth Avenue, B-168, Denver, Colorado 80262.

Clinical Infectious Diseases 1996;22:567–8

1058–4838/96/2203–0027$02.00

Discussion

The major site of *M. hominis* colonization in adults is the genitourinary tract [1, 2], but the organism is found in the respiratory secretions from 1% to 3% of healthy individuals [4]. Disruption of the genitourinary or respiratory tract may lead to hematogenous dissemination or contiguous spread of infection. In several reviews [3, 5, 6], 67 adults with extragenital infections due to *M. hominis* were described, and immunocompromised patients appeared to be at greatest risk. In these reports 13 isolates were obtained from sputum, bronchoalveolar lavage fluid, lung tissue, or pleural fluid. Six isolates were recovered from recipients of organ transplants (heart, heart-lung, and pancreas). All six transplant patients died, although death was not directly attributable to *M. hominis* infection in most cases. Successfully treated *M. hominis* empyema in a heart-lung transplant recipient [7] and *M. hominis*–associated alveolar hemorrhage in a bone marrow transplant recipient have also been recently described [8]. *M. hominis* infections may cause serious nonpulmonary infections in kidney allograft and liver allograft recipients [9, 10].

The intrinsic pathogenicity of *M. hominis* appears to be low. In one review [5] 77% of extragenital infections resolved spontaneously or with drainage alone. However, *M. hominis* can cause significant morbidity and mortality in immunocompromised hosts [3, 8–10]. *M. hominis* infection should be considered when examination of body fluids demonstrates purulence with negative gram staining and cultures. Although marked peripheral leukocytosis is often seen, patients may not appear seriously ill [5], which provides a useful diagnostic clue.

M. hominis is the only pathogenic *Mycoplasma* species cultivatable on routine bacterial media such as chocolate or blood agar, but the characteristic translucent pinpoint colonies are easily overlooked. Such colonies should be subcultured to mycoplasmal medium. Blind subcultures of blood cultures to solid media are required as *M. hominis* produces little turbidity in liquid media [5] and may not trigger automated detection systems.

The important elements of therapy are drainage, debridement, and reduction of immunosuppression [3]. Although some infections resolve spontaneously, antimicrobial therapy is advisable, particularly for immunocompromised hosts. *M. hominis* is resistant to many agents used in the posttransplantation setting including β-lactam agents, trimethoprim-sulfamethoxazole, and vancomycin [11]. Susceptibility to aminoglycosides is variable [11]. Unlike *Mycoplasma pneumoniae, M. hominis* is resistant to erythromycin [11] as well as to newer macrolides. Tetracyclines are often considered the antimicrobial agents of choice, but the incidence of resistance appears to be increasing [11, 12]. Clindamycin is an alternative agent [11], and quinolones are also active agents [10, 11, 13, 14]. The use of combination therapy (e.g., clindamycin and doxycycline) has been recommended [6].

This report describes the remarkable occurrence of respiratory infection due to *M. hominis* in two recipients of lung allografts from a single donor. Although *M. hominis* was not identified by routine cultures of preoperative bronchial specimens from the donor, selective procedures for isolating *Mycoplasma* species were not employed. Isolation of *M. hominis* from pleuropulmonary secretions from both recipients in the immediate postoperative period implicates the donor's respiratory tract as the site of origin. To our knowledge, transmission of this strongly host-associated microbe by fomites has never been demonstrated, thus making an environmental source highly unlikely. Recipients of other organs (heart, kidney, and liver) from the same donor had no postoperative evidence of *M. hominis* infection. *M. hominis* may be added to the expanding list of pathogens transmissible from donors to allograft recipients [15]. Because *M. hominis* infection is probably underrecognized, further study is needed to clarify its actual incidence in the transplantation setting as well as optimal preventive and therapeutic measures.

References

1. Taylor-Robinson D, McCormack WM. The genital mycoplasmas: first of two parts. N Engl J Med **1980**;302:1003–10.
2. Taylor-Robinson D, McCormack WM. The genital mycoplasmas: second of two parts. N Engl J Med **1980**;302:1063–7.
3. Meyer RD, Clough W. Extragenital *Mycoplasma hominis* infections in adults: emphasis on immunosuppression. Clin Infect Dis **1993**;17(suppl 1):S243–9.
4. Mufson MA. *Mycoplasma hominis:* a review of its role as a respiratory tract pathogen of humans. Sex Transm Dis **1983**;10:335–40.
5. Madoff S, Hooper DC. Nongenitourinary infections caused by *Mycoplasma hominis* in adults. Rev Infect Dis **1988**;10:602–13.
6. McMahon DK, Dummer JS, Pasculle AW, Cassell G. Extragenital *Mycoplasma hominis* infections in adults. Am J Med **1990**;89:275–81.
7. Boyle EM Jr, Burdine J, Bolman RM III. Successful treatment of mycoplasma mediastinitis after heart-lung transplantation. J Heart Lung Transplant **1993**;12:508–12.
8. Kane JR, Shenep JL, Krance RA, Hurwitz CA. Diffuse alveolar hemorrhage associated with *Mycoplasma hominis* respiratory tract infection in a bone marrow transplant recipient. Chest **1994**;105:1891–2.
9. Miranda C, Carazo C, Banon R, Mendoza J, Montes A, de la Rosa M. *Mycoplasma hominis* infection in three renal transplant patients. Diagn Microbiol Infect Dis **1990**;13:329–31.
10. Haller M, Forst H, Ruckdeschel G, Pratschke E. *Mycoplasma hominis* in liver transplantation [letter]. Surgery **1993**;113:359–60.
11. Bygdeman SM, Mardh P-A. Antimicrobial susceptibility and susceptibility testing of *Mycoplasma hominis:* a review. Sex Transm Dis **1983**; 10(suppl):366–70.
12. Cummings MC, McCormack WM. Increase in resistance of Mycoplasma hominis to tetracyclines. Antimicrob Agents Chemother **1990**;34: 2297–9.
13. Ridgway GL, Mumtaz G, Gabriel FG, Oriel JD. The activity of ciprofloxacin and other 4-quinolones against *Chlamydia trachomatis* and mycoplasmas in vitro. Eur J Clin Microbiol **1984**;3:344–6.
14. Furneri PM, Tempera G, Caccamo F, Speciale AM. In vitro activity of ciprofloxacin against clinical isolates and standard strains of mycoplasmas and chlamydiae. Rev Infect Dis **1988**;10(suppl 1):S55–6.
15. Gottesdiener KM. Transplanted infections: donor-to-host transmission with the allograft. Ann Intern Med **1989**;110:1001–16.