Perihepatitis and Perinephric Abscess Due to *Mycoplasma hominis* in a Kidney Transplant Patient

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***Mycoplasma hominis* has been incriminated in several genital and extragenital infections. Here, we report the first case of perihepatitis associated with a perinephric abscess in a woman who had received a kidney transplant. Four months after the transplant, the patient was admitted for perirenal allograft pain, fever, and elevated inflammatory parameters and liver enzyme levels. A renal ultrasonography found a collection of fluid. Results of blood and urine analyses were within normal limits. Fluid aspiration of the peritoneal cavity was performed, and the results of cultures for bacteria and fungi were negative. The patient was treated by surgical lavage of the peritoneal cavity. Her fever resolved 5 days later. Two months after surgical lavage of the peritoneal cavity, her liver enzyme levels returned to the normal range. Three months after surgical lavage, cultures of the perinephric fluid showed *Mycoplasma hominis*. We conclude that in patients who present with perinephric fluid suspected of being infected, bacteriologic analysis of the fluid (from surgical lavage of the peritoneal cavity) should be performed. Antibiotics active against intracellular bacteria should be administered.**

Key words: *Renal transplantation, Hepatitis, Infection, Perinephric space, Antibiotics*

Mycoplasma hominis is a common micro-organism found in the urogenital tract. *Mycoplasma hominis* has been incriminated in several genital and extragenital infections following organ transplant (1). Here, we report the first case of perihepatitis associated with a perinephric abscess in a kidney transplant patient.

Case Report

A 29-year-old white woman received a kidney transplant from a deceased-donor for end-stage renal failure of an undetermined etiology. Her immunosuppressive therapy was based on a quadruple sequential immunosuppressive therapy including induction therapy of basiliximab (20 mg at days 0 and 4), followed by cyclosporine (C2 target level, 1200-1400 ng/mL), mycophenolate sodium (720 mg b.i.d.), and steroids (prednisone). At day 10, she developed acute rejection, which was successfully treated with anti-CD3 monoclonal antibodies. Subsequently, she was converted from cyclosporine to tacrolimus (0.1 mg/kg/d, targeting a trough level of 8-12 ng/mL). At day 14, she developed multiresistant *Escherichia coli* acute pyelonephritis, which was treated by cefepime (1 g b.i.d. × 21 days) and amikacin (15 mg/kg/d × 5 days).

At discharge at 30 days, her serum creatinine level was 100 μmol/L. Her creatinine clearance, calculated according to the Cockcroft and Gault formula, was 1.169 mL/s/m^2, and her C-reactive protein level was within the normal range (< 66.668 nmol/L). Two ultrasonographies, 1 performed when the pyelonephritis was diagnosed and the other at discharge, revealed the presence of 2 regions of perinephric fluid (3 and 7 cm in diameter), which were considered to be residual lymphoceles. Three months after the pyelonephritis episode, her serum creatinine level, C-reactive protein, and the size of the fluid collections, as detected by ultrasonography, remained unchanged.

Four months after the transplant, the patient was admitted for perirenal allograft pain and fever (38°C). Results of a physical examination were unremarkable. Initial laboratory analyses showed the following: serum creatinine level, 104 μmol/L;

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C-reactive protein, 209.528 nmol/L; and white blood cell count, 9.8 × 10^9/L; alanine aminotransferase and aspartate aminotransferase levels were 1.169 μkat/L (normal range, 0.17-0.68 μkat/L) and 1.584 μkat/L (normal range, 0.17-0.51 μkat/L), respectively, and the γ-glutamyltransferase level was 7.335 μkat/L (normal range, 0.03-0.51 μkat/L). Urine culture results were negative for bacteria and fungi. Hepatitis B virus, hepatitis C virus, and cytomegalovirus viremia were not detected by polymerase chain reaction (standardized quantitative reverse-transcription polymerase chain reaction assay [Roche COBAS, Amplicor hepatitis C virus quantitative monitor assay; Roche Diagnostics, Branchburg, NJ, USA] for hepatitis C virus viremia; the Amplicor hepatitis B virus monitor test [Roche Diagnostics] for hepatitis B virus viremia; and real-time polymerase chain reaction for cytomegalovirus viremia). Hepatitis A virus serology (ie, IgM) was negative. The results of an abdominal ultrasonography were normal. In contrast, a renal ultrasonography showed a third region of fluid in addition to the previous two. A fluid aspiration was performed. The results of cultures for bacteria and fungi in the fluid were negative. Empiric antibiotherapy with imipenem (2 g/d) and gentamicin (3 mg/kg/d for 3 days) was initiated. Because of the persisting fever and elevated inflammatory parameters, and despite 10 days of antibiotic therapy, a surgical lavage of the peritoneal cavity was performed. Her fever disappeared 5 days later, and C-reactive protein level returned to its normal range Two months after surgical lavage of the peritoneal cavity, her liver enzyme levels returned to normal.

Three months after surgical lavage, the results of long-term cultures on routine blood agar of the perinephric fluid showed *Mycoplasma hominis*. Although *Mycoplasma hominis* was found on analysis of the urethral fluid (10^6 CFU/mL) of the patient and her husband 3 months after surgical lavage, in the absence of any fever or inflammatory syndrome, no antibiotics were administered. In contrast, *Mycoplasma hominis* serology was negative in the patient and her husband. Retrospectively, initial liver enzyme abnormalities were attributed to *Mycoplasma-hominis*–induced perihepatitis.

Discussion

In immunocompromised patients, *Mycoplasma hominis* is often responsible for extragenital infections of wounds, perinephric fluid, joints, and the central nervous system (1,2). We report the first case of perihepatitis associated with a perinephric abscess caused by *Mycoplasma hominis*.

In the setting of kidney transplants, *Mycoplasma hominis* infections have led to graft loss and death (3). *Mycoplasma hominis* infections occur mainly in severely immunosuppressed patients (4). Our patient developed an acute steroid-resistant rejection, which required anti-CD3 monoclonal antibody therapy for 4 months before the occurrence of the *Mycoplasma hominis* infections. Isolation of *Mycoplasma hominis* is difficult because it cannot be demonstrated by Gram stain; it grows slowly and requires specific media. This can delay the diagnosis of a *Mycoplasma hominis* infection. In the present case, the patient was treated by lavage of the peritoneal cavity rather than by antibiotics.

Hence, in patients who present with a perinephric fluid suspected of being infected, bacteriologic analysis of the fluid associated with surgical lavage of the peritoneal cavity should be performed. Antibiotics that are active against intracellular bacteria should be considered.

References

1. McMahon DK, Dummer JS, Pasculle AW, Cassell G. Extragenital *Mycoplasma hominis* infections in adults. Am J Med. 1990;89(3):275-281.
2. Madoff S, Hooper DC. Nongenitourinary infections caused by *Mycoplasma hominis* in adults. Rev Infect Dis. 1988;10(3):602-613.
3. Pastural M, Audard V, Bralet MP, et al. *Mycoplasma hominis* infection in renal transplantation. Nephrol Dial Transplant. 2002;17(3):495-496.
4. Meyer RD, Clough W. Extragenital *Mycoplasma hominis* infections in adults: emphasis on immunosuppression. Clin Infect Dis. 1993;17(suppl 1):S243-S249.