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Case Report

Urogenital mycoplasma: an emerging cause of deep wound infection after kidney transplantation?

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Case reports

We report here three cases of mycoplasma deep wound infections that occurred shortly after kidney transplantation during a 12-month period in our transplant unit. For all cases, the diagnosis of mycoplasma infection was definite since at least three different samples were positive for mycoplasma for each patient, and no other bacterial pathogens were obtained on standard cultures. Bacterial identification was performed using A7 Mycoplasma agar plates (bioMérieux, Marcy l'Etoile, France) and the Mycofast Evolution 3® commercial kit (International Microbio, Signes, France), and confirmed by 16S rRNA sequencing.

A 64-year-old diabetic male was admitted in July 2006 for a first kidney transplant. At Day 19, a sudden decrease of the urinary flow was noticed, associated with low-grade fever. On physical examination, the patient had an abdominal tenderness in the area of the graft and an inflammatory surgical wound with a urine-like liquid output. White blood cell count was 12 200/mm^3, and the CRP level rose to 228 mg/L. Numerous polymorphonuclear neutrophils were observed on the exteriorized liquid, contrasting with the absence of bacteria by Gram staining. Urine analysis showed significant leucocyturia (10^6/mm^3) without bacteriuria. Urine and blood cultures were all negative. An empirical antimicrobial therapy consisting of ceftriaxone, vancomycin and amikacin was started immediately before surgical revision that confirmed the diagnosis of urinary anastomosis leakage by necrosis of the anastomotic site. Examination of the purulent fluid taken from the wound collection showed numerous polymorphonuclear neutrophils but no identifiable microorganism by Gram staining. Two days later, bacteriological cultures of the peroperative liquids collected from the wound collection and of the urine revealed numerous pinpoint translucent colonies on blood agar plates that did not take up Gram staining. The colonies were transferred on a specific A7 Mycoplasma agar plate. Numerous typical *Mycoplasma hominis* 'fried-egg' colonies and a small number of dark brown colonies of *Ureaplasma sp.* were observed 48 h later. Confirmatory tests realized in the French Reference Laboratory for Mycoplasma (Bacteriology Department, Bordeaux, France) confirmed the identification of *M. hominis* and *U. urealyticum* and showed that the *M. hominis* isolate was susceptible to ofloxacin and moxifloxacin (MICs of 0.5 and 0.06 mg/L, respectively) whereas the *U. urealyticum* isolate showed only intermediate susceptibility to ofloxacin but adequate susceptibility to moxifloxacin (MICs of 2 and 0.5 mg/L respectively). The intravenous treatment was therefore replaced by oral moxifloxacin (400 mg/day) and continued for 17 days with a favourable outcome.

The second case occurred in February 2007 in a 55-year-old diabetic woman who experienced fever and a perinephric collection 25 days after a first kidney transplantation. The collection was drained and the suction drain liquid was sent for microbiological analysis. Typical *M. hominis* colonies grew on Mycoplasma agar plates. The outcome was rapidly favourable under oral levofloxacin therapy.

Another wound infection due to *M. hominis* was diagnosed in June 2007 in a 52-year-old patient who experienced a urinary leak associated with fever 4 days after kidney transplantation. The CT scan revealed two perinephric collections, and the urinalysis showed aseptic leucocyturia. The collections were drained and an empirical antimicrobial therapy including ceftriaxone and amikacin was started. Bacterial cultures only grew numerous typical colonies of *M. hominis*. The patient rapidly recovered under oral levofloxacin therapy.

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Discussion

M. hominis and *U. urealyticum* are commensal bacteria of the urogenital tract in sexually active men and women. They are usually associated with localized infections (urethritis, vaginosis and perinatal infections) but can induce extragenital infections in immunosuppressed patients [1,2]. In patients undergoing renal transplantation, mycoplasma deep wound infections are rare since only 20 cases have been published since 1982 and *M. hominis* was involved in all cases [3,4]. These infections are associated with a significant morbidity and mortality in the early post-transplant period [3]. To our knowledge, we report here the first *M. hominis* and *U. urealyticum* co-infection diagnosed after kidney transplantation.

The clinical diagnosis of mycoplasma infections after kidney transplantation is a difficult task because of the rarity of the disease and because of the latent and pleomorphic clinical presentation although, it can be suspected when clinical samples show the presence of purulent material but with negative smears. The microbiological diagnosis of mycoplasma infections is also difficult unless samples are directly transferred to specific culture media since this bacterium grows very slowly on blood agar plates and does not take up Gram stain [5,6]. The three cases presented here were all associated with an anastomosis leakage that appeared to be the obvious consequence of the infectious process as none of the predisposing factors were identified. Moreover, the involvement of other microorganisms in the infectious process is unlikely since in these cases, repeated cultured samples were negative except for colonies of mycoplasma. In the first case where a co-infection with *M. hominis* and *U. urealyticum* was diagnosed, the respective role of each of these two pathogens in the infectious process cannot be formally determined. It has been postulated that *U. urealyticum* may have a lower pathogenicity outside the urinary tract. Indeed, it has been shown to colonize urinary tract of renal allograft recipients without any infectious symptoms [7], and severe post-kidney transplant infections involving *U. urealyticum* have only been described in highly immunocompromised patients, resulting in disseminated infection in a hypogammaglobulinaemic patient [8] and multiple abscess formation in a patient previously treated with an anti-CD20 monoclonal antibody for a post-transplant lymphoproliferative disorder [9].

The occurrence of three post-transplant infections involving *M. hominis* in a single transplant unit during a 12-month period is unusual. The immunosuppressive regimen of these patients was similar to that of other patients admitted for kidney transplantation during the same period, consisting of an induction with basiliximab, low-dose steroids, cyclosporine and mycophenolate mofetil. Interestingly, these three cases occurred 1 year after a modification of the surgical antimicrobial prophylaxis that formerly included ofloxacin and that was replaced by amoxicillin–clavulanic acid. Contrary to fluoroquinolones that are usually highly active against mycoplasma species, mycoplasma are naturally resistant to broad-spectrum antibiotics that are commonly used for perioperative prophylaxis (usually beta-lactam antibiotics), as well as to *Pneumocystis jirovecii* prophylaxis (trimethoprime-sulfamethoxazole) [10]. Fluoroquinolones have been commonly used for initial or empirical treatment of urinary tract infections in this population, which is probably the major reason for the paucity of mycoplasma infections reports in the literature. The emergence of mycoplasma infections after kidney transplantation in our unit may have been facilitated by this change of surgical prophylaxis. As increasing quinolone resistance to common uropathogenic bacteria reduces their use for initial and empiric treatment [11], infections due to mycoplasma may become more common and should be considered in the differential diagnosis of surgical-site infections.

Conflict of interest statement. None declared.

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