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Case Report

Perinephric Abscess Attributed to *Mycoplasma hominis* Identified Through Next-Generation Sequencing in A Renal Transplant Recipient

Yunheng Zhou[1], Jing Liu[2], Weihua Xu[3], Min Min[4*]

[1]Department of Clinical Laboratory, Zhabei Central Hospital of Jing'an District, Shanghai, China

[2]Department of Radiology, Zhabei Central Hospital of Jing'an District, Shanghai, China

[3]Department of Ultrasonography, Zhabei Central Hospital of Jing'an District, Shanghai, China

[4]Department of Kidney Transplantation, Zhabei Central Hospital of Jing'an District, Shanghai, China

Yunheng Zhou and Jing Liu contributed equally to this work.

[*]**Corresponding author:** Min Min, Department of Kidney Transplantation, Zhabei Central Hospital of Jing'an District, No. 619, Zhonghuaxin Road, 200070, Shanghai, China

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Abstract

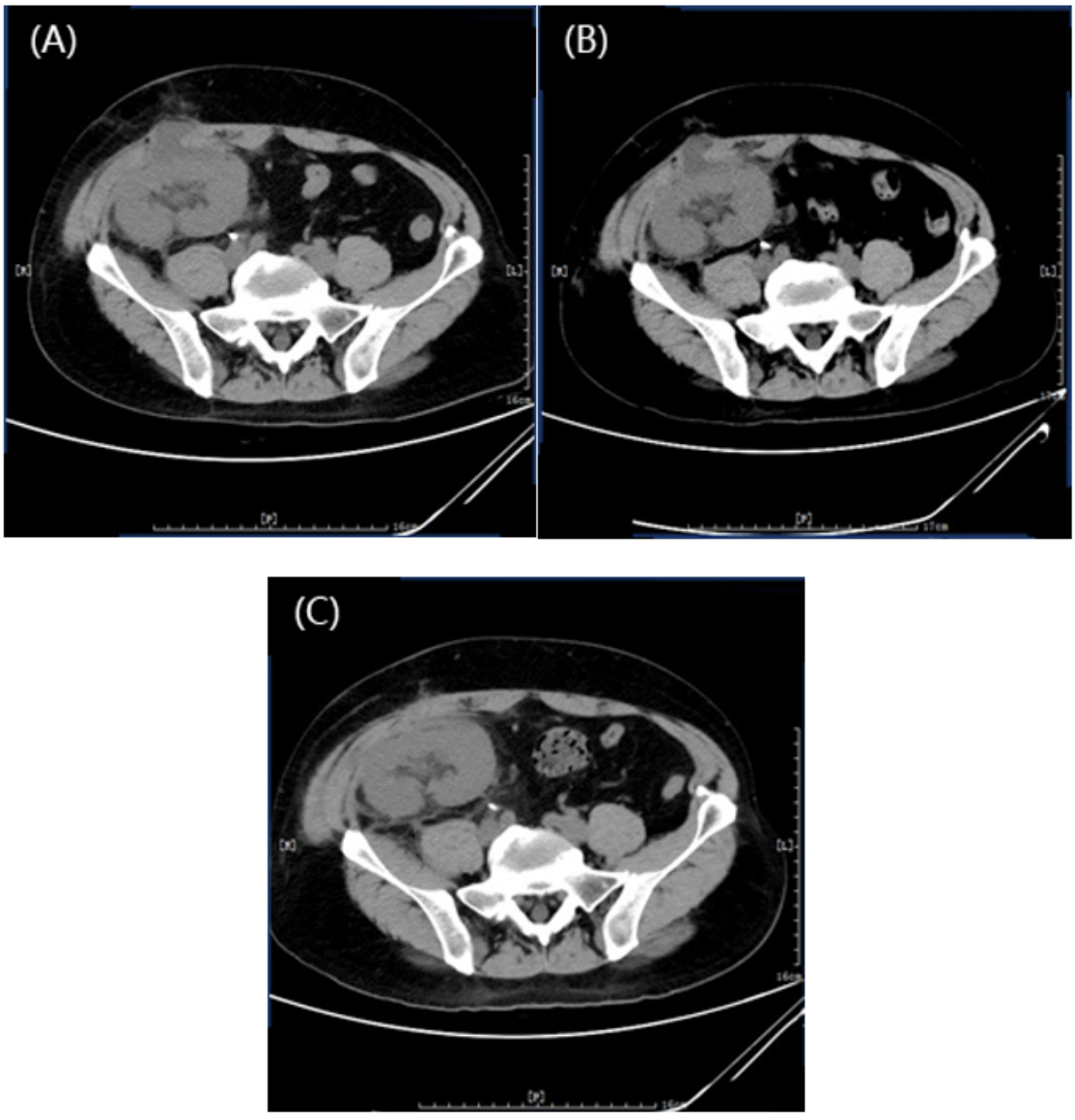
Mycoplasma hominis (M. hominis), typically regarded as a commensal microorganism, can sometimes be overlooked as a pathogen capable of causing both genitourinary and extragenital infections. In this case report, we present a male patient who underwent a renal transplant and, twenty days later, was admitted due to perirenal allograft pain. Abdominal computed tomography revealed perirenal effusion, yet bacterial and fungal cultures from the collected purulent fluid at the incision site yielded negative results. However, M. hominis was successfully detected using metagenomic next-generation sequencing. The patient underwent a 14-day course of minocycline treatment, with daily surgical dressing changes. Tacrolimus levels were closely monitored and adjusted during follow-up. Remarkably, the patient's symptoms showed significant improvement by day 46. In conclusion, we recommend vigilant monitoring of tacrolimus levels to manage the infection effectively, and we emphasize the importance of considering M. hominis as a potential pathogen in renal transplant recipients, mainly through specialized culture or molecular techniques.

Keywords: *Mycoplasma hominis*; Next-generation sequencing; Perinephric abscess; Renal transplantation; Tacrolimus

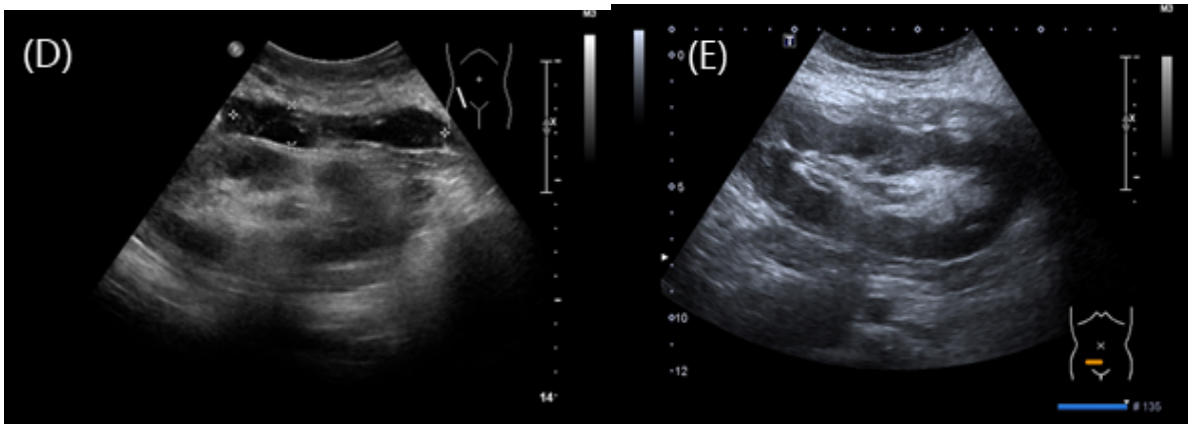
Introduction

Mycoplasma hominis (M. hominis), typically residing as a commensal microorganism in the urogenital tract, presents an intriguing yet sometimes underestimated pathogenic potential, giving rise to a spectrum of genitourinary and extragenital infections, including neonatal cases. Among extragenital infections, infective endocarditis and abscess formation are notably common, particularly in individuals with immunosuppression, accounting for approximately 50% of documented cases [1,2]. While M. hominis is conventionally viewed as having low pathogenicity and remains a subject of controversy, it can culminate in severe infections with adverse outcomes in immunocompromised patients [3]. In this context, we present a unique case of acute gastroenteritis coexisting with a perinephric abscess, both attributed to M. hominis, in a renal transplant recipient occurring within one month post-transplantation. Our patient, a 50-year-old male, underwent renal transplantation from a donor following cardiac death, subsequently receiving immunosuppressive therapy comprising tacrolimus (FK506), Miff, and Medrol (with the main active ingredient being Methylprednisolone). The primary active component of Miff is mycophenolic acid, primarily employed for

the prevention of acute rejection. On postoperative day 21 (POD 21), he presented with distending pain in the transplanted area, accompanied by fatigue and anorexia. Consequently, he sought medical attention at the kidney transplantation department on POD 23. Abdominal Computed Tomography (CT) imaging unveiled perirenal effusion, exudation, and infectious lesions extending from the perirenal area to the right anterior abdominal wall (Figure 1 A-C). Renal color ultrasonography corroborated the presence of a fluid collection (Figure 1 D,E).



Fingure 1 (A-C): Abdominal computed tomography scan displaying perirenal effusion with inflammatory exudate around the transplanted kidney.



Fingure 1 (D,E): Renal color ultrasonography depicting a collection of fluid.

Initial laboratory investigations revealed a white blood cell count of 7.2 ×109/L, serum C-reactive protein level < 0.5 mg/L (normal 0-10), BUN 13.7 mmol/L, creatinine 176.6 μmol/L (normal 70-115), cystatin C 2.31 mg/L (normal 0.55-1.05), and IL-6 at 37.77 pg/mL (normal 0-5.4). Urinary proteins exhibited elevated levels: microalbumin at 69.3 mg/L (normal 0-30), microglobulin at 56.3 mg/L (normal 0-12), transferrin at 3.0 mg/L (normal 0-2.5), and IgG at 16.3 mg/L (normal 0-9.6). Blood culture results returned negative. Purulent fluid sampled from the incision underwent culture and analysis through metagenomic next-generation sequencing (mNGS). The results of these cultures for bacteria and fungi yielded negative outcomes, while M. hominis was identified via PMseq-DNA analysis at the Beijing Genomics Institute. Notably, mNGS did not detect any viruses, fungi, parasites, or Mycobacterium tuberculosis. Suspected background microorganisms included gram-positive Staphylococcus and gram-negative Cutibacterium, as indicated in Table 1.

	Genus	The total number of sequences	Species	The total number of sequences
Detected pathogen	*Mycoplasma*	15,987	*Mycoplasma hominis*	15,499
Suspected background microorganism	*Staphylococcus*	5	*Staphylococcus hominis*	4
Suspected background microorganism	*Cutibacterium*	*3*	*Cutibacterium acnes*	3

Table 1: Genus and the sequence number detected by metagenomic next-generation sequencing.

The renal transplantation procedure progressed uneventfully, with the initiation of triple immunosuppressive therapy consisting of tacrolimus (FK506) at a dosage of 2.0 mg/day, Miff at 720 mg/day, and Medrol at 4 mg/day, aimed at preventing postoperative infections. The patient was diagnosed with renal transplant status complicated by acute gastroenteritis and a perinephric abscess attributed to M. hominis. Treatment involved a 14-day regimen of minocycline (100 mg bid on the first day, followed by 50 mg bid for 13 days) in conjunction with cefminox (2 g/day), accompanied by daily drainage and dressing changes. Subsequently, the patient's symptoms progressively ameliorated, with significant resolution of the abscess. On day 46, the patient was discharged home without experiencing any relapses. Follow-up assessments revealed abdominal CT imaging demonstrating near-complete absorption of the encapsulated effusion. Laboratory analyses indicated BUN levels of 9.1 mmol/L and creatinine levels of 155.8 μmol/L, all within normal limits. On POD 71, following treatment with triple immunosuppressive therapy, CT scans, and color ultrasonography reported no evidence of effusion or fluid accumulation around the perirenal area. Urinary proteins were within the normal range, except for microglobulin at 34.1 mg/L. Notably, BUN and creatinine levels remained stable, while IL-6 levels returned to normal. Tacrolimus levels ranged between 5 and 8 ng/mL from day 46 to day 71. At the 12-month follow-up, no perinephric abscess was detected, and the patient was in robust health, devoid of any signs of infection. Figure 2 illustrates the concentration curve of tacrolimus during the treatment course.

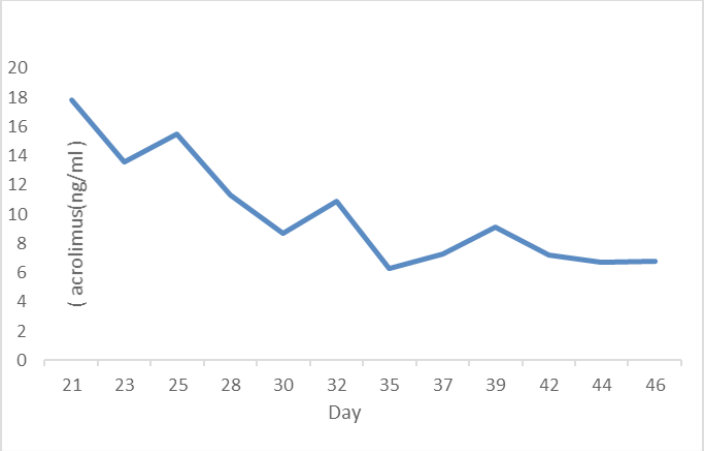


Figure 2: Concentration curve of tacrolimus during the follow-up.

Discussion

In most renal transplant centers worldwide, tacrolimus forms an integral part of immunosuppressive regimens post-transplantation, designed to forestall organ rejection. Immediate post-transplantation, it is imperative to consistently monitor tacrolimus trough levels and fine-tune dosages to strike a delicate balance between insufficient immunosuppression and excessive immunosuppression [4,5]. Presently, there exists no universally endorsed standard for the optimal blood concentration range. The target tacrolimus trough levels vary, encompassing 10-15 ng/mL within the first three months [6], 8-16 ng/mL from day 0 to day 14, and 5-15 ng/mL thereafter [7], or 8-12 ng/mL during the initial three months, 8-10 ng/mL from months 4 to 6 post-transplant, and 6-8 ng/mL subsequently [8,9]. During the initial 14 days from day 21 to day 35 in this case, tacrolimus blood concentrations consistently exceeded 8 ng/mL, particularly within the initial 7 days, surpassing 10 ng/mL. Elevated tacrolimus concentrations can exacerbate infections. In our clinical practice, in accordance with the experience of our clinicians, we maintain tacrolimus trough levels between 6-8 ng/mL during the first three months.

Throughout the patient's follow-up, renal function levels displayed a transient elevation, notably during the admission episode for perirenal allograft pain, with creatinine at 176.6 μmol/L, cystatin C at 2.31 mg/L, and heightened urinary protein levels. Numerous studies have indicated that cystatin C, as a serum marker, exhibits greater sensitivity in reflecting the glomerular filtration rate in post-renal transplant patients compared to serum creatinine. Nevertheless, cystatin C's specificity is indicated to be lower than that of creatinine [10]. Importantly, serum cystatin C levels remain relatively stable and unaltered by external factors [11]. The increase in renal function levels could be attributed to ischemia-reperfusion injury during renal transplantation. Furthermore, the decline in the recipient's residual renal function contributes to heightened renal function levels and urinary protein levels. Conversely, a decrease in renal function levels is conducive to infection control and renal function recovery. Serum creatinine levels decreased by 11.8% upon the patient's discharge and by 17.7% upon complete recovery compared to levels during admission. Urinary proteins normalized, except for microglobulin. These changes underscore the effectiveness of renal function control and deliberate renal function reduction in infection management.

Traditionally, patients are initially treated with empiric antimicrobials, typically beta-lactam or cephalosporin agents, for extended periods, often obviating the need for anti-mycoplasma drugs. However, an increasing number of reports have shed light on severe and occasionally fatal infections caused by Ureaplasma spp. and Mycoplasma spp. in immunocompromised patients, particularly renal transplant recipients. These infections manifest atypical sites such as the bloodstream, joints, perinephric fluid, or wound sites, thereby challenging the historically benign reputation of these microorganisms [12,13]. Hinić's work also advocates screening young, sexually active deceased and living kidney donors to mitigate immunosuppression-associated post-transplant infections involving these facultative pathogens [14]. It is not uncommon for patients to initially present with graft site pain or fever, only to subsequently be diagnosed with a perinephric abscess. The utilization of rapid diagnostics and recognition of M. hominis's pathogenic role within the organ transplant population proved invaluable in our case. Moreover, the incidence of these infections may be underreported due to the challenges in detecting Mycoplasma using conventional microbiological diagnostic methods. In our case, cultures of the preservation fluid from the donor's kidney failed to detect Ureaplasma and M. hominis, leading us to hypothesize that the donor might have been colonized or infected in the urogenital system. Consequently, detecting M. hominis should not solely rely on routine bacteriological culture. According to our clinical experience, parallel testing with a liquid culture medium and selective solid agar (A8) can reliably identify Ureaplasma spp. and M. hominis, ensuring specificity and accuracy [15-16]. Notably, next-generation sequencing (NGS) emerged as an invaluable tool for epidemiological investigations, notably shortening hospitalization times, even for slow-growing bacterial pathogens [17].

Conclusion

In this report, we present a case of perinephric abscess induced by M. hominis occurring 20 days after renal transplantation. We advocate for the routine monitoring of tacrolimus levels and dose adjustments to strike an optimal balance in infection control. Mycoplasma should be contemplated as a potential etiological

agent in perinephric abscess cases among renal transplant recipients, necessitating specialized culture or molecular techniques such as next-generation sequencing for detection. Controlling renal function and deliberately inducing a reduction in renal function levels prove to be highly effective strategies in infection management.

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