



## Case report

# *Mycoplasma hominis* hematoma infection in patient following kidney transplant



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## ABSTRACT

*Mycoplasma* species, specifically *Mycoplasma hominis* (*M. hominis*), are commonly associated with genitourinary (GU) tract infectious syndromes. However, *Mycoplasma* spp. can also be involved in extragenital infections, primarily in immunosuppressed patients. A 61 year old female was successfully treated with moxifloxacin and doxycycline combination therapy for an infected hematoma secondary to *M. hominis* following a renal transplant. Microbiology technologists noted the growth of pinpoint, translucent non-hemolytic colonies, but no organisms seen on Gram stain. These findings prompted the updated culture report of, "Growth on culture plates, gram stain suggestive of organism lacking cell wall." Empiric antimicrobials were initiated to cover both *Mycoplasma* spp. and *Ureaplasma* spp before resulting *M. hominis*. Initiating empiric therapy directed against *Mycoplasma* spp. following Gram stain results and before organism speciation may prevent a lapse in effective therapy. This is especially important as perioperative antimicrobial prophylaxis regimens consist of beta-lactam regimens directed against common GI and GU pathogens, which lack activity against *Mycoplasma* spp.

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## Introduction

*Mycoplasma* species, specifically *Mycoplasma hominis* (*M. hominis*), are commonly associated with genitourinary (GU) tract infectious syndromes. However, *Mycoplasma* spp. can also be involved in extragenital infections, primarily in immunosuppressed patients. Existing literature of *M. hominis* associated with extragenital infections include mediastinitis, sternal wound infections, pericarditis, septic arthritis, and prosthetic valve endocarditis [1].

Limited literature exists describing infected hematomas with *M. hominis* following kidney transplantation, with current data largely involving liver transplantation, pelvic trauma, and transplant nephrectomy [2–5]. We report the successful treatment of *M. hominis* infected hematoma following a renal transplant with moxifloxacin and doxycycline combination therapy. Initiating empiric therapy directed against *Mycoplasma* spp. following Gram stain results and before organism speciation may prevent a lapse in effective therapy.

## Case report

A 61 year old female with a past medical history significant for type II diabetes complicated by retinopathy, cerebral vascular accident, obstructive sleep apnea, and end stage renal disease secondary to diabetic nephropathy on peritoneal dialysis (PD) presented to the hospital for a deceased donor kidney transplant. Induction therapy consisted of anti-thymocyte globulin and methylprednisolone. She was subsequently started on mycophenolate, tacrolimus, and prednisone post transplantation. The surgical transplant procedure was complicated by hypoperfusion of the renal allograft requiring re-do of arterial anastomosis, and persistent leukocytosis. An abdominal CT was done on post-operative day (POD) 8 and demonstrated a large right retroperitoneal and perinephric hematoma measuring 20 cm. The patient was started on piperacillin/tazobactam for empiric coverage of an intra-abdominal infection and also coverage of *Aerococcus* spp. that was isolated from the urine culture. On post-operative day (POD) 9, IR placed a 14 F locking drainage catheter within the right lower quadrant perinephric fluid collection yielding 240 mL of seroanguinous fluid that was sent for bacteriological culture. Gram staining of drain fluid suggested bacteria without the presence of a cell wall. Given concern for *Mycoplasma* spp. and *Ureaplasma* spp., oral doxycycline 100 mg twice daily and oral moxifloxacin 400 mg once daily were started empirically prior to speciation.

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**Table 1**  
Microbiology.

Date	Specimen Information	Gram Stain	Culture Result
9/18/20	Hematoma; aspirate	2+ WBCs, no bacteria seen	No growth
9/22/20	Hematoma; aspirate	2+ WBCs, no bacteria seen	3+ Growth on culture plates, gram stain suggestive of organism lacking cell wall. Identification in progress. <i>Consider ID consultation.</i>
9/24/20	Hematoma; aspirate	2+ WBCs, no bacteria seen	3+ <i>Mycoplasma hominis</i>
9/22/20	Peritoneal; fluid	3+ WBCs, no bacteria seen	No growth
10/2/20	Peritoneal; fluid	3+ WBCs, no bacteria seen	1+ <i>Mycoplasma hominis</i>
9/22/20	Peritoneal dialysis catheter tip	3+ WBCs, no bacteria seen	No growth
9/27/20	Peritoneal dialysis catheter tip	3+ WBCs, no bacteria seen	2+ <i>Mycoplasma hominis</i> , no anaerobes isolated
9/23/20	Peritoneal; swab	3+ WBCs, no bacteria seen	No growth
10/2/20	Peritoneal; swab	3+ WBCs, no bacteria seen	1+ <i>Mycoplasma hominis</i> <i>Identified by DNA sequencing</i>

The patient continued to have worsening right sided flank pain and worsening leukocytosis. On POD 13, repeat urine and blood cultures were obtained and the PD catheter was removed. A repeat CT of abdomen and pelvis on POD 14 demonstrated decreased size of the hematoma, but also a new incisional seroma. This was opened at the bedside with vacuum-assisted closure of the wound. Ultimately, cultures from peritoneal fluid, swab, and peritoneal dialysis catheter tip all speciated to *Mycoplasma hominis* (Table 1). The patient responded clinically to source control and antibacterial therapy and was discharged on POD 16 on both moxifloxacin and doxycycline. She was readmitted on POD 18 due to wound dehiscence and underwent wound irrigation and debridement on POD 22 with negative cultures. A follow up CT abdomen and pelvis was done on POD 27 as an outpatient and revealed interval increase in right perinephric hematoma and she was readmitted on POD 30 for drainage. A drain was placed on POD 31 with negative bacterial cultures. Repeat abdominal imaging on POD 40 showed decrease in size of right peritoneal hematoma and moxifloxacin and doxycycline were discontinued on POD 45. At outpatient follow-up visit after completion of antibacterials, the patient was reportedly doing well with no acute issues.

## Discussion

The fluid from the perinephric hematoma was set up for culture, with first witnessed growth four days later. This is expected of *M. hominis*, as it is a slow growing organism on both blood agar and chocolate media. At the time of growth, pinpoint, translucent non-hemolytic colonies, were noted which prompted performance of a Gram stain. No organisms were seen on Gram stain, but the specimen was significant for pink debris indicating the possibility of bacteria. These findings prompted the updated culture report of, "Growth on culture plates, gram stain suggestive of organism lacking cell wall." The isolate was identified as *Mycoplasma hominis* later that day by the Vitek<sup>®</sup> MS. However, *M. hominis* is not FDA cleared on the Vitek<sup>®</sup> MS, and thus identification required validation by 16S DNA sequencing. DNA sequencing results were available two days later and verified the identification of *M. hominis*. Susceptibilities for *M. hominis* are not routinely performed at our institution, as *Mycoplasma* spp. are usually an incidental finding and there is a lack of susceptibility validation for the organism.

Although sensitivity is poor, *M. hominis* can potentially be detected using methods routinely employed in the clinical microbiology laboratory. However, it requires highly skilled laboratory personnel to visually recognize colonies of *M. hominis* that appear as tiny, pinpoint colonies. From clinical experience, our microbiology technologists were able to recognize that the pink debris seen at the time of Gram stain could possibly be bacteria, specifically *Mycoplasma* spp. This prompted the technologists to

subculture the potential bacteria on chocolate agar and store it in an anaerobic environment to encourage better growth. Once adequate growth occurred, *M. hominis* was able to be identified through molecular methods.

Once the microbiology technologist updated the culture report indicating growth of an organism without a cell wall, the infectious diseases (ID) consult service transitioned antimicrobial therapy to moxifloxacin and doxycycline to provide combination empiric therapy against *Mycoplasma* spp. and *Ureaplasma* spp. Both species lack a cell wall and therefore are innately resistant to antibiotics that target penicillin binding protein (PBP), making conventional beta-lactam empiric therapy ineffective against these organisms. This demonstrates the importance of a proper Gram stain, as recognition of organisms without a cell wall can lead clinicians to prescribe targeted therapy for *Mycoplasma* spp. and *Ureaplasma* spp. Additionally, if the Gram stain is incorrectly interpreted as negative, it could lead to premature discontinuation of antimicrobials. In this patient case, it was imperative to utilize the Gram stain results to initiate active therapy while awaiting speciation.

Optimal antimicrobial therapy for *M. hominis* is not well defined. *Mycoplasma* spp. localizes and survives intracellularly, requiring the use of agents that have high intracellular concentrations, such as tetracyclines, macrolides, and fluoroquinolones. In this patient case, with a Gram stain revealing bacteria without a cell wall, the ID consult service chose to cover both *Mycoplasma* spp. and *Ureaplasma* spp. Tetracyclines may be effective against both genus of organisms, however some strains of *Mycoplasma* spp., particularly *M. genitalium*, have increasing resistance to doxycycline [6,7]. *Ureaplasma* spp. such as *Ureaplasma parvum* and *Ureaplasma urealyticum* have shown increased failure rates with tetracyclines and necessitate the use of fluoroquinolones. Of note, fluoroquinolones also have demonstrated activity against *Mycoplasma* spp [8]. In an attempt to provide coverage for both *Mycoplasma* spp. and *Ureaplasma* spp. and potentially provide synergistic therapy, the ID consult team recommended combination therapy with doxycycline and moxifloxacin. Combination therapy was continued even after the identification of *M. hominis*, given the lack of susceptibility testing, bacteriostatic effect of tetracyclines, and the patient's clinical improvement on therapy.

## Conclusion

The source of infection in our patient case is not clearly elucidated. It is postulated that the donor kidney was colonized with *M. hominis* and led to infection within the post-transplant hematoma of this immunocompromised patient. Donor-host transmission of *M. hominis* has been previously reported in a lung allograft recipient and a cardiothoracic transplant recipient [9,10]. Of note, the patient's donor had a urinary tract infection (UTI) with bladder trauma. This case highlights the importance of proper

microbiologic sampling, especially when patients are not responsive to empiric therapy. Lastly, when an organism is not identified on Gram stain because it lacks a cell wall, one should consider *M. hominis*, especially in the setting of immunosuppression. In our case, it was critical to have experienced and highly trained clinical microbiology technologists to recognize potential bacteria on Gram stain and pinpoint colonies on media. This is especially important as perioperative antimicrobial prophylaxis regimens consist of beta-lactam regimens directed against common GI and GU pathogens, which lack activity against *Mycoplasma* spp.

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### Consent

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### Author statement

Mackenzie Dolan, PharmD: Drafting of the manuscript and review and revisions of revised manuscript.

Zachary Elliott, PharmD: Drafting of the manuscript, critical editing of the manuscript, and addition of revisions for revised manuscript.

Christopher Arnold, MD: Critical editing of the manuscript and review of revised manuscript.

### Declaration of Competing Interest

The authors report no declarations of interest.

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