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MAJOR ARTICLE

Donor-Derived *Mycoplasma hominis* and an Apparent Cluster of *M. hominis* Cases in Solid Organ Transplant Recipients

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Background. Invasive and disseminated *Mycoplasma hominis* infections are well recognized but uncommon complications in solid organ transplant recipients. In a single center, a cluster of *M. hominis* infections were identified in lung transplant recipients from the same thoracic intensive care unit (ICU). We sought to determine the source(s) of these infections.

Methods. Medical records of the donor and infected transplant recipients were reviewed for clinical characteristics. Clinical specimens underwent routine processing with subculture on *Mycoplasma*-specific Hayflick agar. *Mycoplasma hominis* identification was confirmed using sequencing of the 16S ribosomal RNA gene. *Mycoplasma hominis* isolates were subjected to whole-genome sequencing on the Illumina NextSeq platform.

Results. Three lung transplant recipients presented with invasive *M. hominis* infections at multiple sites characterized by purulent infections without organisms detected by Gram staining. Each patient had a separate donor; however, pretransplant bronchoalveolar lavage fluid was only available from the donor for patient 1, which subsequently grew *M. hominis*. Phylo- and pangenomic analyses indicated that the isolates from the donor and the corresponding recipient (patient 1) were closely related and formed a distinct single clade. In contrast, isolates from patients 2 and 3 were unrelated and divergent from one another.

Conclusions. *Mycoplasma hominis* should be considered a cause of donor-derived infection. Genomic data suggest donor-to-recipient transmission of *M. hominis*. Additional patients co-located in the ICU were found to have genetically unrelated *M. hominis* isolates, excluding patient-to-patient transmission.

Keywords. *Mycoplasma hominis*; solid organ transplant; donor-derived infection.

Mycoplasma hominis is a fastidious bacterium not readily cultured using standard methods and not detectable by Gram staining due to the lack of a peptidoglycan cell wall [1]. It is a human commensal, found in 20%–50% of asymptomatic adult female urogenital tract specimens [2] and 1%–3% of respiratory tract specimens from healthy adults [3]. The pathogenicity of *M. hominis* is generally considered low but has long been associated with urogenital and peripartum infections [4]. It is known also to cause a variety of extragenital infections, including wound infection, septic arthritis, endocarditis, brain abscess, pneumonia, and bacteremia [5, 6]. Extragenital infection is usually associated with surgery, trauma, and/or immunosuppressive conditions. Invasive and disseminated *M. hominis* infections are well-recognized but uncommon complications in solid organ transplant (SOT) recipients [7–9].

In a single center, 3 cases of *M. hominis* infection were identified in close succession in recipients of bilateral lung transplants who were nursed in close proximity within the same thoracic intensive care unit (ICU). Donor microbiology was available for only 1 of the 3 cases, which retrospectively tested positive for *M. hominis* by both culture and molecular methods. We report on the investigation of these cases.

MATERIALS AND METHODS

Identification of *M. hominis*

Clinical specimens (bronchoalveolar lavage [BAL], sputum, blood culture, pleural fluid, joint tissue, and fluid) underwent routine processing as per local protocols, including Gram stain and culture on horse blood agar, chocolate agar, MacConkey agar, and/or nalidixic acid colistin agar, with incubation at 35°C in 5% CO_2 for 72 hours. Pinpoint colonies on these media, consistent with *M. hominis*, prompted subculturing on a *Mycoplasma*-specific medium: Hayflick agar (Media Preparation Unit, University of Melbourne), at 35°C in 5% CO_2. Growth was examined under a stereomicroscope at ×40 magnification and considered positive when colonies with typical "fried egg" morphology were present.

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Molecular confirmation of species identification was performed using 16S ribosomal RNA (rRNA) gene sequencing. DNA was extracted using the Isolate II Genomic DNA Kit (Bioline). A 798 base pair (bp) sequence of the 16S rRNA gene was amplified using primers described by Goldenberger et al [10]. The conditions used for this amplification are described in the Supplementary Data. Sequences were compared to the GenBank database (accessed May 2016).

Whole-Genome Sequencing, Phylogenomics, and Resistance Gene Detection

Five *M. hominis* isolates from our cluster and 9 unrelated historical isolates, previously submitted to the Microbiological Diagnostic Unit Public Health Laboratory for identification, were analyzed by whole-genome sequencing (WGS) (Supplementary Table A). Genomic DNA from pure isolates was extracted using the JANUS automated workstation with the Chemagic Viral DNA/RNA kit (PerkinElmer). Dual-indexed paired-end DNA was sequenced on the Illumina NextSeq 500 as described by the manufacturer. The raw sequencing data was uploaded to the European Nucleotide Archive under study accession PRJEB20455.

Paired-end sequence reads were analyzed using the public health bioinformatics pipeline, Nullarbor (https://github.com/tseemann/nullarbor), with sequence alignment and single-nucleotide polymorphisms (SNPs) identified by mapping the quality-trimmed paired-end reads to the *M. hominis* ATCC 23114 (Accession, NC_013511) reference genome. After sequence alignment, recombination was detected using Gubbins version 2.2.0 with default settings [11]. A maximum-likelihood phylogenetic tree was then inferred from the recombination-free polymorphic sites using IQ-tree version 1.4.3 [12] with the best-fit substitution model automatically selected during the run. Branch supports were calculated using the ultrafast bootstrap method and an Shimodaira–Hasegawa approximate likelihood ratio test [13] with 1000 replicates. Abricate (https://github.com/tseemann/abricate) was used to scan the SPAdes version 3.9.0 [14] assemblies for resistance genes in the ResFinder (https://cge.cbs.dtu.dk/services/data.php) database.

Patient Information

Clinical and epidemiological data were obtained from the patients' medical records at Alfred Health. The 3 patients' clinical course is summarized in Supplementary Figure 1. Individual patient consent was sought and granted from patients 1, 2, and 3 to participate in this study.

RESULTS

Patient 1

A 46-year-old woman underwent a redo bilateral sequential lung transplant for chronic allograft dysfunction (the initial lung transplant was for bronchiolitis obliterans secondary to seropositive rheumatoid arthritis in 2012). Her perioperative immunosuppression is summarized in Table 1. Day 1 posttransplantation, she became febrile and, despite broad-spectrum antimicrobials, developed progressive respiratory failure with bilateral interstitial infiltrates and pleural effusions, requiring bilateral anterior thoracotomies with evacuation of pleural collections and right lung decortication (Supplementary Figure 1). Repeated bacterial culture from multiple sites during these 2 weeks posttransplant were negative and the patient was treated for presumed acute rejection with pulsed methylprednisolone. On day 13, she developed an acute oligoarthritis involving the left elbow and right index finger proximal and distal interphalangeal joints requiring surgical drainage and debridement of involved joints. On day 19, routine culture of BAL identified typical pinpoint, nonhemolytic colonies of *M. hominis* on the horse blood agar. Subsequent targeted culture of tissue and fluid from the left elbow joint, metacarpophalangeal joint, pleural fluid, and terminal blood cultures isolated *M. hominis* (Table 2).

Table 1. Characteristics of Organ Recipients and the Peritransplant Microbiology and Medications

Characteristic	Patient 1	Patient 2	Patient 3
Clinical history			
Transplant	Redo bilateral lung for chronic allograft rejection	Bilateral lung	Redo bilateral lung for chronic allograft rejection
Underlying pathology	RA-related bronchiolitis obliterans	Non-CF bronchiectasis	CF-related bronchiectasis
Comorbidities	RA arthropathy, chronic kidney disease, depression	Posttraumatic spinal surgery, osteopenia	Pancreatic exocrine insufficiency, stage 2 chronic kidney disease, mitral valve prolapse, idiopathic inflammatory constrictive pericarditis, gastroparesis
Pretransplant microbiology			
Recipient	Oral flora	*Pseudomonas aeruginosa*	MDR *Pseudomonas aeruginosa*
Donor	MSSA	MSSA	*Candida albicans*
Perioperative medications			
Immunosuppression	Mycophenolate, methylprednisolone, tacrolimus, basiliximab	Azathioprine, methylprednisolone, tacrolimus, basiliximab	Mycophenolate, methylprednisolone, tacrolimus, basiliximab
Antimicrobials	Piperacillin-tazobactam	Piperacillin-tazobactam	Meropenem, nebulized colistin

Abbreviations: CF, cystic fibrosis; MDR, multidrug resistant; MSSA, methicillin-sensitive *Staphylococcus aureus*; RA, rheumatoid arthritis.

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Table 2. Identification of *Mycoplasma hominis* From Donor and Recipient Specimens

	Patient 1	Donor 1	Patient 2	Patient 3
Day posttransplant isolated specimen(s)	15	…	2	25
	BAL fluid, pleural fluid, blood, elbow and MCP joint fluid and tissue	…	BAL fluid, sputum	Hip joint fluid aspirate
HBA/chocolate agar	Pinpoint colonies	…	Pinpoint colonies	Pinpoint colonies
Gram stain	No organisms	…	No organisms	No organisms
Specimen	Blood culture isolate	Endotracheal aspirate	BAL fluid isolate	Hip joint aspirate isolate
Hayflick's agar	Fried egg colonies	Fried egg colonies	Fried egg colonies	Fried egg colonies
Gram stain	No organisms	No organisms	No organisms	No organisms
16S rRNA sequencing	*M. hominis*	*M. hominis*	*M. hominis*	*M. hominis*

Abbreviations: BAL, bronchoalveolar lavage; HBA, horse blood agar; MCP, metacarpophalangeal; rRNA, ribosomal RNA.

The patient received dual *M. hominis* therapy with moxifloxacin 400 mg daily and doxycycline 200 mg daily for 2 weeks followed by single-agent doxycycline for a further 4 weeks. The patient improved markedly and was discharged home on day 48 posttransplantation.

Patient 2

A 65-year-old man underwent a bilateral lung transplant for non–cystic fibrosis–related bronchiectasis. His perioperative immunosuppression and antimicrobial regimen is listed in Table 1. On day 3 posttransplantation, he developed a leukocytosis, worsening respiratory function, and bilateral interstitial pulmonary infiltrates. Culture of BAL fluid collected on day 2 and 4 posttransplantation isolated a pure growth of *M. hominis* (Table 2). He was treated with 10 days of moxifloxacin with significant clinical improvement and was discharged home on day 18 posttransplantation.

Patient 3

A 51-year-old man underwent redo bilateral lung transplantation for chronic rejection, having been originally transplanted in 2014 for cystic fibrosis–related bronchiectasis. Perioperative immunosuppression and the antimicrobial regimen are detailed in Table 1. On day 15 posttransplant, he developed new right hip and groin pain that continued to worsen, and by day 23 he was unable to bear weight. Ultrasound demonstrated a moderate-sized, uncomplicated right hip joint effusion. He underwent surgical washout and debridement of the right hip on day 29 with evacuation of purulent fluid. Subsequent culture of the hip joint tissue and fluid isolated a pure growth of *M. hominis* (Table 2). He received dual therapy with moxifloxacin 400 mg daily and doxycycline 200 mg daily for 2 weeks and subsequently single-agent doxycycline for a further 4 weeks. His pain and mobility returned to his premorbid state and he was discharged home on day 37 posttransplantation.

Organ Donor

The donor (donor 1) for patient 1 was a previously well young adult female who developed a hypoxic brain injury following cardiac arrest. Culture of BAL fluid prior to donation resulted in a pure growth of methicillin-sensitive *Staphylococcus aureus*, but following the diagnosis of patient 1, this specimen was retrieved (3 weeks later) and *M. hominis* was identified by specific culture and molecular techniques (Table 2). Attempts to isolate *M. hominis* from specimens from the donors for patient 2 and 3 were not possible as specimens were not available.

Clinical and Epidemiologic Review

All 3 patients were nursed in adjacent cubicles within the ICU (Supplementary Figure 1). A review of nursing and medical staff did not identify any unwell staff members. Staff screening was not undertaken. The government-run Organ and Tissue Authority was contacted and advised regarding the cases and asked to contact centers with recipients of organs from the 3 donors for patients 1–3. There were no notifications of *M. hominis* infection in these recipients reported.

Whole-Genome Sequencing and Phylogenomics

The phylogenetic tree inferred from the recombination-free polymorphic sites is presented in Figure 1. Isolates from donor 1 and patient 1 were monophyletic with 100% bootstrap support and <20 SNP differences between them in an alignment of 26 464 (SNP distances not shown). In contrast, patients 2 and 3 were nonmonophyletic with each other and with the donor and patient 1 isolates. These 2 isolates were separated from each other and all other isolates by >8600 SNPs. (The NCBI Sequence identification numbers are given in Supplementary Table A.) Calculated from a symmetrical pairwise distance matrix, the mean SNP distance among the isolates in this study was 8872.724 (median, 9171; min-max, 13–10 191; standard deviation, 1600.443; standard error of the mean, 156.1872), again emphasizing the relatedness of donor 1 and patient 1 isolates.

The *tetM* gene was identified in the genome of *M. hominis* from donor 1 and patient 1 only, adding a further point of difference to separate these isolates from the others. Inspection of the mapped pair-end reads, from both donor and recipient, showed a single nucleotide deletion in a poly-A sequence toward the 3′ end of *tetM* (nucleotide position 1584 in *tetM*5 reference sequence, NCBI accession U58985.1) leading to a frameshift

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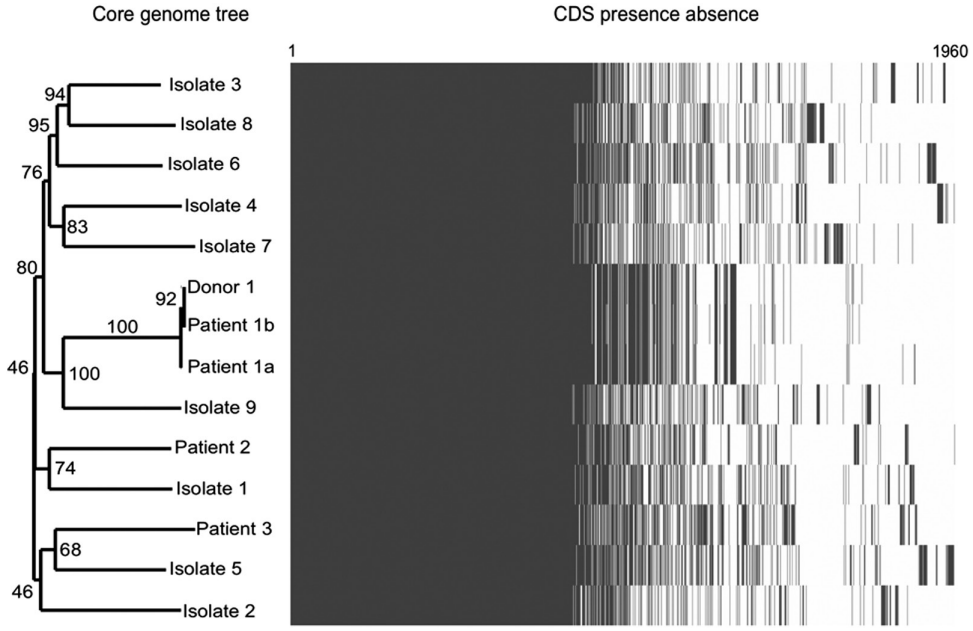

Figure 1. Maximum likelihood phylogeny of *Mycoplasma hominis* isolates (left, inferred from recombination filtered whole-genome alignment), with percentage bootstrap support overlaid on the branches, plotted next to a gene presence (gray)–absence (white) pangenome matrix (right). Columns in the pangenome matrix indicate unique coding DNA sequences (CDS), with 1960 unique CDS. *M. hominis* isolated from: donor 1, endotracheal aspirate; patient 1a, elbow tissue; patient 1b, blood; patient 2, bronchoalveolar lavage fluid; patient 3, hip aspirate; isolates 1–9, unrelated historical isolates detailed in the Supplementary Table.

mutation and premature termination of the coding sequence likely rendering the gene nonfunctional. Unfortunately the functionality of the *tetM* gene was unable to be verified as phenotypic antimicrobial susceptibility testing was unavailable.

DISCUSSION

To our knowledge, this is the first reported case of a donor-derived *M. hominis* infection confirmed using WGS. More importantly, this investigation demonstrates the utility of WGS to assist in the investigation of a cluster of unusual infections that were initially presumed to represent nosocomial transmission. The origins of *M. hominis* infection in patients 2 and 3 remain unknown, but potential sources include transposition of endogenous flora or acquisition from respective lung donors.

While established as a cause of infection after SOT, donor-derived infections with *M. hominis* have only been suggested in prior reports, without any proven cases. Similarly, while clusters of cases in single institutions are described, neither direct contamination nor nosocomial transmission has been demonstrated.

Gass et al provided the first record suggesting *M. hominis* donor-derived infection, detailing 2 cases of invasive *M. hominis* pleuropulmonary infection in the early posttransplant period in 2 recipients, each of a single lung, from an otherwise well 20-year-old man who died from a traumatic brain injury [15]. Both cases cultivated *M. hominis* on selective culture media from pleural fluid after routine examination and culture had failed to identify an organism. Routine peritransplant culture from the donor bronchial specimens did not isolate *M. hominis*. Without ready access to molecular analysis, clonality was not demonstrated. In contrast, in our cluster of cases, WGS provided evidence that patient 1 was infected with the same *M. hominis* isolate as that found in donor 1 and, importantly, that patients 2 and 3 were found to have distinct isolates from patient 1 and from each other.

Similarly, there has only been 1 other reported cluster of *M. hominis* infection in SOT recipients [16]. Four cases of disseminated infection occurred in an Australian tertiary hospital, in 3 cardiac and 1 bilateral lung transplant recipients nursed in a single ward over a 3-week period. All developed sternal wound infections, with and without complications of sternal osteomyelitis, mediastinitis, and pleuropericarditis. Donor microbiology was not reported. While a number of possible routes of infection were considered, based on the epidemiology, the authors felt that person-to-person transmission was most likely. Unfortunately the isolates were unable to be analyzed further for clonality and, therefore, questions regarding the source(s) of infection in this cluster of cases have remained unanswered. Our series of *M. hominis* cases were similarly clustered in a single ICU without a common source identified; however, using WGS, we were able to exclude patient-to-patient transmission and halt an unnecessary investigation into a possible outbreak in the ICU.

The exclusion of nosocomial transmission between our transplant recipients raises the question of the source of infection for patients 2 and 3. The most widely considered source of disseminated *M. hominis* is a translocation of endogenous

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flora in the setting of instrumentation and immunosuppression [6, 9, 17]. Neither patient 2 nor 3 had a history of *M. hominis* in prior specimens, but an understanding of the prevalence of genital and respiratory tract colonization makes this plausible. Also possible is a common environmental exposure. Whether the inanimate environment might serve as an unrecognized reservoir of *M. hominis* is yet to be demonstrated. *M. hominis* lacks a cell wall, rendering it osmotically fragile and vulnerable to environmental exposures [1]. Furthermore, due to a small genome, this organism has a limited biosynthetic capacity, and prolonged growth outside of a host is not readily supported [1]. Though *M. hominis* has been cultured from inside the rim of public toilet bowls, there is otherwise limited evidence to support it as a source of environmental outbreaks [18].

Our study highlights the invasive potential of *M. hominis* in SOT recipients and raises important questions regarding the identification of this potentially pathogenic organism. Successful laboratory isolation of *M. hominis* requires targeted culture methods as the organism does not stain by Gram stain, is slow to grow (often >72 hours), and produces small and translucent colonies, easily overlooked particularly in the presence of competing bacterial flora [6, 19]. In addition, *M. hominis* produces little turbidity in liquid media and will not trigger automated blood culture systems [2]. Based on our findings, we recommend laboratories decrease the threshold for using specialized techniques to isolate *M. hominis* particularly in high-risk, immunosuppressed cohorts where there is a purulent infection with a negative Gram stain.

Antimicrobial therapy directed at *M. hominis* is recommended for invasive and disseminated infections in immunocompromised hosts and the optimal duration of therapy must be individualized, with recommendations ranging between 14 days and 6 weeks [7]. Surgical debridement of devitalized and infected tissue and drainage of collections is an important adjuvant to cure [7]. The Clinical and Laboratory Standards Institute has published a consensus on the methods for antimicrobial susceptibility testing for human mycoplasmas with defined minimum inhibitory concentration ranges and interpretive breakpoints, but there are currently no European Committee on Antimicrobial Susceptibility Testing breakpoints for *M. hominis*. Effective *M. hominis* antimicrobial therapy includes tetracyclines, lincosamides, and fluoroquinolones, none of which are commonly used as peritransplant prophylaxis [2]. As described above, 2 isolates (from patient 1 and her donor) were found to have the *tetM* gene, which encodes a tetracycline resistance protein, conferring resistance to tetracyclines; however, patient 1 was successfully treated with an antimicrobial combination that heavily relied on doxycycline. WGS explained this apparent paradox by revealing that the *tetM* gene had a frameshift mutation that was predicted to prevent the expression of tetracycline resistance, thereby allowing doxycycline to be therapeutically effective.

In summary, *M. hominis* should be recognized as an uncommon, though perhaps underrecognized, source of donor-derived infection. In light of limitations of the routine staining and culture techniques, we recommend that all specimens from sterile sites, especially in the immunocompromised cohort, with negative Gram stains but with high polymorph counts, be considered for further investigation for more fastidious organisms, including specialized culture and molecular techniques for *M. hominis*.

Supplementary Data

Supplementary materials are available at *Clinical Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

Note

Potential conflicts of interest. All authors: No reported conflicts of interest. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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