cases reported to health authorities. The chronic form of paracoccidioidomycosis will probably develop in some of these patients.

This study underscores the need for paracoccidioidomycosis surveillance, especially in the context of environmental alterations enhanced by climate change and affected by construction, deforestation, and other human interventions. Enhanced surveillance will more fully identify relative risks of different human enterprises and facilitate interventions for at-risk populations to reduce and prevent future outbreaks of paracoccidioidomycosis.

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References
1. Coutinho ZF, Silva D, Lazera M, Petri V, Oliveira RM, Sabroza PC, et al. Paracoccidioidomycosis mortality in Brazil (1980–1995). Cad Saude Publica. 2002;18:1441–54. http://dx.doi.org/10.1590/S0102-311X2002000500037
2. Prado M, Silva MB, Laurenti R, Travassos LR, Taborda CP. Mortality due to systemic mycoses as a primary cause of death or in association with AIDS in Brazil: a review from 1996 to 2006. Mem Inst Oswaldo Cruz. 2009;104:513–21. http://dx.doi.org/10.1590/S0074-02762009000300019
3. Shikanai-Yasuda MA, Mendes RP, Colombo AL, Moretti ML, Queiroz-Telles F, Kono AS, et al. Brazilian guidelines for the clinical management of paracoccidioidomycosis. Rev Soc Bras Med Trop. 2017;July 20: [Epub ahead of print]. http://dx.doi.org/10.1590/0037-8682-0230-2017
4. Franco M, Bagagli E, Scapolio S, da Silva Lacaz C. A critical analysis of isolation of Paracoccidioides brasiliensis from soil. Med Mycol. 2000;38:185–91. http://dx.doi.org/10.1080/ mmy.38.3.185.191
5. de Macedo PM, Almeida-Paes R, Freitas DF, Varon AG, Paixão AG, Romão AR, et al. Acute juvenile Paracoccidioidomycosis: a 9-year cohort study in the endemic area of Rio de Janeiro, Brazil. PLoS Negl Trop Dis. 2017;11:e0005500. http://dx.doi.org/10.1371/ journal.pntd.0005500
6. do Valle AC, Coimbra Júnior CE, Linares FI, Monteiro PC, Guimarães MR. Paracoccidioidomycosis among the Indian group Suruí of Rondonia, Amazonia, Brazil. A case report [in Portuguese]. Rev Inst Med Trop Sao Paulo. 1991;33:407–11. http://dx.doi.org/10.1590/S0036-46651991000500012
7. Coimbra Júnior CE, Wanke B, Santos RV, do Valle AC, Costa RL, Zancopé-Oliveira RM. Paracoccidioidin and histoplasmin sensitivity in Tupi-Mondé Amerindian populations from Brazilian Amazonia. Ann Trop Med Parasitol. 1994;88:197–207. http://dx.doi.org/10.1080/00034983.1994.11812858
8. Barrozo LV, Benard G, Silva ME, Bagagli E, Marques SA, Mendes RP. First description of a cluster of acute/subacute paracoccidioidomycosis cases and its association with a climatic anomaly. PLoS Negl Trop Dis. 2010;4:e463. http://dx.doi.org/10.1371/journal.pntd.0000643
9. Brazilian Institute of Geography and Statistics. Cities, September 12, 2016 [in Portuguese] [cited 2017 June 29]. http://cidades.ibge.gov.br/xtras/perfil.php?codmun=330170
10. Press Rio de Janeiro News. Arco Metropolitan discovers new archaeological sites, Rio de Janeiro, Brazil, April 21, 2012 [in Portuguese] [cited 2017 May 29]. http://www.rj.gov.br/web/ imprensa/exibeconteudo?article-id=869952

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Mycobacterium shimoidei, a Rare Pulmonary Pathogen, Queensland, Australia

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Nontuberculous mycobacteria are human pathogens with increasing incidence and prevalence worldwide. Mycobacterium shimoidei is a rare cause of pulmonary disease, with only 15 cases previously reported. This series documents an additional 23 cases of M. shimoidei from Queensland, Australia, and highlights the pathogenicity and clinical role of this species.
In Queensland, Australia, NTM is a reportable condition, requiring all isolates to be reported to the Queensland Mycobacterium Reference Laboratory. This series examines all *M. shimoidei* cases in Queensland during January 1, 2000–December 31, 2014.

We extracted data from the Queensland Notifiable Condition System with ethics approval obtained from the Metro North Human Research Ethics Committee (HREC/15/QPCH/65). Confirmatory testing was conducted at the Queensland Mycobacterium Reference Laboratory using 2 methods: the Hain Genotype CM/AS Line Probe Assays (Hain Lifescience, Nehren, Germany) and 16S rRNA sequencing.

We obtained clinical information from treating physicians and patient medical records. We recorded each isolate as being likely clinically significant, possibly significant, or unlikely significant, and as being consistent or not with NTM lung disease according to the 2007 American Thoracic Society and Infectious Disease Society of America criteria.

Specimens from 23 patients (35 total isolates) cultured *M. shimoidei* in Queensland during the study period. Individual clinical characteristics, treatment, and outcomes can be seen in the Table. Previously reported cases are summarized in the online Technical Appendix (https://wwwnc.cdc.gov/EID/article/23/11/17-0999-Techapp1.pdf).

Sixteen (69.6%) patients were male (mean ± SD age 66.2 ± 12.6 years), consistent with previous case reports (3–10). Nine (39.1%) were classified as being likely clinically significant and 7 (30.4%) possibly significant. Ten patients (43.5%) met the 2007 American Thoracic Society and Infectious Disease Society of America criteria for having NTM lung disease. All isolates were cultured from respiratory specimens, with 15 (65.2%) isolated from sputum; 2 (8.7%) from bronchial washings; 2 (8.7%) from both bronchial washings and sputum; and 2 (17.4%) from lung tissue either with computed tomography–guided biopsy or at autopsy. Only 4 (17.4%) specimens were smear-positive by microscopy.

### Table. Clinical characteristics, treatment, and outcomes of *Mycobacterium shimoidei* isolates, Queensland, Australia*

| Specimen (isolates) | Age, y/sex | Significant | Signs/ symptoms | Radiology | Concurrent conditions | Management (time) | Outcome |
|---------------------|------------|-------------|-----------------|-----------|-----------------------|------------------|---------|
| Sp and Br (×4)      | 60/M       | Likely      | C, Sp, WL       | Cavities, nodule | COPD, asthma          | Observed        | Stable  |
| LTis (×1)           | 56/M       | Likely      | Died            | Unknown     | COPD, HF, AF, GED     | None            | Died    |
| Sp (×1)             | 75/F       | Likely      | C, Sp, WL       | Cavities, nodule | COPD, bronchiectasis, IHD | None            | Died of other cause |
| LTis (×1)           | 72/M       | Likely      | C, D, WL        | Cavity      | COPD, bronchiectasis, IHD | Observed        | Died of lung disease |
| LTis (×1)           | 62/F       | Likely      | C, WL, NS       | Cavity      | COPD, aspergillus, HTN | INH, RFP, PZA, EMB (6 mo) | Stable |
| Sp (×2)             | 68/M       | Likely      | C, Sp, H, WL, Fa| Cavities, consolidation | Lung cancer, COPD, bronchiectasis | CLA, MFX, SMX (12 mo) | Improved |
| LTis (×1)           | 70/M       | Likely      | C, Sp, CP       | Cavities    | COPD, bronchiectasis, GED | CLA, RIF, EMB (12 mo) | Died of lung disease |
| Sp (×3)             | 77/F       | Likely      | C, WL, Fa       | Cavity, nodule | COPD, RA, anemia       | Observed        | Stable  |
| Br (×1)             | 76/M       | Possibly    | D, WL           | Mass, effusion | COPD, anemia           | None            | Unknown |
| Br (×1)             | 84/M       | Possibly    | C, Sp           | Nodules     | Lung cancer, GED       | Observed        | Unknown |
| Sp (×1)             | 84/M       | Possibly    | C, D, Fa        | Consolidation | COPD, bronchiectasis, CF | Observed     | Improved |
| Sp (×1)             | 29/M       | Possibly    | C, D, WL        | Nodules     | COPD, bronchiectasis, CF | Observed     | Improved |
| Sp (×1)             | 74/F       | Possibly    | C, Sp           | Nodules, consolidation | Bronchiectasis | Observed        | Improved |
| Sp (×5)             | 84/F       | Possibly    | C, Sp, H, WL    | Nodules     | Bronchiectasis, type 2 diabetes, HTN | CLA (2 mo) | Improved |
| Sp (×1)             | 58/M       | Possibly    | C, Sp           | Normal      | Obesity, HTN           | Observed        | Stable  |
| Sp (×1)             | 57/M       | Unlikely    | Unknown         | Unknown     | Unknown                | Unknown         | Unknown |
| LTis (×1)           | 55/F       | Unlikely    | Unknown         | Unknown     | Unknown                | Unknown         | Unknown |
| Sp (×1)             | 67/M       | Unlikely    | Unknown         | Unknown     | Unknown                | Unknown         | Unknown |
| Sp (×1)             | 60/M       | Unlikely    | C, D            | Normal      | Asthma                 | None            | Unknown |
| Sp (×1)             | 59/F       | Unlikely    | C               | Normal      | Asthma, GED            | None            | Unknown |
| Sp (×1)             | 73/M       | Unlikely    | Unknown         | Unknown     | Unknown                | Unknown         | Unknown |
| Sp (×1)             | 54/M       | Unlikely    | Unknown         | Unknown     | Unknown                | Unknown         | Unknown |

*AF, atrial fibrillation; AMK, amikacin; AZA, azithromycin; Br, bronchoscopic washing; C, cough; CF, cystic fibrosis; CFX, cefoxitin; CFZ, cefazidime; CLA, clarithromycin; COPD, chronic obstructive pulmonary disease; CP, chest pain; D, dyspnea; EMB, ethambutol; Fa, fatigue, GED, gastrolesophageal reflux disease; HTN, hypertension; IHD, ischemic heart disease; INH, isoniazid; LTis, lung tissue; MFX, moxifloxacin; NS, night sweats; PZA, pyrazinamide; RA, rheumatoid arthritis; RFP, rifampin; RIF, rifabutin; SMX, sulfamethoxazole, Sp, sputum; WL, weight loss.*
The most common symptoms were cough or sputum (16; 69.6%); weight loss (9; 39.1%); dyspnea (5; 21.7%); fevers or sweats (4; 17.4%); and fatigue (2; 8.7%). Cough and sputum predominated in previous cases, but not weight loss (3–10). Radiology demonstrated cavitary disease in 9 patients (39.1%). Similar to our cohort, 9 of the 15 previously reported cases had cavities, highlighting a potentially distinguishing feature of M. shimoidei lung disease (3–7).

The most common associated concurrent conditions were obstructive airway disease (10; 43.5%), bronchiectasis (6; 26.1%), gastroesophageal reflux disease (4; 17.4%), and malnutrition (3; 13.0%). Underlying chronic lung disease was also present in previously reported cases and included chronic obstructive pulmonary disease, past tuberculosis, pneumocooniosis, and bronchiectasis (3–10).

Although 16 patients (69.5%) were deemed to have either likely or possibly clinically significant disease, only 6 (26.0%) underwent medical treatment, with 7 (30.4%) being actively observed. These low treatment numbers may reflect a lack of knowledge in relation to M. shimoidei; however, they may also be an indirect result of the underlying comorbidities and poor functional status of infected patients.

When medical treatment was offered, however, 5 of the 6 patients improved or had stable disease, with the sixth patient dying of lung cancer while undergoing antimicrobial therapy. Of the 7 patients who were observed, 3 remained stable, 2 improved, and 2 died of either chronic lung disease or progression of their M. shimoidei infection. In comparison, 6 of the 15 previous cases in the literature improved with medical treatment, with 4 dying during treatment and 1 remaining stable with observation alone (3–10). Although this relatively high death rate may reflect the nature of the patients’ comorbidities, it still highlights the clinical significance of M. shimoidei if isolated.

Although none of the Queensland cohort underwent drug susceptibility testing, review of previous cases suggests that a combination of rifabutin, ethambutol, and clarithromycin may be an effective drug regimen, with moxifloxacin/levofloxacin, sulfamethoxazole, pyrazinamide, and linezolid as other potential agents (3–7).

Our study has several limitations. First, it is a retrospective case series with data extracted from a passive surveillance system. Even though all laboratory-confirmed cases were captured, it is possible that not all patients with M. shimoidei infection received this diagnosis or were able to provide an appropriate specimen for identification. Furthermore, due to both the clinical characteristics being reported by various treating physicians and a large proportion not having complete clinical or follow-up data available, we may have captured inaccurate or inconsistent data.

This case series highlights the clinical significance and pathogenicity of M. shimoidei. Cases have been isolated only from respiratory specimens, occur predominantly in male patients with underlying chronic lung disease, and commonly present with cavitary disease. Although illness and death are associated with M. shimoidei infection, a reasonable outcome can be achieved with treatment. Possible drug regimens involve a combination of rifabutin, ethambutol, and clarithromycin, with moxifloxacin/levofloxacin, sulfamethoxazole, pyrazinamide, and clofazimine also potentially being useful. Increased recognition and understanding of this pathogenic organism are necessary to improve patient outcomes.

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References

1. Thomson RM; NTM working group at Queensland TB Control Centre and Queensland Mycobacterial Reference Laboratory. Changing epidemiology of pulmonary nontuberculous mycobacteria infections. Emerg Infect Dis. 2010;16:1576–83. http://dx.doi.org/10.3201/eid1610.091201
2. Tsukamura M, Shimoide H, Saefer WB. A possible new pathogen of group III Mycobacteria. J Gen Microbiol. 1975:88:377–80. http://dx.doi.org/10.1099/00221287-88-2-377
3. Takayama S, Tominaga S, Tuskada Y, Ohkochi M, Inase N. A case of pulmonary Microbacterium shimoidei infection [in Japanese]. Kekakaku. 2006;81:537–41.
4. Kamaji N, Kushida Y, Bandoh S, Ishii T, Haba R, Tadokoro A, et al. Membranous glomerulonephritis associated with Mycobacterium shimoidei pulmonary infection. Am J Case Rep. 2013;14:543–7. http://dx.doi.org/10.12659/AJCR.889684
5. Galizzi N, Tortoli E, Gori A, Morini F, Lapadula G. A case of mild pulmonary disease due to Mycobacterium shimoidei with a favorable outcome. J Clin Microbiol. 2013;51:3467–8. http://dx.doi.org/10.1128/JCM.01028-13
6. Tortoli E, Simonetti MT. Isolation of Mycobacterium shimoidei from a patient with cavitary pulmonary disease. J Clin Microbiol. 1991;29:1754–6.
7. Mayall B, Gurtler V, Irving L, Marzec A, Leslie D. Identification of Mycobacterium shimoidei by molecular techniques: case report and summary of the literature. Int J Tuberc Lung Dis. 1999;3:169–73. http://www.ncbi.nlm.nih.gov/pubmed/10091886.
8. Heller R, Jaulhac B, Charles P, De Briel D, Vincent V, Bohnier C, et al. Identification of Mycobacterium shimoidei in a tuberculosis-like cavity by 16S rRNA sequence analysis. J Eukaryot Microbiol. 1996;43:57–61. http://dx.doi.org/10.1002/1555-2179(199601)43:1<57::aid-emj2>3.0.co;2-2
9. Sundman K, Chryssanthou E, Petrini B. Microbacterium shimoidei, an easily misdiagnosed non-tuberculous pulmonary mycobacterium. Scand J Infect Dis. 2000;32:450–1. http://dx.doi.org/10.1080/00365400750045187
The Breadth of Viruses in Human Semen

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Zika virus RNA is frequently detected in the semen of men after Zika virus infection. To learn more about persistence of viruses in genital fluids, we searched PubMed for relevant articles. We found evidence that 27 viruses, across a broad range of virus families, can be found in human semen.

The finding by Atkinson et al. that Zika virus RNA is frequently detected in the semen of men after infection (1) highlights our knowledge gaps regarding the persistence of viruses in genital fluids, especially semen. Replicating Zika virus (2), like Ebola and Marburg viruses (3), has been isolated from semen and has been sexually transmitted. However, it is probable that many more viruses capable of causing viremia (presence of virus in the blood) can be found in semen. Seeding to the male reproductive tract may frequently occur in the context of viremia because the blood–testes/deferens/epididymis barriers are imperfect barriers to viruses, especially in the presence of systemic or local inflammation (4). Virus may persist even if incapable of replicating within the male reproductive tract because the testes are immunologically privileged (4); that is, within the testes, the immune response is restricted to enable the survival of sperm, which are immunogenic. Virus may also be transmitted to semen as a result of survival and replication within the accessory glands (5).

To investigate the breadth of viruses in semen, we performed a PubMed search by using the terms “virus* AND semen OR sperm* OR seminal.” We imposed no date or language restrictions. This search returned 3,818 results. We screened the titles, abstracts, and full text articles for data that described detection of viruses in semen by nucleic acid amplification or detection, antigen detection, replication in cell culture, or replication in an animal system. We restricted the results to viruses capable of causing viremia. Where we found evidence for virus in semen, we then searched PubMed for evidence of sexual transmission by using the terms “(name of virus) AND sex* AND Transm*.”

Our search revealed that 27 viruses that can result in viremia have been found in human semen (Table). For many of these, data on sexual transmission are lacking. Of these 27 viruses, many cause chronic or latent infection (e.g., HIV virus, cytomegalovirus). However, several cause acute infections, including Lassa fever, Rift Valley fever, and chikungunya viruses. Of those causing acute infections, only Zika and Ebola viruses have been systematically screened for in semen (i.e., in case series or cohort studies rather than case reports). These 27 viruses come from diverse families, suggesting that the presence of many viruses in semen is unlikely to be exclusively dependent on specific or conserved viral epitopes, ability of virus to replicate within the male reproductive tract, or common mechanisms of immune evasion. Other factors that may also influence whether viruses exist in semen are level of viremia, inflammatory mediators (altering blood–barrier permeability), systemic immunosuppression, male reproductive tract immune responses, presence of sexually transmitted diseases, and virus structural stability. In mammals, numerous viruses are detectable in semen, including viruses that can cause disease in humans, such as Japanese encephalitis virus, foot and mouth disease virus, parainfluenza virus, and paravaccinia virus (6). Several other viruses that result in viremia can cause orchitis and have been detected in human testes, suggesting the possibility that these viruses may also be detectable in semen. These viruses include influenza virus, lymphocytic choriomeningitis virus, phlebotomus fever virus, cocksackie B virus, echovirus, dengue virus, systemic acute respiratory syndrome virus, parvovirus, smallpox virus, vaccinia virus, and rubella virus (7).

Given these findings, the following questions need to be addressed: which viruses are shed and remain viable in semen, for how long, and at what concentrations? The answers to these questions have implications for risks for sexual transmission and, therefore, embryonic infection, congenital disease, miscarriage, and effects on epidemiologic and transmission models. The presence of virus in the male reproductive tract may increase the risk for acquisition of sexually transmitted infections and may reduce male fertility through spermatogonial stem cell infection or local inflammation. Infection of spermatozoa could result in transmission of virus-induced mutations to subsequent

10. Koukila-Kähkölä P, Paulin L, Brander E, Jantzen E, Eho-Remes M, Katila ML. Characterisation of a new isolate of Mycobacterium shimoidei from Finland. J Med Microbiol. 2000;49:937–40. http://dx.doi.org/10.1099/0022-1317-49-10-937

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### Mycobacterium shimoidei, a Rare Pulmonary Pathogen, Queensland, Australia

#### Technical Appendix Table. Clinical characteristics, treatment, and outcomes of previously reported *Mycobacterium shimoidei* isolates

| Author, year | Country | Age, y, sex | Symptoms | Radiology | Comorbidities | Management | Outcome |
|--------------|---------|-------------|----------|-----------|---------------|------------|---------|
| Tsukumura et al., 1975 (1) | Japan | 56/M | Unknown | Cavity | Unknown | INH | Died of lung disease |
| Rusch-Gerdes et al., 1985 (2) | Germany | 77/M | F | Cavity | Silicosis | INH, PRO, RFP, SM, ISO (3 mo) | Improved |
| Chromyc et al., 1989 (3) | Canada | 84/M | C, S | Consolidation | COPD, pneumoconiosis | Unknown | Died of lung disease |
| Tortoli and Simonetti, 1989 (4) | Italy | 68/M | U | Cavity | Old TB, Addison's | INH, SM, EB, RFP, KM, PAS (4 mo) | Unknown |
| Miller et al., 1991 (5) | Canada | 65/M | F | Mass | COPD, lung Ca | Unknown | Died of lung disease |
| Furrer et al., 1994 (6) | Switzerland | 34/M | U | Unknown | HIV | None | Died of other cause |
| Heller et al., 1996 (7) | France | 48/F | A | Cavity, nodules | Old TB | SM, EMB, CLA, RIF, EM, CPFX, RIF (18 mo) | Improved |
| Auregan et al., 1997 (8) | Madagascar | 43/F | H | Cavity | Old TB | INH, RFP, PZA, EMB, SM (17 mo) | Died of lung disease |
| Goudge et al., 1998 (9) | Australia | 75/M | C, S, WL, NS | Cavity, nodules | COPD, previous lobectomy | EMB, PZA, CLA, RIF (5 wk) | Died of lung disease |
| Mayall et al., 1999 (10) | Australia | 53/F | C, S, WL | Cavity | COPD, fibrosis, esophageal Ca | INH, RFP, PZA, EMB (10 d) | Died of lung disease |
| Koukila-Kahkola et al., 2000 (11) | Finland | 78/F | C, S | Normal | None | Observation | Stable |
| Sundman et al., 2000 (12) | Sweden | 59/F | F, CP, D | Cavity | COPD | CPFX, CLDM; MDZ, TFX (6 wk) | Improved |
| Takayama et al., 2005 (13) | Japan | 68/M | F, C | Consolidation | COPD | RFP, EMB, CAM, PZA, CPFX (6 mo) | Improved |
| Kanaji et al., 2011 (14) | Japan | 83/M | C, S | Cavity | COPD | CLA, RFP, EMB (18 mo) | Improved |
| Galizzi et al., 2013 (15) | Italy | 53/F | C, H | Nodules | Bronchiectasis | AMK, RIF, EMB, CLA (18 mo) | Improved |

* A, asymptomatic; AMK, amikacin; C, cough; CLA, clarithromycin; CLDM, clindamycin; COPD, chronic obstructive pulmonary disease; CPFX, ciprofloxacin; D, dyspnea; EMB, ethambutol; F, fever; H, hemoptysis; INH, isoniazid; KM, kanamycin; MDZ, metronidazole; NS, night sweats; PAS, para-aminosalicylic acid; PRO, prothionamide; PZA, pyrazinamide; RFP, rifampin; RIF, rifabutin; SM, streptomycin; Sp, sputum; TB, tuberculosis; TX, trifluridin; U, unknown; WL, weight loss
References

1. Tsukamura M, Shimoide H, Shaefer WB. A possible new pathogen of group III Mycobacteria. J Gen Microbiol. 1975;88:377–80. http://dx.doi.org/10.1099/00221287-88-2-377

2. Rusch-Gerdes S, Wandelt-Freerksen E, Schroder K-H. Vorkommen von Mycobacterium shimoidei in der Bundes-republik Deutschland. Zentralb Bakteriol Hyg Orig Reihe A. 1985;259:146–50.

3. Chomyc SA, Pearson JH, Helbecque D. Mycobacterium shimoidei—Alberta. Can Dis Wkly Rep. 1990;17:85–6.

4. Tortoli E, Simonetti MT. Isolation of Mycobacterium shimoidei from a patient with cavitary pulmonary disease. J Clin Microbiol. 1991;29:1754–6.

5. Miller MA, Eymard D, Thibert L. Mycobacterium shimoidei: first reported isolate in Canada. Can Dis Wkly Rep. 1991;17:11–2.

6. Furrer H, Bodmer T, von Overbeck J. Disseminated nontuberculous mycobacteriosis in AIDS patients. Schweiz Med Wochenschr. 1994;124:96–8.

7. Heller R, Jaulhac B, Charles P, De Briel D, Vincent V, Bohner C, et al. Identification of Mycobacterium shimoidei in a tuberculosis-like cavity by 16S ribosomal DNA direct sequencing. Eur J Clin Microbiol Infect Dis. 1996;15:172–5. http://dx.doi.org/10.1007/BF01591494

8. Auregan G, Ramaroson F, Génin C, Vincent Lévy-Frébault V. Un cas d’infection pulmonaire à Mycobacterium shimoïdei à Madagascar. Bull Soc Pathol Exot. 1997;90:75–7.

9. Gough RJ, Mayall BC, Leslie DE, Holmes PW, Robinson SL. An Australian isolate of Mycobacterium shimoidei. Pathology. 1998;30:399–401. http://dx.doi.org/10.1080/00313029800169706

10. Mayall B, Gurtler V, Irving L, Marzec A, Leslie D. Identification of Mycobacterium shimoidei by molecular techniques: case report and summary of the literature. Int J Tuberc Lung Dis. 1999;3:169–73 http://www.ncbi.nlm.nih.gov/pubmed/10091886.

11. Koukila-Kähkölä P, Paulin L, Brander E, Jantzen E, Eho-Remes M, Katila ML. Characterisation of a new isolate of Mycobacterium shimoidei from Finland. J Med Microbiol. 2000;49:937–40. http://dx.doi.org/10.1099/0022-1317-49-10-937
12. Sundman K, Chryssanthou E, Petrini B. *Mycobacterium shimoidei*, an easily misdiagnosed non-tuberculous pulmonary mycobacterium. Scand J Infect Dis. 2000;32:450–1 http://www.ncbi.nlm.nih.gov/pubmed/17338311.

13. Takayama S, Tominaga S, Tsukada Y, Ohkochi M, Inase N. [A case of pulmonary *Mycobacterium shimoidei* infection]. Kekkaku. 2006;81:537–41.

14. Kanaji N, Kushida Y, Bandoh S, Ishii T, Haba R, Tadokoro A, et al. Membranous glomerulonephritis associated with *Mycobacterium shimoidei* pulmonary infection. Am J Case Rep. 2013;14:543–7. http://dx.doi.org/10.12659/AJCR.889684

15. Galizzi N, Tortoli E, Gori A, Morini F, Lapadula G. A case of mild pulmonary disease due to *Mycobacterium shimoidei* with a favorable outcome. J Clin Microbiol. 2013;51:3467–8. http://dx.doi.org/10.1128/JCM.01028-13