that the clinical isolate and the larvae from the Pacific cod were identified as *P. azarasi*, 1 of 2 species found in water near Japan. Adult worms live in the intestines of seals and sea lions, and infective larvae live in the tissues of various marine fish, including cod, pollack, and smelt (1).

In Japan, most patients infected with *Pseudoterranova* spp. have acute or subacute abdominal pain, and larvae are extracted from the stomach endoscopically. However, for some patients, diagnosis is made when 4th-stage larvae are expelled from the mouth, indicating that the larvae developed from the 3rd to 4th stage during the time of infection, as did the worm reported here. Expulsion of *Pseudoterranova* spp. larvae from the mouth in the absence of severe gastric symptoms occurs more commonly in Chile (5). Whether the varied symptoms triggered by infection with *Pseudoterranova* spp. larvae reflect different responses of individual hosts to the worms or whether the pathogenicity of *Pseudoterranova* spp. in humans differs among worm species remains to be elucidated.

Because of the increasing worldwide popularity of eating sushi and sashimi made of raw marine fish, consumers should be made aware of the possible risk for fish-borne parasitoses. Freezing and storing fish at −20°C for 7 days or freezing at −35°C until solid and storing at −35°C for 15 hours is sufficient to kill parasites (6).

This work was supported in part by grants-in-aids from the Ministry of Health, Labour and Welfare of Japan (H20-Shinko).

Naoki Arizono, Toshiyuki Miura, Minoru Yamada, Tatsuya Tegoshi, and Kotaro Onishi

Author affiliations: Kyoto Prefectural University of Medicine, Kyoto, Japan (N. Arizono, M. Yamada, T. Tegoshi, K. Onishi); and The University of Tokyo, Tokyo, Japan (T. Miura)

DOI: 10.3201/eid1703.101350

References

1. Mattiucci S, Nascetti G. Advances and trends in the molecular systematics of anisakid nematodes, with implications for their evolutionary ecology and host–parasite co-evolutionary processes. Adv Parasitol. 2008;66:47–148. DOI: 10.1016/S0065-308X(08)00202-9

2. Zhu XQ, D’Amelio S, Palm HW, Paggi L, George-Nascimento M, Gasser RB. SSCP-based identification of members within the *Pseudoterranova decipiens* complex (Nematoda: Ascaridoidea: Anisakidae) using genetic markers in the internal transcribed spacers of ribosomal DNA. Parasitology. 2002;124:615–23. DOI: 10.1017/S0031182002001579

3. Ishikura H. Anisakiasis. 2. Clinical pathology and epidemiology. Progress of Medical Parasitology in Japan. 2003;8:451–73.

4. Audicana MT, Kennedy MW. *Anisakis simplex*: from obscure infectious worm to inducer of immune hypersensitivity. Clin Microbiol Rev. 2008;21:360–79 DOI: 10.1128/CMR.00012-07.

5. Torres P, Jericic MI, Weitz JC, Dobrew EK, Mercado RA. Human pseudoterranovosis, an emerging infection in Chile. J Parasitol. 2007;93:440–3. DOI: 10.1645/GE-946R.1

6. US Food and Drug Administration. Parasites. In: Fish and fisheries products hazards and controls guidance, 3rd ed. Rockville (MD): The Administration; 2001. p. 65–72.

Address for correspondence: Naoki Arizono, Department of Medical Zoology, Kyoto Prefectural University of Medicine, Kawaramachi-Hirokoji, Kyoto 602-8566, Japan; email: arizonon@koto.kpu-m.ac.jp

Mycobacterium mageritense
Pulmonary Disease in Patient with Compromised Immune System

To the Editor: *Mycobacterium mageritense* is one of the rapidly growing mycobacteria (RGM). It was first isolated in Spain in 1987, described as a new species in 1997 by Domenech et al. (1), and first described and associated with disease in the United States in 2002 (2). In the 2002 report, 6 isolates were recovered from sputum, a bronchoscopy sample, a wound infection after liposuction, the blood of an immunosuppressed patient with a central catheter and sepsis, a patient with severe sinusitis, and from a wound infection in a patient who had probable osteomyelitis after fixation of an open fracture. It has since

![Phylogenetic analysis of members of *Pseudoterranova decipiens* species complex roundworms. Genetic relationships between NADH dehydrogenase subunit 1 sequences in clinical and Pacific cod isolates and species were inferred by using the neighbor-joining method. Bootstrap values (1,000 replicates) are shown next to the branches. The final dataset contained 498 positions. *P. decipiens* sensu stricto. Scale bar indicates nucleotide substitutions per site.](image-url)
been reported as a cause of water-related skin and soft tissue infections (3,4). A study from Japan in 2007 reported recovery of *M. mageritense* from the sputum of a woman with noncaseating granulomas by transbronchial biopsy who improved without therapy (5). We describe a case of *M. mageritense* pneumonia in an immunocompromised patient.

In 2009, a 54-year-old woman was admitted to the hospital in Austin, Texas, with a 5-day history of upper back pain and occasional hemoptysis and yellow sputum production. She had a long history of systemic lupus erythematosus and associated nephritis and vasculitis, rheumatoid arthritis, hypothyroidism, sleep apnea, and hepatitis C infection. She was taking prednisone 15 mg/day at the time of admission.

Five months earlier, organizing pneumonia was diagnosed in the patient by computed tomography-guided lung biopsy of a pleura-based mass; special stains and cultures on tissue for acid-fast bacilli (AFB), other bacteria, and fungi were negative. She was readmitted several times over subsequent months and treated with various antimicrobial agents and corticosteroids but did not show clinical or radiographic improvement. Chest computed tomographic scan performed at admission again demonstrated bilateral lung masses and infiltrates, with new areas of necrosis. A second needle biopsy sample showed chronic inflammation with a histiocytic reaction and negative stains for AFB and fungi, but it was deemed nondiagnostic. Subsequent open lung biopsy sample showed necrotizing granulomatous inflammation with possible vascular involvement suggestive of Wegener granulomatosis.

Fite staining showed rare clusters of AFB within the granulomas. The postoperative course was complicated by a multiloculated left pleural effusion. AFB smear of pleural fluid obtained from video-assisted thoracoscopy showed 1–5 bacilli per high power field. Cultures of lung tissue and pleural fluid grew mycobacteria initially identified as *M. fortuitum* group but subsequently identified as *M. mageritense* by PCR followed by restriction enzyme analysis of the 65-kDa heat-shock protein (*hsp65*) (6). Results of susceptibility testing by broth microdilution are shown in the Table.

Testing for Wegener granulomatosis by antineutrophil cytoplasmic and myeloperoxidase antibody yielded negative results. Imipenem and amikacin were prescribed, and gradual resolution of clinical signs and symptoms was observed. Oral linezolid and trimethoprim/sulfamethoxazole were prescribed at discharge. Chest radiographs taken 4 months after the open lung biopsy showed resolution of the masses.

The isolate was a nonpigmented RGM that matched the American Type Culture Collection (Manassas, VA, USA) type strain and 10 published clinical isolates of *M. mageritense* by PCR restriction enzyme analysis of the 65-kDa heat-shock protein (*hsp65*) (6). By gene sequencing of region V of the RNA polymerase (*rpoB*) gene, it exhibited 99.7% identity to the GenBank type strain sequence of *M. mageritense* (acceptable interspecies relatedness for this sequence is ≥98.5% identity) (8). The most closely related species determined by using this sequence and previously submitted sequences were other *M. fortuitum* species: *M. porcinum* (94% sequence identity), *M. wolinskyi* (94%), and *M. peregrinum* (93%).

Susceptibility testing of 23 clinical isolates of *M. mageritense* from the United States previously submitted to the Mycobacteria/Nocardia Research Laboratory (University of Texas Health Science Center, Tyler, TX, USA) and identified by *hsp65* PCR restriction analysis (6,7) was performed (Table). These results confirmed the potential utility of the drugs used in this case for future cases.

* M. mageritense has not been reported as a cause of pulmonary

---

### Table. In vitro activity of 23 isolates of *Mycobacterium mageritense*, United States, 2009

| Antimicrobial agent | No. isolates tested | MICs of current isolate, μg/mL | Intermediate breakpoint, μg/mL | MIC range, μg/mL | MIC<sub>50</sub>, μg/mL | MIC<sub>90</sub>, μg/mL | % S/I |
|---------------------|---------------------|-------------------------------|-------------------------------|-----------------|---------------------|---------------------|------|
| Amikacin            | 23                  | 8                             | 32                            | ≤1–32           | 16                  | 32                  | 100  |
| Cefoxitin           | 23                  | 16                            | 32–64                         | ≤25–256         | 32                  | 64                  | 91   |
| Ciprofloxacin       | 23                  | 0.25                          | 2                             | ≤0.25–0.5       | 0.25                | 0.5                 | 100  |
| Clarithromycin†     | 23                  | 8                             | 4                             | 1–64            | >32                 | >64                 | 4    |
| Doxycycline         | 22                  | 1                             | 2–8                           | 0.25–>64        | 8                   | >32                 | 50   |
| Imipenem            | 22                  | 4                             | 8                             | ≤0.5–8          | 2                   | 4                   | 100  |
| Linezolid           | 22                  | 4                             | 16                            | ≤2–16           | 4                   | 8                   | 100  |
| Sulfamethoxazole    | 21                  | 4                             | 32                            | ≤2–32           | 8                   | 32                  | 100  |
| Trimethoprim/sulfamethoxazole | 6   | 1/19                          | 2/38‡                         | ≤0.25/4.8–2/38  | 0.5/9.5             | 2/38                | 100  |
| Tobramycin          | 23                  | ≤2                            | 8                             | ≤2–64           | >16                 | >32                 | 30   |
| Tigecycline         | 5                   | 0.12                          | <§                            | ≤0.03–0.12      | 0.06                | 0.12                | NA   |

*Includes 6 isolates previously reported (2). S, susceptible; I, intermediate; NA, not available.
†Three days’ incubation.
‡Proposed breakpoint (7).
§No Clinical and Laboratory Standards Institute breakpoints established for tigecycline.
disease in an immunocompromised patient. However, most cases of M. fortuitum pneumonia were reported before the use of molecular technology for species identification. Newer species such as M. mageritense resemble M. fortuitum and would not have been differentiated without this method.

Our patient met the criteria for diagnosing nontuberculous mycobacterial lung disease as established by the American Thoracic Society and the Infectious Diseases Society of America (9). Her therapeutic response also supports a cause-and-effect relationship.

The identity of an RGM isolate as M. mageritense may be suspected by its unusual antimicrobial drug susceptibility pattern, which showed an intermediate MIC to amikacin and resistance to clarithromycin at 3 days (Table). However, definitive identification requires molecular methods. Previous studies have shown that M. mageritense contains an inducible erythromycin methylase gene (erm 40) that confers macrolide resistance (10). The use of molecular studies and greater attention to susceptibility patterns should enable increased recognition of M. mageritense as a human pathogen.

We thank Steven McNulty, Linda Bridge, and Ravikiran Vasireddy for laboratory assistance and Joanne Woodring for typing the manuscript.

R. Gordon Huth, Barbara A. Brown-Elliott, and Richard J. Wallace, Jr.

Author affiliations: University of Texas Southwestern Residency Programs, Austin, Texas, USA (R.G. Huth); and University of Texas Health Science Center, Tyler, Texas, USA (B.A. Brown-Elliott, R.J. Wallace, Jr.)

DOI: 10.3201/eid1703.101279

References

1. Domenec P, Jimenez MS, Menendez MC, Bull TJ, Samper S, Manrique A, et al. Mycobacterium mageritense sp. nov. Int J Syst Bacteriol. 1997;47:535–40. DOI: 10.1099/00207713-47-2-535
2. Wallace RJ, Brown-Elliott BA, Hall L, Roberts G, Wilson RW, Mann LB, et al. Clinical and laboratory features of Mycobacterium mageritense sp. nov. J Clin Microbiol. 2002;40:2930–5. DOI: 10.1128/JCM.40.8.2930-2935.2002
3. Appelgren P, Farnebo F, Dotevall L, Studahl M, Jonsson B, Petriani B. Late-onset posttraumatic skin and soft tissue infections caused by rapid-growing mycobacteria in tsunami survivors. Clin Infect Dis. 2008;47:e11–6. DOI: 10.1086/589300
4. Gira AK, Reisenauer AH, Hammock L, Nadiminti U, Macy JT, Reeves A, et al. Furunculosis due to Mycobacterium mageritense associated with foot baths at a nail salon. J Clin Microbiol. 2004;42:1813–7. DOI: 10.1128/JCM.42.4.1813-1817.2004
5. Miki M, Shimizukawa M, Okayama H, Kazumi Y. Case of pulmonary Mycobacterium mageritense infection: the difficulty of differential diagnosis of granulomatous lung disease. Kekkaku. 2007;82:189–94.
6. Steingrube VA, Gibson JL, Brown BA, Zhang Y, Wilson RW, Rajagopalan M, et al. PCR amplification and restriction endonuclease analysis of a 65-kiloDalton heat shock protein gene sequence for taxonomic separation of rapidly growing mycobacteria [ERRATUM 1995;33:1686]. J Clin Microbiol. 1995;33:149–53.
7. Woods GL, Brown-Elliott BA, Desmond EP, Hall GS, Heifets L, Pfyffer GE, et al. Susceptibility testing of mycobacteria, nocardia, and other aerobic actinomycetes; approved standard. NCCLS Document M24-A. Wayne (PA): Clinical and Laboratory Standards Institute; 2003.
8. Adékambi T, Colson P, Drancourt M. rpoB-based identification of nonpigmented and late pigmented rapidly growing mycobacteria. J Clin Microbiol. 2003;41:5699–708. DOI: 10.1128/JCM.41.12.5699-5708.2003
9. Griffith DE, Aksamit T, Brown-Elliott BA, Catanzaro A, Daley C, Gordin F, et al. An official ATS/IDSA statement: diagnosis, treatment and prevention of nontuberculous mycobacterial diseases. Am J Respir Crit Care Med. 2007;175:367–416. DOI: 10.1164/rccm.200604-571ST
10. Nash KA, Andini N, Zhang Y, Brown-Elliott BA, Wallace RJ Jr. Intrinsic macrolide resistance in rapidly growing mycobacteria. Antimicrob Agents Chemother. 2006;50:3476–8. DOI: 10.1128/AAC.00402-06

Address for correspondence: R. Gordon Huth, 601 E 15th St, Austin, TX 78701, USA; email: ghuth@seton.org

Extensively Drug-Resistant Tuberculosis, China

To the Editor: The prevalence of drug-resistant tuberculosis (TB) is a serious problem in the People’s Republic of China. China is 1 of 22 countries with the highest incidence of TB (1). It is also 1 of 27 countries with the highest incidence of multidrug-resistant TB (MDR TB) and extensively drug-resistant TB (XDR TB). According to the national baseline survey on TB in 2007 and 2008, the frequency of MDR TB among pulmonary TB patients in China was 8.3%. We estimate that there are 120,000 new cases of MDR TB in China per year, which accounts for 24.0% of new cases worldwide (510,000) per year.

XDR TB has recently emerged as a global public health problem (2). It is defined as TB with resistance to at least isoniazid, rifampin, a fluoroquinolone, and 1 of 3 injectable second-line drugs (amikacin, kanamycin, or capreomycin). XDR TB is a type of MDR TB that shows resistance to isoniazid and rifampin. Recent reports on current prevalence of XDR TB (3,4) indicate that China now has the second highest incidence of MDR TB worldwide. However, there is no information available on XDR TB in China.

To obtain information on XDR TB in China, we conducted a study