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Address for correspondence: Melissa Nolan, Arnold School of Public Health, University of South Carolina, 915 Greene St, Lab Rm 327, Columbia, SC 29208, USA; email: msnolan@mailbox.sc.edu

Osteomyelitis Caused by *Metamycoplasma gateae* after Cat Bite, Japan, 2026

Takehiro Hashimoto, Miyuki Uehara, Yuta Shingai, Daisuke Tamura, Kazunori Kimitsuki, Hirotaka Shibata, Kazufumi Hiramatsu

Author affiliations: Oita University Hospital, Yufu, Japan (T. Hashimoto, K. Hiramatsu); Oita University Faculty of Medicine, Yufu (M. Uehara, Y. Shingai, D. Tamura, K. Kimitsuki, H. Shibata)

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We report a case of osteomyelitis caused by *Metamycoplasma gateae* after a cat bite in an immunocompromised patient in Japan. No standardized treatment has been established; however, the patient recovered after surgery and minocycline treatment. Clinicians should consider *M. gateae* infection in immunocompromised patients with unidentified infections and cat-bite exposure.

Metamycoplasma gateae, a member of the family Mycoplasmataceae, was first isolated from cat saliva in 1967 (*1*) and reclassified from *Mycoplasma* into the genus *Metamycoplasma* in 2018 (*2*). Although *M. gateae* is known to colonize cats and cause feline infections (*3*–*6*), human infections have not been reported. We report a case of osteomyelitis caused by *M. gateae* in an immunocompromised patient with cat-bite exposure.

A 39-year-old woman was admitted to Oita University Hospital with fever and pain in her right hand, which her pet cat had bitten 42 days before admission. She had systemic lupus erythematosus treated with methylprednisolone (8 mg/d) and mycophenolate mofetil (2,000 mg/d) and type 1 diabetes mellitus managed with continuous subcutaneous insulin infusion. Swelling and pain in the right hand developed 32 days before admission and was diagnosed by a dermatologist at the hospital as cellulitis and treated with intravenous ceftriaxone, followed by oral clindamycin for 2 weeks. Magnetic resonance imaging, performed 31 days before hospital admission, had shown no osteomyelitis. The pain persisted despite treatment. Two days before admission, swelling spread to the wrist and fourth and fifth proximal interphalangeal joints, and fever (38.3°C) developed along with worsening pain.

At admission, physical examination revealed swelling and tenderness of the right hand. Laboratory tests showed elevated leukocytes (9,660 cells/μL, reference range 3,300–8,600 cells/μL), C-reactive protein (15.3 mg/dL, reference range 0.0–0.14 mg/dL), and glycated hemoglobin (7.6%, reference range 4.9%–6.0%) and markedly decreased serum immunoglobulin levels (88 mg/dL IgG [reference range 861–1,747 mg/dL]; 13 mg/dL IgA [reference range 93–393 mg/dL]; and 2 mg/dL IgM [reference range 50–269 mg/dL]). Magnetic resonance imaging revealed subcutaneous fluid collection within the dorsum of the hand and osteomyelitis of the third metacarpal bone, leading to a diagnosis of cellulitis with osteomyelitis. We performed an incision with drainage and collected pus, tissue, and blood culture specimens. We cultured the pus and tissue specimens in aerobic, anaerobic, and enrichment media under standard conditions (Appendix https://wwwnc.cdc.gov/EID/article/32/12/26-0515-App1.pdf). We initiated empirical antimicrobial drug therapy with piperacillin/tazobactam (4/0.5 g every 8 h). Pus and tissue cultures were negative; serologic testing for *Bartonella* antibodies was negative. We changed the antimicrobial drug therapy to ampicillin/sulbactam (2/1 g every 6 h) on day 5. However, pain in the right hand worsened; we changed the treatment back to piperacillin/tazobactam only on day 8.

On day 11, 16S rRNA gene sequencing of DNA from a tissue specimen identified *M. gateae* as the causative bacterium. The nucleotide sequence was deposited in GenBank (accession no. LC934417). The sequence showed 99.86% (1,450/1,452 nt) sequence

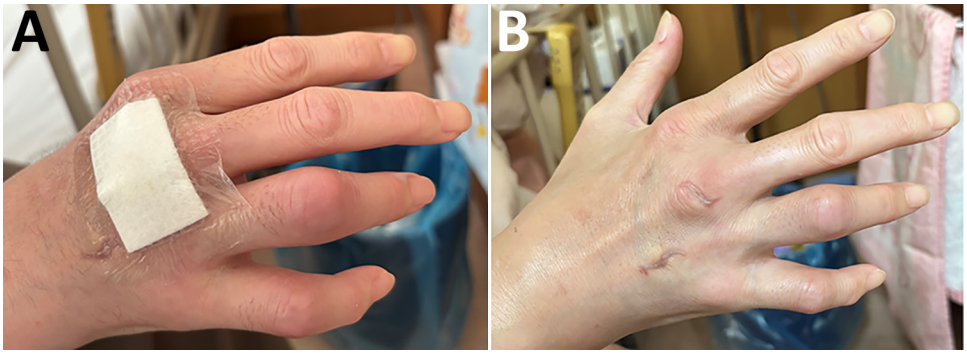


Figure. Patient's right hand in case study of osteomyelitis caused by *Metamycoplasma gateae* after cat bite, Japan, 2026. A) Erythema and swelling of the wrist, dorsum of the hand, and proximal interphalangeal joints of the fourth and fifth fingers on day 12 after hospital admission, immediately before initiation of minocycline therapy. B) Reduced erythema and swelling on day 20, eight days after initiation of minocycline therapy.

similarity to a reference strain (GenBank accession no. CP143578.1) (Appendix Table). Because the swelling persisted on day 12 (Figure, panel A), we switched treatment to minocycline (100 mg every 12 h). The swelling improved by day 19 (Figure, panel B) along with gradual recovery of the range of motion. We discharged the patient on day 29 and advised her to continue oral minocycline for ≥4 weeks.

M. gateae can cause severe infections in feline hosts, including erosive polyarthritis, tenosynovitis, ulcerative keratitis, and lower respiratory tract infections, particularly in immunocompromised cats (*3*–*6*). *M. gateae* infections have not been reported in humans. Because of the high prevalence of *M. gateae* in domestic cat populations, which is present in >80% of adult cats (*7*), cases of human infection might be unrecognized. No standardized antimicrobial drug treatment regimens have been established for *M. gateae* infections in humans. We selected minocycline because of its high level of bone penetration. The patient's immunocompromised state, caused by hypogammaglobulinemia, steroid therapy, and type 1 diabetes, might have contributed to infection susceptibility. Hypogammaglobulinemia is a known risk factor for *Mycoplasma* infections (*8*).

No pathogens were identified in tissue or blood cultures, and β-lactam/β-lactamase inhibitor therapy was ineffective. However, BLAST (https://blast.ncbi.nlm.nih.gov) analysis of the 16S rRNA gene sequence obtained from a tissue sample revealed the highest sequence identity to *M. gateae* (99.86% identity to GenBank accession nos. CP143578.1 and NR_029180.1). The next closest match was *Mycoplasma arginini* (99.31% sequence identity), a common colonizer of feline respiratory tracts (*8*). The differences in sequence identities support the identification of the isolate as *M. gateae*. We did not perform whole-genome sequencing, and misclassification cannot be excluded. Clinicians should consider *M. gateae* in immunocompromised patients with indolent infections after animal contact or bites. If *Mycoplasma* infection is suspected, specimens should be cultured on specialized media under appropriate conditions (*8*). Previous antimicrobial drug exposure might have reduced culture yield and masked other pathogens. Although incision and drainage might have contributed to recovery, we did not observe clinical improvement until minocycline was initiated, suggesting that targeted antimicrobial drug therapy played the primary role in clinical improvement.

In conclusion, we report a case of *M. gateae*–mediated osteomyelitis in an immunocompromised patient. Negative routine tissue and blood cultures, despite cat-bite exposure, together with a poor clinical response to β-lactam/β-lactamase inhibitor therapy prompted 16S rRNA gene sequencing, which identified *M. gateae*. *M. gateae* infection should be considered in patients with cat-bite exposure when routine cultures are negative and β-lactam/β-lactamase inhibitor therapy is ineffective.

The patient consented to publication of her case details and photographs of her right hand.

About the Author

Dr. Hashimoto is a medical doctor at the Infection Control Center, Oita University Hospital, Yufu, Japan. His research interests include microbiology and infectious diseases.

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Address for correspondence: Takehiro Hashimoto, Hospital Infection Control Center, Oita University Hospital, 1-1 Idaigaoka, Hasama-machi, Yufu, Oita 879-5593, Japan; email: hashimo2013@oita-u.ac.jp

Cross-Protection against Influenza A(H5) Clade 2.3.4.4b Viruses after A(H1N1)pdm09 Infection

Xiangjie Sun, Jessica A. Belser, Zhu-Nan Li, Nicole Brock, Joanna A. Pulit-Penaloza, Troy J. Kieran, Claudia Pappas, Hui Zeng, Jessie C. Chang, Paul J. Carney, Brandon L. Bradley-Ferrell, James Stevens, Min Z. Levine, Taronna R. Maines

Authors affiliation: Centers for Disease Control and Prevention, Atlanta, Georgia, USA

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We challenged ferrets previously infected with influenza A(H1N1)pdm09 virus with H5N1 or H5N5 clade 2.3.4.4b viruses to assess cross-protection. Results show that previous A(H1N1)pdm09 virus immunity conferred partial protection against both viruses, with slightly reduced protection against H5N5 compared with H5N1, potentially reflecting the absence of cross-reactive N5 neuraminidase antibodies.

Influenza A(H5) clade 2.3.4.4b viruses have spread widely among wild birds and poultry and caused infections in mammals, including humans, raising public health concerns (*1*). A case of human infection with H5N5 clade 2.3.4.4b virus was reported in Washington, USA during 2025; the patient subsequently died, representing the second fatal A(H5) case in the United States (*1*,*2*). A(H1) and A(H5) hemagglutinins (HAs) belong to influenza A virus phylogenetic group 1. Most antibodies targeting the HA stalk, a conserved, membrane-proximal region of HA, are cross-reactive with HAs within the same phylogenetic group (*3*). Studies have demonstrated that previous influenza A(H1N1)pdm09 virus infection can provide some protection against H5N1 clade 2.3.4.4b viruses (*4*,*5*). The emergence of H5N5 clade 2.3.4.4b virus raises questions about whether previous A(H1N1)pdm09 immunity confers similar protection against A(H5) viruses bearing antigenically distinct neuraminidases (NAs). We used a preimmune ferret model to compare cross-protection conferred by previous A(H1N1)pdm09 infection against A(H5) viruses with homologous or heterologous NA subtypes. We conducted all experiments with H5N5 and H5N1 viruses in a Biosafety Level 3 enhanced facility with approval from the Centers for Disease Control and Prevention Institutional Animal Care and Use Committee.

We intranasally inoculated 12 ferrets serologically naive to seasonal influenza viruses with 10^3 PFU of A(H1N1)pdm09 A/Nebraska/14/2019 virus. We confirmed seroconversion 3 months postinfection by using a hemagglutination inhibition (HAI) assay, which detects antibodies that inhibit HA-mediated hemagglutination, with a geometric mean titer of 320 (95% CI 245.3–417.3) (Appendix Figure, panel A; https://wwnc.cdc.gov/EID/article/32/12/26-1032-App1.pdf). Using previously described multiplex binding assays (*6*), we detected high levels of antibodies binding to the head of A(H1N1)pdm09, but not HA head or ectodomain from H5N1 or H5N5 viruses, and antibodies binding to N1 NAs from A(H1N1)pdm09 and H5N1 clade 2.3.4.4b viruses, but not N5 NA from H5N5 virus. In addition, we detected antibodies binding to the group 1 HA stalk derived