recommended breakpoint, we designed MHC1 with a colistin concentration of only 1 μg/mL to minimize false-negative results. However, some colistin-susceptible organisms might grow on MHC1 (<5% in our study), resulting in the low PCR-positive rate for mcr-1 among isolates.

Exact epidemiology of the mcr-1 gene is unknown, indicating a need to conduct accurate surveillance of the gene’s prevalence in humans. Additional mechanisms unique to the mcr-1 gene may contribute to colistin resistance, suggested by the wide variation in colistin MICs among mcr-1–carrying Enterobacteriaceae.

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Cryptococcus gattii Meningitis Complicated by Listeria monocytogenes Infection

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To the Editor: Among immunocompetent persons with cryptococcal disease, infection with a second organism is rare; all reported cases have involved concomitant mycobacterial infections (1). Immunosuppression is not a necessary precondition for infection with Cryptococcus gattii (2), and among immunocompetent persons, C. gattii infection confers high mortality rates: up to 24% according to a large case series (3). In addition, cryptococcomas are frequently observed in patients with C. gattii, as opposed to C. neoformans, infection, commonly necessitating longer courses of treatment. We report a fatal case of C. gattii and Listeria monocytogenes co-infection in an immunocompetent woman with cryptococcomas.

The patient was a previously healthy 23-year-old Hispanic woman who was hospitalized in 2009 after weeks of headache and recent-onset diplopia. Lumbar puncture revealed elevated opening pressure of 52 cm H₂O; elevated leukocytes (1,030 cells/μL: 31% neutrophils, 55% lymphocytes, 14% monocytes); elevated protein concentration (117 g/L); and decreased glucose concentration (30 mg/dL). Cerebrospinal fluid (CSF) cryptococcal antigen (CrAg) titer was 1:64, and culture grew C. gattii. HIV antibody test result was negative. Magnetic resonance imaging of the brain demonstrated scattered enhancing round lesions within the cerebral and cerebellum, consistent with cryptococcomas. The patient was prescribed intravenous amphotericin B (1 mg/kg/d) and intravenous fluocytosine (2 g/6 h) (Table); after 5 days of therapy, culture of a meningitis isolate showed C. gattii and C. neoformans. Lumbar puncture cultures confirmed C. gattii. The patient was then given intravenous liposomal amphotericin at 7 mg/kg, and after a 14-day induction period she was discharged with instructions to take fluconazole orally (400 mg 2×/d) and to continue amphotericin B infusions (3×/wk) (Table).
One week after hospital discharge, the patient experienced recurrent headache and low-grade fever and was readmitted. Repeat lumbar puncture indicated an opening pressure of 46 cm H$_2$O but improvement of all other clinical parameters (Table). CSF CrAg titer was 1:8 and culture result was negative. Repeat brain magnetic resonance images revealed no hydrocephalus, minimal edema, and decreased size and number of cryptococcomas. She was again given amphotericin B (5 mg/kg/d) and intravenous flucytosine (3 g/6 h) and fluconazole (600 mg/12 h). Placement of a ventricular-peritoneal shunt was deferred, and the patient experienced symptomatic improvement and was discharged from the hospital.

Two weeks later (11 weeks after initial admission), she returned to the hospital with worsening headache and fever. Lumbar puncture demonstrated a leukocyte count of 1,010 cells/μL (74% neutrophils, 12% lymphocytes, 14% monocytes), glucose 17 mg/dL, protein 258 g/L, and an opening pressure of >55 cm H$_2$O. CSF culture grew L. monocytogenes. The patient was prescribed ceftriaxone, ampicillin, and trimethoprim/sulfamethoxazole. Shortly after the lumbar puncture, she experienced status epilepticus and became comatose. Despite emergent craniotomy for relief of intracranial pressure, she remained comatose for several days; subsequently, supportive care was withdrawn and the patient died shortly thereafter.

This case highlights the difficulties of managing severe cryptococcal disease. This patient experienced headache over 3 months and symptom relapse during 10 weeks of antifungal therapy. As was done in this case, practice guidelines recommend a longer duration of polyene antifungal induction for patients with cryptococcomas than for those without (4), and longer courses of therapy are commonly described for infections caused by C. gattii than for those caused by C. neoformans (5). Corticosteroids are commonly used to treat immune reconstitution inflammatory syndrome associated with cryptococcal meningitis (6), although recently, they have been associated with adverse outcomes (7). As indicated by this case, corticosteroids remain a risk factor for secondary infection with several pathogens, including Listeria. No epidemiologic exposure to Listeria was identified for this patient.

C. gattii infection has been reported in 8 states, including California (3); we have found the pathogen in the soil south of Los Angeles, California, particularly in association with Canary Island pines and sweet gum trees (8). Some patients with C. gattii infection have autoantibodies to...
granulocyte–macrophage (GM) colony-stimulating factor (9). Although these autoantibodies have not been reported in patients with Listeria infections, susceptibility to infection caused by this bacterium is increased in GM–colony-stimulating factor –/– mice (10). Autoantibodies against GM–colony-stimulating factor or perhaps other cytokines might have impaired the patient’s host defense against these organisms; unfortunately, our report is limited by lack of serum for further testing.

This case demonstrates the difficulties of managing patients with C. gattii infection and an unusual co-infection with L. monocytogenes. Initiation of corticosteroids for the management of severe cryptococcal disease should be undertaken with caution. The differential diagnosis for worsening cryptococcal disease should include acute or subacute bacterial meningitis, particularly when the patient is receiving corticosteroids for the management of immune reconstitution inflammatory syndrome or associated complications.

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Melioidosis in Travelers Returning from Vietnam to France

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To the Editor: Melioidosis, a potentially fatal infectious disease, occurs predominantly across much of Asia and in northern Australia because of the soil and water bacterium Burkholderia pseudomallei (1). We report 2 related cases of suppurative cervical lymphadenitis, an unusual adult presentation of melioidosis, in 2 men who returned to France from Vietnam on the same trip (2).

Patient 1, a 28-year-old previously healthy man, was admitted to our hospital in Lyon, France, in October 2013 for the evaluation of a palpable neck mass, which had been growing steadily for the previous 2 months. Examination of the head and neck revealed a fluctuant, tender mass located in the inferior angle of the right side of the mandible, mimicking lymph node tuberculosis. Ultrasonographic investigation confirmed a level II enlarged cervical lymph node

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