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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 H_2O; 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 cerebrum and cerebellum, consistent with cryptococcomas. The patient was prescribed intravenous amphotericin B (1 mg/kg/d) and intravenous flucytosine (2 g/6 h) (Table); after 5 days of therapy, culture of a repeat lumbar puncture sample was negative. 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).

Table. Clinical events, management, and parameters for patient with *Cryptococcus gattii* meningitis complicated by *Listeria monocytogenes* infection*

Clinical event (day)	Therapy (days)	Opening pressure, cm H_2O (day)	Leukocyte count, cells/μL (day)	Protein, g/L (day)	Glucose, g/L (day)	CrAg titer (day)	Culture result (day)
Days 1–15: induction therapy	AMB 1 mg/kg (1–4); 5FC 2 g q6h (1–14); L-AMB 7/mg/kg (5–14)	52 (1), 12 (12)	1,030 (1), 123 (12)	117 (1), 104 (12)	30 (1), 29 (12)	1:64 (1)	*Cryptococcus gattii* (1), negative (12)
Days 16–30: discharge, outpatient infusion, readmission	L-AMB 7/mg/kg M,W,F (15–22); FLZ 400 mg q12h (15–22); L-AMB 5 mg/kg (23–30); FLZ 600 mg q12h: (23–30); 5FC 3 g q6h (23–30)	46 (23)	111 (23)	81 (23)	34 (23)	1:8 (23)	Negative (23)
Days 31–45: inpatient therapy	L-AMB 5 mg/kg (31–45); FLZ 600 mg q12h (31–45); 5FC 3 g q6h (31–45)	44 (38)	17 (38)	66 (38)	64 (38)	NA	Negative (38)
Days 46–60: inpatient therapy	L-AMB 5 mg/kg (46–60); FLZ 600 mg q12h (46–60); DEX 2 mg q6h (46–60)	35 (48)	18 (48)	25 (48)	85 (48)	NA	Negative (48)
Days 61–75: discharge and outpatient infusion	L-AMB 5 mg/kg (61–65); FLZ 600 mg q12h (61–75); DEX 2 mg q8h (61–75); L-AMB 7/mg/kg M,W,F (66–75)	13 (63)	8 (63)	28 (63)	91 (63)	1:4 (63)	Negative (63)
Days 76–83: readmission/coma (80); death (83)	L–AMB 7/mg/kg M,W,F (76–79); DEX 2 mg q12h (76–79); FLZ 600 mg q12h (76–83); CRO 2 gm q12h (80–83); AMP 2 gm q4h (80–83); TMP/SMX 320–1,600 mg (2 double-strength tablets) q8h (80–83)	>55 (80)	1,010 (80)	258 (80)	17 (80)	1:4 (80)	*Listeria monocytogenes* (80)

*5FC, flucytosine; AMB, amphotericin B; AMP, ampicillin; CrAg, cryptococcal antigen; CRO, ceftriaxone; DEX, dexamethasone; F, Friday; FLZ, fluconazole; L-AMB, liposomal amphotericin; M, Monday; NA, not available; q, every; TMP/SMX, trimethoprim/sulfamethoxazole; W, Wednesday.

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_2O but improvement of all other clinical parameters (Table). CSF CrAg titer was 1:8 and culture 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 required frequent lumbar punctures to relieve elevated intracranial pressure. After 3 weeks of therapy, she began taking oral dexamethasone (2 mg 4×/d) to reduce intracranial pressure and control symptoms consistent with immune reconstitution inflammatory syndrome. After 30 days of antifungal therapy during this second hospitalization, she experienced symptomatic improvement and was discharged with amphotericin B (5 mg/kg to be infused 3×/wk), fluconazole (600 mg 2×/d), and dexamethasone (tapering dosage).

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_2O. 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 anticryptococcal therapy. As was done in this case, practice guidelines recommend a longer duration of polyene antimycotic 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

[1]These authors contributed equally to this article.