with metronidazole (250 mg t.i.d.), prednisone (40 mg/d), and mesalamine (80 mg t.i.d.) was initiated, and within 2 weeks, both his GI and psychiatric symptoms dramatically abated. Although the mesalamine dosage was continued without change, metronidazole treatment was discontinued at 4 weeks, and the prednisone dosage was tapered to a maintenance dosage of 15 mg every other day over 3 months.

Subsequent deterioration in behavior was first noted 2 months after metronidazole treatment was discontinued (while he was still receiving mesalamine treatment and the maintenance dosage of prednisone). At admission to Rush Children’s Hospital (Chicago), he had only trace abdominal tenderness, heme-negative stool, and an ESR of 18 mm/h. Colonoscopy confirmed active lesions in the right colon consistent with Crohn’s disease. There was no evidence of luminal stricture or narrowing related to Crohn’s disease. Electroencephalograms obtained throughout a 24-h period were normal. Treatment with prednisone (60 mg/d) and mesalamine (800 mg t.i.d.) was initiated for his Crohn’s disease, with the prednisone dosage tapered to 20 mg/d after several months. Although his subtle GI symptoms resolved, his psychotic condition failed to improve, despite further treatment with antipsychotic agents, including lithium, pimozide, valproic acid, flufenazine, and clozapine.

Finally, 3 months after the psychiatric relapse, a second 1-month course of metronidazole (500 mg t.i.d. orally for 30 days) was initiated. Within 3 weeks, a dramatic improvement in his psychiatric condition was again noted, and by 5 weeks, he was essentially normal and not receiving any antipsychotic medication. Five months later, he remained free of psychiatric and GI symptoms, although repeated colonoscopy confirmed that his Crohn’s disease was quiescent.

The cause of the observed improvement in this patient’s psychiatric condition is unknown. Treatment with prednisone and mesalamine was continued throughout his course, and although these agents may have contributed to improvement in his condition, the behavioral changes were not noted on either occasion until the addition of metronidazole therapy. This finding suggests that improvement may have been due, at least in part, to this latter agent.

If this fact is true, the mechanism of action is not known. It is intriguing to speculate that benefit may have been secondary to an antimicrobial action of metronidazole. For example, it is at least conceivable that an opportunistic neurotoxin-producing organism may have colonized the GI tract. Both Crohn’s disease [4] and broad-spectrum antimicrobials are known to alter the GI flora, which might have allowed such colonization. This patient had both risk factors, the latter including frequent exposure to broad-spectrum antibiotics for several years prior to the onset of his psychiatric symptoms.

Psychiatric symptoms have previously been associated with altered intestinal flora. For example, patients with D-lactic acidosis (a condition caused by bacterial overgrowth in the small intestine) may present with a range of behavioral changes (e.g., hostility, slurred speech, stupor, deranged mental status, dizziness, and ataxia), and treatment with oral antibiotics usually results in rapid improvement [5]. Whatever the mechanism, further research into a possible gut microbe–brain connection may be helpful.

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References

Outbreak of Q Fever Following a Safari Trip

Travelers returning from the tropics may acquire miscellaneous febrile respiratory illnesses. Reports of Q fever pneumonia acquired by travelers are anecdotal, and a cluster of Q fever pneumonia has never been reported [1, 2]. We describe an outbreak of Q fever in safari travelers.

A 41-year-old man presented with fever (temperature, 38.2°C), headache, and backache of 3 days’ duration. Four weeks earlier, he had headed a group of 50 employees on a 1-week safari trip to Kenya. His initial physical examination was unremarkable. Three days later, he presented again with continuing fever and a mild cough. His erythrocyte sedimentation rate was 86 mm/h, and microscopic hematuria was found. Smears were negative for malarial parasites. Treatment with ciprofloxacin for a presumed urinary tract infection was commenced.
A chest radiogram showed a mild left sub hilar infiltrate. Ami notransferase levels were mildly elevated, and a PPD skin test was negative. Treatment was changed to clarithromycin (250 mg b.i.d.), and 3 days later, his temperature decreased. Antibiotic therapy was continued for 10 days. Serology for *Coxella burnetii* phase II antigen (microimmunofluorescence) showed an increase of the IgG titer from <1:100 to 1:1600 and a biphasic pattern for the IgM titer (<1:100 to 1:800 and then 1:200). Serological tests for *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Chlamydia psittaci*, *Leptospira*, enterovirus, respiratory syncytial virus, adenovirus, influenza A and B viruses, hantavirus, Rift Valley fever virus, Sindbis virus, chikungunya virus, dengue virus, West Nile virus, and *Legionella* urinary antigen were all negative. Because his cough and fatigue continued, doxycycline therapy was initiated for 10 days.

At his second visit, the index patient mentioned that a 32-year-old female employee had pneumonia unresponsive to cefuroxime axetil. A common source outbreak was suspected, and an inquiry was instituted. Q fever serology for the woman was strongly positive for IgM and IgG antibodies to *C. burnetii* phase II antigen (titers of 1:2000 for both antibodies). Serologies for other agents of atypical pneumonia were negative. She was treated with doxycycline for 2 weeks, and her fever resolved rapidly.

The travel itinerary included a stop at the Masai Mara reserve. The 2 sick patients denied drinking unpasteurized milk. As far as they could recall, the only place that they had visited together was a tribal shack in the game reserve that was 2 × 3 m in size, made of cattle hides and straw, and covered with mud and/or manure. Inside the shack, the group observed 2 goats and a burning oven. Both patients recalled inhaling noxious fumes during that short visit and feeling sick the next day.

Because 4 other people entered the shack, a case-control investigation was carried out. Blood samples were obtained from all 6 people who entered the shack and from 17 travelers who visited other shacks. Some of the 17 travelers indicated that they had peeked into this particular shack. Serology revealed an additional asymptomatic case in those people who entered the shack and 1 asymptomatic case in those who did not enter the shack, but this patient admitted to having peeked into the shack. Entering the shack thus constituted a significant risk factor for contracting Q fever (RR, 8.5; *P* = .04, two-tailed Fisher’s exact test). After departing Kenya, none of the 3 travelers who entered the shack and were asymptomatic, nor the 17 who entered other shacks, became ill during a 6-week follow-up period.

Our investigation found that 4 (8%) of 50 safari travelers to Kenya contracted Q fever; 2 travelers developed overt infection, whereas 2 others developed asymptomatic illness. Information about the prevalence of *C. burnetii* in Africa is scant [3, 4]. To our knowledge, this is the first report of an outbreak of Q fever acquired on this continent. The prevalence of antibodies to *C. burnetii* among various ethnic populations in Kenya has ranged from 10% to 20% [5, 6]. *C. burnetii* is transmitted to humans mainly via airborne dust. It is therefore conceivable that this cluster of cases occurred during exposure at the Masai shack. Either the shack or the 2 goats could have served as a source for transmission; furthermore, entrance to the shack was significantly associated with disease, which attests to the respiratory mode of transmission.

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**References**


**Pneumocephalus Due to Invasive Fungal Sinusitis**

Pneumocephalus is an accumulation of intracranial air that occurs when there is a connection between the intracranial space and the extracranial space. Rarely, tension pneumocephalus develops—an accumulation of air through a one-way valve that causes dramatic neurological deterioration. Two proposed mechanisms are a ball-valve effect—where air is pulled into the intracranial space during coughing, swallowing, or straining—and an inverted-bottle effect—where excessive leakage of CSF leads to negative intracranial pressure and replacement of fluid with air [1]. We present a unique case of pneumocephalus secondary to a fungal infection.

A 77-year-old man with a history of steroid-dependent autoimmune hemolytic anemia and myelodysplastic syndrome was admitted to the hospital with a 1-day history of altered mental status. He was somnolent and withdrew only from painful stimuli. His vital signs were stable except for a respiratory