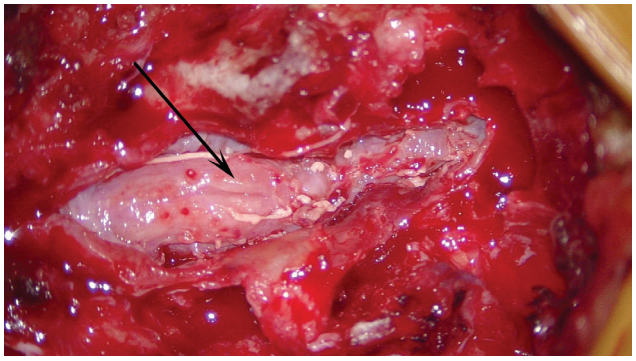

Figure. Intraoperative image demonstrating postevacuation cauda equina nerve roots that are grossly edematous and adherent (arrow), consistent with arachnoiditis, in a patient with recurrent infection from fungal-contaminated methylprednisolone, North Carolina, USA, 2015.

condition was treated with intravenous amphotericin B and voriconazole during her 12-day hospitalization, and she was discharged on oral voriconazole for outpatient treatment, with an anticipated duration of 1 year. At 5-month follow-up, she had complete resolution of her back pain and was full strength with some intermittent left radicular pain.

Only 3 other cases of intradural abscess were reported from the initial outbreak, making this recurrence a notable CNS disease manifestation (*5*). The patient had several risk factors for recurrence. She had received epidural steroid injections after antifungal treatment; the steroids resulted in an immunocompromised environment, potentially allowing for immune evasion and residual disease. A dural rent during multiple spinal taps or posttreatment epidural steroid injections may have seeded the fungus in the intradural space, which then expanded because antifungal agents demonstrate relatively poor CSF penetration. She also underwent a 3-month initial treatment; at least 6 months of antifungal treatment is currently recommended, although optimal treatment duration remains uncertain because objective criteria for infection clearance are lacking.

Given the potential for recurrence, fungal disease should remain on the differential diagnosis list for patients with prior exposure. In addition, long-term follow-up could identify patients needing further treatment (*4*).

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Successful Treatment of Human Plague with Oral Ciprofloxacin

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The US Food and Drug Administration recently approved ciprofloxacin for treatment of plague (*Yersina pestis* infection) based on animal studies. Published evidence of efficacy in humans is sparse. We report 5 cases of culture-confirmed human plague treated successfully with oral ciprofloxacin, including 1 case of pneumonic plague.

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Plague is a life-threatening zoonotic disease caused by *Yersinia pestis*. Zoonotic foci exist on several continents; however, resource-poor areas in sub-Saharan Africa account for most human cases (*1*). The pathogenesis of plague involves facultative intracellular infection of host macrophages, followed by fulminant extracellular growth and bacteremia (*2*). In the absence of effective antimicrobial drug treatment, bubonic plague is fatal in ≈50% of cases and pneumonic plague in >90% (*1*,*3*).

Drugs approved by the US Food and Drug Administration (FDA) for treatment of plague include streptomycin and doxycycline. Streptomycin is bactericidal but rarely used because of limited availability and serious toxicities. Doxycycline is bacteriostatic and lacks concentration-dependent activity or a postantibiotic effect, which might limit its efficacy for serious *Y. pestis* infections (*4*). Nevertheless, low cost and oral dosing have made doxycycline a first-line treatment in several countries (*5*,*6*). Fluoroquinolones, include ciprofloxacin, have recently been approved by the FDA for treatment of plague based on animal and in vitro studies (*4*,*7*,*8*). Clinical experience with these agents, however, is limited (*1*,*5*).

During 2011–2014, patients with suspected plague seen at 6 clinics and 2 hospitals in the West Nile region of Uganda were offered enrollment in an open-label study evaluating the safety and efficacy of ciprofloxacin for treatment of plague. Patients were excluded if they were pregnant, <8 years of age, considered too ill to receive oral treatment, or had received antimicrobial drug treatment in the preceding 7 days. After written consent was obtained, diagnostic samples were collected and oral ciprofloxacin administered for 10 days at a weight-calibrated dosage of ≈15 mg/kg twice daily (range 13–17 mg/kg), with a maximum dose for adults of 750 mg twice daily. Diagnostic samples were cultured on sheep blood agar and suspect isolates confirmed by bacteriophage lysis (*9*). Patients were monitored daily during treatment, and clinical outcome was assessed 14–21 days after initial evaluation. Because of simultaneous prevention efforts and lower than expected enrollment, the study was terminated early. The study was approved by Institutional Review Boards at the Uganda Virus Research Institute, the Uganda National Council for Science and Technology, and the US Centers for Disease Control and Prevention.

Five patients with culture-confirmed plague were enrolled and treated with oral ciprofloxacin (Table). Median patient age was 27 years (range 10–52 years); median time between illness onset and enrollment was 4 days (range 1–7 days). Four patients had bubonic plague, with *Y. pestis* isolated from bubo aspirates or blood cultures. The fifth patient, a 13-year-old boy, had pneumonic plague as indicated by hemoptysis, patchy bilateral infiltrates on chest radiograph, and *Y. pestis* isolated from sputum. The illness had evolved over 6 days, a clinical course suggestive of secondary rather than primary pneumonic plague (*3*); the primary focus of infection was not identified.

Three patients were admitted and 2 treated as outpatients. In addition to ciprofloxacin, all received acetaminophen, and 2 received a bolus of normal saline. All became afebrile within 2 days. At 14 days, all had been discharged and returned to their normal activities. The 13-year-old boy with culture-confirmed pneumonic plague reported mild, nonproductive cough, but no complications were identified.

Fluoroquinolones have pharmacokinetic properties that make them attractive for treatment of plague, including bactericidal activity, good oral bioavailability, excellent tissue penetration, and an established safety record (*8*,*10*). In vitro assays suggest that ciprofloxacin is comparable to streptomycin and superior to doxycycline or gentamicin for killing of intracellular *Y. pestis* (*4*), and efficacy has been demonstrated in rodent and nonhuman primate models (*8*). Along with FDA approval, our results add to growing clinical experience (*5*) and support the broader use of oral ciprofloxacin for treatment of human plague, especially in resource-poor areas where intravenous treatment is limited.

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Table. Demographic and clinical characteristics of 5 patients with culture-confirmed plague (*Yersina pestis* infection) who were treated successfully with oral ciprofloxacin, Uganda, 2011–2014

Patient no.	Age, y/sex	Length of illness, d*	Symptoms	Laboratory evidence	Ciprofloxacin dose, mg†
1	10/F	7	Fever, left axillary bubo	Bubo, blood cultures positive	250
2	52/F	4	Fever, right axillary bubo	Bubo, blood cultures positive	650
3	27/F	1	Fever, left inguinal bubo	Bubo, blood cultures positive	750
4	36/M	1	Fever, left axillary bubo	Blood culture positive	625
5	13/M	6	Fever, chest pain, cough, blood-tinged sputum	Sputum culture positive, blood culture negative	375

*At time treatment was sought.
†Orally, twice daily; ≈15 mg/kg bodyweight with a maximum of 750 mg.

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Mycobacterium tuberculosis Infection in Free-Roaming Wild Asian Elephant

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Postmortem examination of a wild Asian elephant at Rajiv Gandhi National Park, India, revealed nodular lesions, granulomas with central caseation, and acid-fast bacilli in the lungs. PCR and nucleotide sequencing confirmed the presence of *Mycobacterium tuberculosis*. This study indicates that wild elephants can harbor *M. tuberculosis* that can become fatal.

Tuberculosis (TB), a pandemic, highly contagious disease caused by *Mycobacterium tuberculosis* complex, has affected up to one third of the world's human population. The South-East Asia Region (SEAR), which contains nearly one fourth of the world population, alone accounts for 38% of illnesses and 39% deaths caused by TB worldwide. India accounts for 58% of all forms of TB in SEAR and 55.6% of deaths caused by TB (excluding those among HIV-positive persons) in SEAR (*1*).

M. bovis is widespread in domestic animals and has been extensively documented in both captive and free-ranging wildlife. Although *M. tuberculosis* is primarily a pathogen of humans (*2*), it has been reported in zoo species (*3*,*4*) as well as in a formerly captive elephant in Africa (*5*) and a free-roaming elephant in Sri Lanka (*6*). We report the pathology and molecular characterization of *M. tuberculosis* in a wild Asian elephant (*Elephas maximus*) that had no known history of human contact and present implications for wildlife health.

In February 2016, a carcass of an ≈65-year-old free-roaming wild Asian elephant was found in the forest of Rajiv Gandhi National Park (RGNP), Karnataka, India. On postmortem examination, the lungs showed widely disseminated white-yellowish firm nodules with central caseous necrosis, distributed throughout the parenchyma (Figure, panel A). The bronchial and mediastinal lymph nodes were enlarged with nodular areas of caseous necrosis and calcification. Impression smears from the cut surfaces of lungs on staining by Ziehl-Neelsen method showed bundles of pink-stained acid-fast organisms.

DNA extracted from the lung tissue were subjected to PCR targeting amplification of a conserved region on *M. tuberculosis* complex by using forward primer 5′-GACCACGACCGAAGAATCCGCTG-3′ and reverse primer 5′-CGGACAGGCCGAGTTTGGTCATC-3′ (*7*), which yielded a specific amplicon of 445 bp, indicating presence of a pathogenic mycobacterium. To detect *M. bovis*, we used forward primer 5′-CACCCCGATGATCTTCTGTT-3′ and reverse primer 5′-GCCAGTTTGCATTGCTATT-3¢to amplify an 823-bp region on a 12.7-kb fragment of *M. bovis*. To detect *M. tuberculosis*, we used forward primer