a PI were included in the study, and subjects were analyzed according to their initial regimen. We excluded persons who dropped out of the study, as well as those who withdrew or discontinued PEP because the person who was the source of potential exposure tested negative for HIV-1 infection. Discontinuation of PEP was defined as a duration of PEP of <28 days.

The updated analysis confirms, in a larger study sample, that a higher proportion of individuals in the 3-drug group reported adverse effects, but the difference in the frequency of PEP discontinuation resulting from adverse effects was not statistically significant. In the 3-drug group, a subgroup of 104 individuals discontinued receipt of the PI alone because of adverse effects. Adverse effects persisted in 20 of these individuals, and PEP was subsequently discontinued after a mean of 7.5 additional days of 2-NRTI therapy (median, 5 days; range, 1–20 days). The remaining 84 individuals discontinued use of the PI after a mean of 9.3 days (median, 7 days; range, 2–25 days) and completed the 4-week course of 2-NRTI PEP.

What constitutes the optimal PEP regimen is still a debatable issue. The Centers for Disease Control and Prevention (CDC) recommend the use of 2 NRTIs as a standard initial regimen, with addition of a third drug for higher-risk exposures, according to a complex risk assessment [5]. Consistent with the conclusions of Bassett et al. [1], the higher toxicity associated with the 3-drug regimen is a major reason for this conservative approach. Other recommendations in Europe [2] and the United States [6, 7] differ from those of the CDC and propose a 3-drug regimen—regimens that, for the most part, include a PI—in all cases in which PEP is started.

In the light of current resistance rates among patients who are sources of occupational exposure [8], we embrace the premise of Bassett et al. [1] that use of a third drug in the regimen would help ensure that at least 1 drug is active against the potentially transmitted virus. According to our data, in the case of adverse effects that are not manageable, stopping the receipt of the PI alone seems to be a practicable approach, which allows the longest possible exposure to a potentially more potent regimen while maintaining the ultimate goal of treatment completion.

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Pulmonary Infection Due to Mycobacterium marinum in an Immunocompetent Patient

Sir—Mycobacterium marinum is a slow-growing and free-living mycobacterium that causes opportunistic infection in hu-
mans. This organism is widely distributed in aquatic environments [1], especially in relatively still or stagnant water. Infection is acquired by direct inoculation with the bacterium in an aquatic environment. It is a well-described cause of “swimming pool granuloma” or “fish tank granuloma” [2], which is manifested by cutaneous ulcer, nodules, or nodular lymphangitis, usually after the exposure of lacerations or abrasions to fresh water or salt water or after injuries associated with fish spines. To our knowledge, *M. marinum* has never been reported as a cause of pulmonary infection in humans in the English-language literature.

The patient was a 51-year-old, previously healthy female homemaker who had 1 episode of cough with a small amount of hemosputum 5 days before hospital admission. The patient had no history of medical illness and had no risk factors for HIV infection. She did not recall receiving any insect bites, injuries, or wounds over the trunk or extremities. The patient denied a history of swimming, handling of fishes, or any exposure to a marine environment or animals in the 3 months before the onset of symptoms. The patient had no history of fever, chills, body weight loss, night sweats, dyspnea, chest pain, or use of an anticoagulant. Physical examination revealed that the breathing sound was clear, and no neck lymphadenopathy was found. The skin of 4 limbs was intact. The WBC count was 8200 cells/mm³ (52.8% neutrophils and 36.7% lymphocytes), and the carciinoembryonic antigen level was 0.65 ng/mL (normal value, <3 ng/mL). The results of cytologic testing, tuberculosis culture, and acid-fast staining of sputum samples were all negative. Bronchoscopy did not detect any bleeder or endobronchial lesion. Chest radiography revealed a nodular lesion with increased infiltration over the right lower lung field. Chest CT disclosed 1 indistinct nodular lesion in the right lower lobe, with pleural retraction and segmental atelectasis over the right lower lobe. No cavitation or calcification was seen. No thoracic adenopathies or abdominal abnormalities were found. Because of concern about possible malignancy, video-assisted thoracoscopic surgery was used to perform open lung biopsy. During the operation, right B6 segment atelectasis with an indurated lesion (size, ∼4 × ∼1.5 cm) was noted, and wedge resection of the right B6 segment was done. Pathologic examination revealed granulomatous inflammation with occasional Langhans’ giant cell formation, but no organisms were identified by mycobacterial and fungal stain. Under the presumption of a diagnosis of tuberculosis, the patient was treated with isoniazid, rifampin, pyrazinamide, and ethambutol. The tissue biopsy specimen was ground and inoculated onto Lowenstein-Jensen media slants and cultured at 35°C in a CO₂ incubator (5% CO₂). After 20 days of incubation, the culture yielded a yellow-pigmented photochromogen, which was later identified as *M. marinum* by conventional biochemical methods.

Combination therapy with isoniazid (300 mg q.d.), rifampin (450 mg q.d.), and ethambutol (800 mg q.d.) was given to treat a commonly encountered photochromogen (*M. kansasii*) associated with lung infection, in addition to treating *M. marinum* [2]. The patient received the aforementioned regimen for a total of 6 months. A postoperative chest radiograph revealed scarring in the area of the biopsy. A chest radiograph obtained several months after completion of the combination therapy showed the absence of any pulmonary lesions. The isolate was further confirmed to be *M. marinum* by sequencing of the 16S rRNA gene [3].

Although pulmonary *M. marinum* infection has been reported in some animals, such as manatees [4] and pythons [5], this is the first reported case of a pulmonary lesion due to *M. marinum* in an immunocompetent host. Numerous cases of infection in humans due to *M. marinum* acquired from contaminated pools, rivers, and old wells have been reported. Exposure to saltwater and freshwater fish, dolphins, shrimp, oysters, and snails has been associated with this infection in humans [6]. This infection may be an occupational hazard for persons in certain professions (e.g., pet shop workers) or with certain hobbies (e.g., fish fanciers). The portal of entry of the organism in our patient was not easily clarified because of the absence of any recent contact with marine animals or environmental conditions suitable for propagation of the organism. The organism could gain access into the lung by aspiration or by hematogenous spread via a contaminated trivial cutaneous lesion.

The optimal regimen for treatment of *M. marinum* infection includes clarithromycin combined with either rifampin or ethambutol [7]. Combination therapy with a regimen that does not include clarithromycin (i.e., rifampin and ethambutol only) has also been demonstrated to be effective for this infection [8]. Our patient was successfully treated with surgical excision of the lung lesion and subsequent treatment with a conventional antituberculosis regimen that included rifampin and ethambutol for 6 months.

This case expands the spectrum of infection caused by *M. marinum* and raises the possibility of *M. marinum* as one of the causes of granulomatous pulmonary disease.

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Occurrence of Ceftriaxone Resistance in Ciprofloxacin-Resistant Salmonella enterica Serotype Choleraesuis Isolates Causing Recurrent Infection

Str—The increasing rates of resistance to traditional anti-Salmonella agents (i.e., ampicillin, chloramphenicol, and trimethoprim-sulfamethoxazole [TMP-SMX]), extended-spectrum cephalosporins, and fluoroquinolones among Salmonella isolates have made treatment of invasive salmonellosis a clinical dilemma [1–3]. In Taiwan, the recent emergence of resistance to ciprofloxacin and ceftriaxone among Salmonella species—particularly Salmonella enterica serotype Choleraesuis—has been reported [2, 3].

A 48-year-old woman had been relatively healthy, without any systemic disease, until she began to experience chills followed by fever starting at the end of August 2002. She was admitted to a hospital once in September 2002 and once in October 2002. Blood cultures performed during the 2 hospitalizations both yielded non-typhoid Salmonella, which was susceptible to ceftriaxone but resistant to ciprofloxacin, as determined with the disk diffusion method. Defervescence was noted after treatment with cefazolin, and gentamicin was started.

In November 2002, the patient experienced fever again and was admitted to National Taiwan University Hospital (Taipei). Initially, S. Choleraesuis was isolated from 2 sets of blood cultures (isolate A) (table 1), which was resistant to ampicillin, chloramphenicol, TMP-SMX, and ciprofloxacin. The patient was treated with ceftriaxone (1 g q12h). However, hypotension developed on the next day. The antibiotic regimen was changed to imipenem (500 mg q8h), and the patient’s fever subsided 2 days later.

Unfortunately, the patient again developed fever associated with abdominal cramping pain and diarrhea on 30 December 2002, at which point she had received imipenem for 13 days and oral ciprofloxacin for 4 days. Blood (isolate B) and stool (isolate C) cultures both yielded the same organism. Intensive diagnostic evaluations, including colonoscopy, abdominal sonography, pelvic CT, and a whole-body gallium scan all yielded negative results. During this episode, imipenem was given for 20 days, followed by oral cefixime (200 mg q12h), and the patient remained afebrile.

In early March 2003, the patient developed fever and bilateral ankle and heel pain. A 3-phase bone scan examination revealed a focal hot spot over the right parietal bone of the skull. Blood culture results were negative. Another course of treatment was given with imipenem for 14 days, and high-dose ciprofloxacin (750 mg q12h) was given for 5 days.

On 30 March 2003, the patient was admitted to the hospital because of a recurrent fever (duration, 4–5 days). Isolates of S. Choleraesuis, which were resistant to chloramphenicol, ampicillin, ceftriaxone, and ciprofloxacin but susceptible to TMP-SMX, were recovered from blood samples (isolate D). Whole-body bone scan demonstrated progression of multiple areas of increased tracer uptake over the skeleton. Chest CT and mammography did not reveal any abnormalities. During this hospitalization, imipenem (500 mg q8h) was administered for 28 days, and the patient was discharged on 2 April 2003.

Another febrile episode occurred on 9 June 2003; it was associated with headache pain over the occipital area, pain over the bilateral calf, and tenderness over both feet. An S. Choleraesuis isolate (isolate E) with different susceptibility patterns (it was resistant to TMP-SMX) was also found in the blood samples. A whole-body gallium survey revealed increased tracer activity in the bilateral para-prevertebral regions at the T12 level. MRI of the spine illustrated multiple bony metastases over the thoracolumbar spine. Fever persisted during imipenem therapy for 12 days. Because drug-associated fever due to imipenem use was suspected, parenteral TMP-SMX was administered, and the fever subsided thereafter. Breakthrough bacteremia due to the same organism (isolate F) occurred on 5 July, and combination therapy with aztreonam, meropenem, and amikacin was given. The patient remained afebrile for 7 days after receiving combination therapy, and she fully recovered after receipt of treatment with the aforementioned agents for 6 weeks.

As determined by the agar dilution method, all of these isolates were found to be resistant to ampicillin, chloramphenicol, ciprofloxacin (MIC, 32–64 µg/mL), levofloxacin (MIC, 16–32 µg/mL), moxifloxacin (MIC, ≥32 µg/mL), and garenoxacin (MIC, >32 µg/mL) but to be susceptible to cefepime (MIC, 0.12–1 µg/mL), imipenem (MIC, 0.25–0.5 µg/mL), ertapenem (MIC, 0.03–0.12 µg/mL), and faropenem (MIC, 0.5–1 µg/mL) (table 1).

All of the 6 ciprofloxacin-resistant isolates had 2-base substitutions in the