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Pandemic (H1N1) 2009 and HIV Co-infection

To the Editor: We report a case of pandemic (H1N1) 2009 infection in a man with serologic evidence of HIV-1 infection. The clinical course was complicated by lung and brain involvement (respiratory failure and lethargy), severe leukopenia, and thrombocytopenia, but complications resolved after treatment with oseltamivir (150 mg 2×/d).

In November 2009, a 47-year-old man who had received a diagnosis of hepatitis C infection 8 months earlier sought treatment at Ospedale Santa Maria Nuova, Reggio Emilia, Italy. He had a 3-day history of fever, dry cough, and drowsiness. Eight days before being admitted, the man had resided in the hospital's inpatient detoxification unit, in which at least 10 influenza-like cases had been recorded. While in the detoxification unit, he had received methadone, 50 mg 1×/d. Computed tomography images of the brain and radiographs of the chest were normal; ultrasound examination showed upper lobe consolidation of the left lung. Hematochemistry showed high creatine phosphokinase levels, leukocyte count 1,380 cell/mm^3 (reference range 4,000–10,000 cells/mm^3), thrombocyte count 34,000 cells/mm^3 (reference range 150,000–450,000 cells/mm^3), partial pressure of oxygen 56 mm Hg, and partial pressure of carbon dioxide 53 mm Hg. Urinalysis results were negative for heroin, cocaine, and alcohol; cerebrospinal fluid (CSF) analysis results were within normal limits. Thrombocyte count returned to reference range after 2 days, and leukocyte count improved but remained <3,500 cells/mm^3 for 3 weeks. After admission to hospital, the man became lethargic and received noninvasive continuous positive airway pressure ventilation and treated with oseltamivir (150 mg 2×/d for 5 d), as well as with ceftriaxone, and levofloxacin. Reverse transcription–PCR on a throat swab confirmed influenza subtype H1N1 infection; blood cultures and urine were negative for pneumococcus, and *Legionella* spp. antigens. In addition, PCR of CSF for enterovirus and herpesvirus had negative results. The patient needed respiratory support for 4 days, after which his mental status and blood gases returned to reference levels. He was discharged from the hospital 2 weeks later.

On day 3 after admission, a nurse was accidentally exposed to the patient's urine through her eye. An ELISA was positive for HIV infection. Negative results for confirmatory Western blot tests on days 5, 15, and 23 showed the p24 and p41 bands; HIV RNA was >6 million copies/mL, CD4 lymphocytes 51% (reference range 29%–59%). Reverse transcription–PCR for influenza subtype H1N1 performed 2 months later on a stored CSF sample gave a negative result; PCR for HIV of the same sample indicated 25,000 copies/mL. In mid-December, because of a further drop in CD4 lymphocytes to 17% (214 cells/mm^3) and blood HIV RNA of 2.8 million copies/mL, the patient started highly active antiretroviral therapy and is being followed up as an outpatient.

Influenza (H1N1) and primary HIV infection share many signs and symptoms, such as fever, cough, sore throat, joint or limb pain, and diarrhea. The infections also share uncommon complications of the central nervous system (CNS); e.g., drowsiness, coma, and seizures. We cannot confirm that CNS involvement in the patient reported here was caused primarily by pandemic (H1N1) 2009, as suggested by influenza-like symptoms and the apparent effect of oseltamivir. Nor can we attribute CNS involvement to primary infection with HIV-1 (*1*); CSF results within normal limits and PCR negative for influenza subtype H1N1 do not rule out a causal relationship with pandemic (H1N1) 2009. In fact, the few cases of pandemic (H1N1) 2009 encephalopathy described show similar characteristics among children and adults (*2*–*4*). Alternatively, some authors have attributed HIV in CSF to brain inflammation and damage (*5*,*6*). The severe leukocytopenia and thrombocytopenia in our patient have not been described, even in complicated influenza subtype H1N1 infections (*7*). Because lymphopenia and mild thrombocytopenia are the usual findings, we believe that they probably resulted from HIV-1 or the effect of both viruses.

HIV seroconversion may initially occur during an acute febrile illness resembling influenza, and CNS involvement can complicate both infections. During an epidemic, acute HIV infection should also be considered (*8*). Less frequently, as in the patient described above, the 2 infections can occur simultaneously. History of recent risk behavior for blood exposure and severe leukocytopenia and thrombocytopenia should alert clinicians to other causes and prompt them to offer an HIV test to the patient.

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Dictyostelium polycephalum Infection of Human Cornea

To the Editor: Although *Dictyostelium* spp. are used for studying signal transduction, cytoskeletal functions, endocytosis, and molecular pathogenesis of infectious and other diseases (*1*), human or animal infections caused by this organism have not been reported. We report a case of keratitis caused by *Dictyostelium polycephalum* in an immunocompetent person.

A 35-year-old man sought treatment for redness, pain, and watering in the left eye of 11 days' duration. He had no history of ocular injury or surgery. At the time of his medical visit, he was using ophthalmic solutions of 5% natamycin sulfate, 0.5% moxifloxacin hydrochloride, and 0.3% gentamicin sulfate, each instilled every hour, and 1% atropine sulfate, 3×/d.

The vision in his right eye and results of a clinical examination were within normal limits. His left eye visual acuity was expressed as the ability to count fingers at 1 m. The eyelids were edematous and the conjunctivae were congested. The cornea showed a large central epithelial defect with underlying stromal infiltrate and Descemet folds. The surrounding cornea had a mild cellular reaction. The anterior chamber was deep, and the pupil was round, regular, and dilated. Iris and lens details could not be distinguished because of corneal haze. We obtained corneal scrapings, and the material was subjected to a detailed microbiologic analysis (*2*).

Microscopic examination showed double-walled spherical cysts in potassium hydroxide with calcofluor white stain, Gram stain (Figure, panels A, B), and Giemsa stain. On the basis of this finding, a presumptive diagnosis of *Acanthamoeba* keratitis was made. The patient was advised to use 0.02% polyhexamethylene biguanide and 0.02% chlorhexidine eye drops every half hour and 1% atropine eye drops 3×/d and was asked to return for a follow-up visit the next day. However, the patient did not return and could not be located. After 48 hours' of incubation, a nonnutrient agar plate showed growth of double-walled, spherical cysts ≈6–7 μm in diameter that had different morphologic features than those of *Acanthamoeba* spp. cysts.

To identify the organism, we extracted DNA from the growth on nonnutrient agar and subjected it to PCR specific for *Acanthamoeba* spp. (*3*); results were negative. The extracted DNA was then subjected to 18S rDNA PCR for free-living amebas as described by Tsvetkova et al. (*4*). A PCR product ≈800 bp was obtained and subjected to bidirectional sequencing with fluorescent-labeled dideoxy nucleotide terminators by using ABI 3130 XI automated sequencer in accordance with the manufacturer's instructions (PE Applied Biosystems, Foster City, CA, USA).

The Mega BLAST search program (www.ncbi.nlm.nih.gov/blast/megablast.shtml) of GenBank identified the sequence as *D. polycephalum* (99% similarity with AM168056). We deposited the sequence of our isolate in GenBank (accession no. GU562439). The organism showed cytotoxicity after in vitro inoculation of a rabbit corneal epithelial cell line.

The patient sought treatment 4 months after his initial visit. The left eye visual acuity was now expressed as the ability to see hand movements near the face. Slit-lamp examination showed lid edema and conjunctival congestion. The cornea showed a ring-shaped infiltrate, central thinning, surrounding corneal edema, and pigments on the endothelium (Figure, panel C); these findings were identical to the clinical picture of *Acanthamoeba* keratitis. Repeat corneal scrapings showed organisms of same morphologic features seen on the first visit by microscopy and culture. Organisms