Clinical and Vaccine Immunology

Lymphocytic Choriomeningitis in a Pet Store Worker in Romania

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Lymphocytic choriomeningitis virus (LCMV) causes aseptic meningitis, although it may be associated with systemic organ failure in immunocompromised patients (1, 3, 5). LCMV is also a teratogenic pathogen causing a severe and often fatal syndrome with hydrocephalus and chorioretinitis (4, 7). Afflicted infants suffer from blindness, deafness, mental retardation, and paraparesis. The reservoirs for the virus are mice and hamsters, which shed the virus in urine and other body excretions. Several cases of LCMV infection were recently reported from southern France (2, 6). Here, we report a case of LCMV infection in a pet store worker in Romania.

A 27-year-old man was admitted to the Hospital of Infectious Diseases, Cluj-Napoca, Romania, on 12 February 2008, due to suspected aseptic meningitis. The patient worked in a pet store selling mice, hamsters, guinea pigs, squirrels, mustelids, chinchillas, and birds. He presented with high fever (>40°C), chills, nausea, persistent vomiting, headache, photophobia, neck stiffness, drowsiness, confusion, and bradycardia (54 beats/min). Kernig’s and Brudzinski’s signs were absent. A computed tomography scan of the patient’s brain showed neither pathological uptake of contrast dye nor cerebral edema. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis (800 cells/mm³), a slightly elevated protein level (77 mg/dl), and a normal blood/CSF glucose ratio. CSF, urine, and blood cultures were negative for fungi and bacteria, including mycobacteria. The patient did not show signs of immune deficiency, and serological testing revealed no evidence of acute infection with herpesviruses, *Toxoplasma gondii*, *Chlamydia pneumoniae* and *C. psittaci*, *Leptospira*, *Mycoplasma pneumoniae*, and *Borrelia* spp. Serum and CSF samples were sent to the Bernhard-Nocht-Institute for Tropical Medicine in Germany for LCMV diagnostics. Immunofluorescence assay of LCMV-infected cells showed an immunoglobulin G (IgG) titer of 1:320 (cutoff, 1:20) and an IgM titer of 1:80 (cutoff, 1:20) in the serum sample from the acute phase and an IgG titer of 1:5120 and an IgM titer of 1:320 in the follow-up sample, demonstrating an acute LCMV infection. IgG (titer, 1:160) and IgM (titer, 1:80) antibodies were also detected in a CSF sample. LCMV reverse transcription-PCR with serum and CSF was negative. The patient was treated with acetylsalicylic acid (30 mg/kg of body weight/day for 10 days) due to an initial suspicion of herpesvirus infection, mannitol (1 g/kg/day for 5 days), and dexamethasone (16 mg/day for 5 days). His condition improved, and he was discharged on 3 March 2008.

After the Romanian public health and veterinary services were notified, all small mammals of the pet store were culled, the premises were disinfected, and wild-rodent control measures were introduced. However, rodents were disposed without samples being collected for laboratory investigation. Serum samples were taken from all employees of the pet store (n = 11) and screened for LCMV-specific antibodies. Evidence of a previous LCMV infection was found in one person (IgG titer, 1:40).

This report describes a case of LCMV infection that was presumably acquired via contact with mice or hamsters in a pet store, although other sources of infection cannot be excluded. Besides the fact that workers involved in breeding and handling pet rodents are at risk of contracting the disease, selling LCMV-infected rodents is a major public health issue (1). Consequences of infection are particularly serious for pregnant women and their unborn children (4, 7). This case underscores the fact that pet rodents should not come in contact with wild rodents, to prevent the spread of virus to the public.

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