In November 2009, a healthy 2-year-old boy was admitted to the pediatric intensive care unit at the Western Galilee Hospital in Nahariya, Israel, after he had been hit by a car. One day before the accident, he had exhibited fever and cough (for which he was treated with acetaminophen). His 4-year-old brother had recovered recently from an influenza-like illness without antiviral treatment. The other household contacts were his parents, who did not have a respiratory illness.

On admission, small, bilateral lung contusions, right pneumothorax, and liver lacerations were shown on computed tomographic scan. The patient was treated with a chest tube for drainage, supplemental oxygen, and oseltamivir from hospital day 1 (30 mg 2 ×/day; child’s body weight = 13 kg) and was placed in droplet isolation. Respiratory swab specimens, obtained on hospital day 1, were sent to the Israel Central Virology Laboratory (ICVL) and found to be positive for pandemic (H1N1) 2009. By the time these laboratory results were known, the patient’s respiratory condition was improving without changing the oseltamivir therapy. Cultures of blood and endotracheal specimens were sterile and antimicrobial drug therapy was stopped. On hospital day 15, he was extubated, oseltamivir therapy was ended, and he was weaned off oxygen a few days later. The respiratory specimen on hospital day 20 was negative for pandemic (H1N1) 2009. No secondary influenza cases were detected among healthcare personnel or patients in the unit.

In Israel, oseltamivir resistance has been detected by ICVL in 6 cases (5). The fact that our patient had oseltamivir-resistant pandemic (H1N1) 2009 without a previous oseltamivir exposure is surprising because almost all cases of oseltamivir-resistance have been associated with previous oseltamivir prophylaxis or therapy and with prolonged viral shedding (which is often combined with oseltamivir therapy) in immunocompromised patients (1–5). Our patient did not attend daycare and his parents had not been ill recently. Therefore, he likely was infected by his older brother who probably had pandemic (H1N1) 2009 but was neither diagnosed nor treated with antiviral medications. This theory suggests that oseltamivir-resistant viruses circulate in the community with the potential to be transmitted between persons.
Lung contusions and pandemic (H1N1) 2009 can cause ARDS (6, 7). We do not know the relative role of each in causing the ARDS that our patient had, but the severity of clinical symptoms, although the lung injury was judged to be only of moderate magnitude, suggests that influenza played a major role in the development of his acute lung disease. The infection with oseltamivir-resistant virus, for which he probably did not receive effective therapy, likely had an effect on the duration and severity of his course.

Although our patient had a favorable outcome, the possibility of widespread resistance, similar to the phenomenon observed with seasonal influenza in the 2008–2009 season, is alarming and should be monitored. The suspicion of resistance should be based upon compatible clinical scenario, such as continuation of symptoms in spite of antiviral therapy (even in patients who are not immunocompromised), combined with early performance of resistance assays. Early and rapid detection of oseltamivir resistance and a change of antiviral treatment (if feasible) might benefit the patient.

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Human Infection with Lymphocytic Choriomeningitis Virus

To the Editor: I read with great interest the article regarding lymphocytic choriomeningitis virus (LCMV) meningitis in a New York City resident (1). The authors’ conclusion that there is a need to ascertain the true incidence of LCMV infection is worthy of underscoreing. Nearly 15 years ago, in this same journal, we described congenital LCMV as an unrecognized teratogen and recommended further “research to define the frequency of LCMV” (2). Five years later, we reiterated that recommendation when reporting acquired LCMV meningoencephalitis in an adolescent from Tucson, Arizona (3). Despite, or because of, the lack of prospective studies, the fact that this author has accrued data regarding >60 congenitally infected infants from all geographic areas in the United States during the past 15 years reinforces the concept that LCMV is a neglected pathogen whose time for more extensive study has indeed come.

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