Perinatal Pandemic (H1N1) 2009 Infection, Thailand

To the Editor: Infection with influenza A pandemic (H1N1) 2009 has been reported worldwide following initial identification of the virus in April 2009 (1). The groups at highest risk for infection or influenza-related complications include pregnant women and children (2). We report a case of pandemic (H1N1) 2009 infection in a newborn whose mother became ill with pandemic (H1N1) 2009 during the perinatal period.

A newborn girl showed signs of respiratory distress. The relevant perinatal history was maternal illness with pandemic (H1N1) 2009 7 days before delivery. The infant, who had a birth weight of 1,560 grams, was delivered by emergency cesarean section after the mother experienced cardiopulmonary failure at the gestational age of 31 weeks. Apgar scores were 9 and 9 at 1 and 5 minutes, respectively. Physical examination at birth showed a premature infant girl with mild substernal retraction. Oxygen saturation at room air was 91%–99%. Other results of the physical examination were unremarkable.

Initial management included routine care for premature infants. On the basis of the perinatal history, a throat swab specimen was collected for pandemic (H1N1) 2009 testing by PCR and oseltamivir, 6 mg, was administered every 12 hours (4 mg/kg/day). The specimen obtained from the throat swab was positive for pandemic (H1N1) 2009 by real-time PCR. The infant required oxygen supplementation gradually decreased and finally discontinued. Her room air oxygen saturation was 95%–98%. Her clinical symptoms gradually improved. Hemoculture was negative after 72 hours. The antimicrobial drugs were given over an 8-day course. Plasma creatinine decreased to 0.9 mg/dL and 0.6 mg/dL at days 6 and 7 of life, respectively. Her average urine output was 2–3 mL/kg/h. She was discharged at the age of 28 days with a body weight of 2,070 grams.

Antibody titers against pandemic influenza (H1N1) 2009 by HI with turkey erythrocytes (4) on days 10, 24, and 42 of life were 10, 160, and 320, respectively (Figure). At day 4 of life, repeated PCR performed on a throat swab specimen was negative for pandemic (H1N1) 2009. Oxygen supplementation was gradually decreased and finally discontinued. Her room air oxygen saturation was 95%–98%. Her clinical symptoms gradually improved. Hemoculture was negative after 72 hours. The antimicrobial drugs were given over an 8-day course. Plasma creatinine decreased to 0.9 mg/dL and 0.6 mg/dL at days 6 and 7 of life, respectively. Her average urine output was 2–3 mL/kg/h. She was discharged at the age of 28 days with a body weight of 2,070 grams.

Figure. Antibody titer against influenza A pandemic (H1N1) 2009 by hemagglutination inhibition (HI) test on days 10, 24, and 42 of life of the patient. A color version of this figure is available online (www.cdc.gov/EID/content/16/2/343-F.htm).
cific. The high plasma creatinine level in the newborn sometimes reflects the mother’s plasma creatinine level (9). However, kidney function of the mother of the newborn was within normal limits at the time of Caesarean section; plasma creatinine level of 0.7 mg/dL. An elevated plasma creatinine level is observed frequently in premature infants due to immaturity of the kidney tissue and will usually decrease within a few weeks. Oseltamivir was administered with dose adjustment based on the infant’s estimated glomerular filtration rate. The recommended dose of oseltamivir for glomerular filtration rate <30 mL/min/1.73 m² is 2–3 mg/kg/day, based on preliminary data obtained by a National Institutes of Health–funded Collaborative Antiviral Study Group (10). The success of our management strategy for this case suggests early treatment with oseltamivir can prevent severe illness in newborns with perinatal influenza A pandemic (H1N1) 2009 infection.

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References

1. Centers for Disease Control and Prevention. Swine influenza A (H1N1) infection in two children—southern California, March–April 2009. MMWR Morb Mortal Wkly Rep. 2009;58:400–42.
2. Centers for Disease Control and Prevention. Use of influenza A (H1N1) 2009 monovalent vaccine: recommendations of the Advisory Committee on Immunization Practices (ACIP). 2009. MMWR Recomm Rep. 2009;58(RR-10):1–8.
3. Schwartz GJ, Brion LP, Spitzer A. The use of plasma creatinine concentration for estimating glomerular filtration rates in infants, children, and adolescents. Pediatr Clin North Am. 1987;34:571–90.
4. Rowe T, Abernathy RA, Hu-Primmer J, Thompson WW, Lu X, Lim W, et al. Detection of antibody to avian influenza A (H5N1) virus in human serum by using a combination of serologic assays. J Clin Microbiol. 1999;37:937–43.
5. Irving WL, James DK, Stephenson T, Laing P, Jameson C, Oxford JS, et al. Influenza virus infection in the second and third trimesters of pregnancy: a clinical and seroepidemiological study. BJOG. 2000;107:1282–9. DOI: 10.1111/j.1471-0528.2000.tb11621.x
6. McGregor JA, Burns JC, Levin MJ, Burlington B, Meiklejohn G. Transplantation Society Guidance on Novel Influenza A/H1N1 [cited 2009 Nov 19]. http://www.transplantation-soc.org/downloads

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Bronchial Casts and Pandemic (H1N1) 2009 Virus Infection

To the Editor: In the late 1990s, triple-reassortant influenza A viruses containing genes from avian, human, and swine influenza viruses emerged and became enzootic in swine herds in North America (1). The first 11 human cases of novel influenza A virus infection were reported to the Centers for Disease Control and Prevention (CDC; Atlanta, GA, USA) from December 2005 through February 2009 (1). In response to those reports, surveillance for human infection with nonsubtypeable influenza A viruses was implemented.

In the spring of 2009, outbreaks of febrile respiratory infections caused by a novel influenza A virus (H1N1) were reported among persons in Mexico, the United States, and Canada (2). Patient specimens were sent to CDC for real-time reverse transcription–PCR (RT-PCR) testing, and from April 15 through May 5, 2009, a total of 642 infections with the virus, now called pandemic (H1N1) 2009 virus, were confirmed. Of those 642 patients, 60% were ≤18 years of age, indicating that