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SHORT REPORT

Influenza A (H1N1) 2009 reinfection in Thailand

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Summary In 2009, a novel influenza A (H1N1) virus emerged and rapidly spread around the world, leading to a pandemic. In contrast to the high rate of primary infection, reinfection with influenza A (H1N1) 2009 is rather rare. In this report, we describe a case of influenza A (H1N1) 2009 reinfection that occurred within an interval of 5 months in Thailand.

Introduction

In April 2009, a novel influenza A (H1N1) virus emerged and rapidly spread across North America [1]. This novel virus is genetically distinct because it derives genes segments from both North American and Eurasian swine influenza virus lineages [2]. Therefore, the worldwide population was highly susceptible to infection with this novel virus. It took less than 3 months after the identification of the first case for the H1N1 pandemic to occur. In contrast to the high rate of primary infection, reinfection with H1N1 is rather rare.

In Thailand, the first cases influenza A (H1N1) were reported in May of 2009 [3], and these initial cases were followed shortly thereafter by a major outbreak. The Department of Disease Control, part of the Thai Ministry of Public Health, reported that the first outbreak lasted for 6 months (May—October 2009) and that the average rate of infection in the Thai population was 13% (about 8.4 million people) [3].

In this report, we describe a case of influenza A (H1N1) 2009 reinfection in Thailand.

Case report

In July 2009, a 39-year-old Thai woman, who was bed-ridden due to cerebral palsy and epilepsy, presented to the emergency room (ER) at the

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Faculty of Medicine, Ramathibodi Hospital, Bangkok, Thailand, with a fever, drowsiness, and a generalized tonic–clonic seizure. Six days prior to admission, she had a low-grade fever and a productive cough. The patient initially received oral co-amoxiclav. However, her clinical condition continued to deteriorate. Three to four days prior to admission, a high-grade fever and a more productive cough producing more sputum were noted. A few hours prior to presenting to the hospital, a generalized tonic–clonic seizure occurred for a brief period of time. Upon arrival, a physical examination revealed a temperature of 37.6 °C, a blood pressure of 106/86 mm Hg, a respiratory rate of 22 breaths per minute and a pulse rate of 101 beats per minute. Her maximum body temperature was 38.5 °C. A lung examination revealed bilateral rhonchi. The exam was otherwise unremarkable. Her room air oxygen saturation was 92%. Chest radiography showed increased interstitial infiltration at the left peri-hilar area of the left lower lobe. A complete blood count revealed that her hemoglobin was 11.3 g/dL; her hematocrit was 34.9%; her white blood cell count was 7650/mm^3 with neutrophils at 51%, lymphocytes at 26%, monocytes at 22%, and basophils at 1%; and her platelets were 274,000/mm^3.

The patient was admitted to the hospital. A nasal swab was tested for influenza virus by real-time polymerase chain reaction (PCR). The detection of influenza virus was carried out by the extraction of viral RNA using the NucliSens easyMAG platform for total nucleic acid extraction (bioMérieux, France) by the off-board protocol according to the manufacturer's instructions. Real-time reverse-transcriptase PCR (RT-PCR) for the detection of influenza A (H1N1) 2009 was conducted using the LightCycler 480 instrument (Roche Diagnostics GmbH, Germany) and the U.S. FDA approved Real-Time ready Influenza A (H1N1) Detection Set (Roche Biochemical; Cat. No. 05 640 393 001), according to the manufacturer's instructions. This real-time PCR test was positive for influenza A (H1N1) 2009. The patient received treatment with oral oseltamivir at 75 mg twice daily for 5 days. Clinical improvement was noted after 6 days in the hospital. After 10 days of hospitalization, follow-up chest radiography showed resolution of the recently noted pulmonary infiltration.

In December 2009, approximately 5 months after her primary infection with the novel 2009 virus, this same patient presented to the emergency room with a displaced gastrostomy tube. In addition, she had symptoms associated with a respiratory tract infection that were noted by her family members. She had a productive cough that produced white sputum, rhinorhea and tachypnea without a fever. On examination, her body temperature was 37.2 °C, her blood pressure was 91/62 mmHg, her respiratory rate was 22 breaths per minute, and her pulse rate was 97 beats per minute. An oral examination was normal, and the lungs were clear. Chest radiography was not performed. A nasal swab was collected and tested by real-time PCR for influenza virus, and this swab was determined to be positive for influenza A (H1N1) 2009. To confirm the presence of influenza A (H1N1) 2009, pyrosequence analysis was performed. RT-PCR amplifications were performed using the OneStep RT-PCR system (Qiagen, Germany). Primers were used at 20 μM in a standard 25 μl reaction mixture, and the amplification was performed for 45 cycles. Pyrosequencing was performed using Pyro Gold reagents (Qiagen, GmbH, Germany) according to manufacturer's recommendations. The accuracy of the matrix protein 2, neuraminidase, and hemagglutinin gene sequences of influenza A (H1N1) 2009 obtained via pyrosequencing were confirmed by comparison of the results with the sequences that were determined by conventional sequencing. The results from the pyrosequence analysis confirmed the presence of influenza A (H1N1) 2009. The patient received a 5-day course of a standard dose of oral oseltamivir. No respiratory complications were noted during the treatment.

In this report, we describe a case of influenza A (H1N1) 2009 reinfection that occurred in a patient who had no known underlying immunological disease. Although the reinfection rate is extremely low, cases of influenza A (H1N1) 2009 reinfection have recently been reported [4]. A recent report indicated that reinfection occurred in patients who remained in an environmental exposure area and during the grey zone when the immunological response has not had enough time to fully develop. However, for our case, the interval between the primary and secondary infections was approximately 5 months. During the primary infection, our patient developed more a severe infection. She was diagnosed with influenza A (H1N1) 2009 pneumonia, and a high viral load was documented by real-time PCR analysis, which demonstrated an extremely high amplitude for the melting curve (Fig. 1). During the second infection, her illness was milder. The patient only experienced symptoms of an upper respiratory tract infection. Moreover, the real-time PCR analysis also suggested a lower viral load (Fig. 1). We hypothesize that this patient had suboptimal immune protection from the first natural infection. Our pyrosequence analysis showed that there were no changes in the amino acid sequence