tamivir-resistant A (H1N1) infections that were treated with oseltamivir reported a shorter duration of cough; however, no other differences were found. We also compared the proportion of patients who reported fever, cough, sore throat, chills, myalgia or arthralgia, and difficulty breathing. Patients with oseltamivir-resistant A (H1N1) infection that was treated with oseltamivir were less likely to report difficulty breathing. However, because we did not collect information on when symptoms occurred with respect to the initiation of treatment, these results are difficult to interpret.

Although treated persons with oseltamivir-susceptible infection tended to show milder disease and faster recovery than those who were not treated, several differences did not reach statistical significance. These comparisons may have reached statistical significance if we had a larger cohort or if more information were available, including time to resolution of all symptoms. Our study was nonrandomized; the few variables that were statistically different among treated and untreated persons with oseltamivir-resistant infection may be due to unaccountable confounders. In conclusion, our results do not support the use of oseltamivir in patients with oseltamivir-resistant influenza virus infection and are consistent with laboratory antiviral tests. Additional studies may provide further information in correlating laboratory-determined antiviral resistance with clinical response to treatment.

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Nila J. Dharan,1,2 Larisa V. Gubareva,2 Alexander I. Klimov,1 Anthony E. Fiore,2 Joseph S. Bresee,2 and Alicia M. Fry2

1Epidemic Intelligence Service, Office of Workforce and Career Development, and 2Influenza Division, Centers for Disease Control and Prevention, Atlanta, Georgia

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* Present affiliation: Division of Infectious Diseases, New York University School of Medicine, New York (N.J.D.).

Reprints or correspondence: Dr Alicia M. Fry, Influenza Division, Centers for Disease Control and Prevention, 1600 Clifton Rd MS A-20, Atlanta, GA 30333 (afry1@cdc.gov).

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Characteristics Derived from Outbreaks of Pandemic Influenza A (H1N1) 2009 Virus

To the Editor—A novel human influenza A (H1N1) virus variant, pandemic influenza A (H1N1) 2009 virus, was spreading around the world [1]. To date, the disease and comorbidity arising from outbreaks had constituted the major part of the cases of pandemic influenza A (H1N1) in China [2]. In this short report, we summarize the epidemiological, clinical, and viral findings from a total of 6 pandemic influenza outbreaks that occurred in confined settings during the period from July to September 2009. The number of cases (hereafter referred to as outbreak cases) ranged from 43 to 230 for each outbreak, with a total of 538 cases. Of these 538 outbreak cases, 397 (73.8%) had test results that were confirmed in the laboratory [3]. The comparison with sporadic cases disclosed the following findings that characterized the outbreaks:

1. The incubation periods for the outbreak cases seemed to be shorter than those calculated for the sporadic cases. The incubation period differed from 1 to 3 days, with most of the outbreak cases having an incubation period of <2 days (for 4 of the outbreaks). This short incubation period might be the result of the outbreak cases occurring in patients who were in closer contact, compared with what is normally seen in sporadic cases.

2. The clinical manifestations of the outbreak cases were comparable to those of the sporadic cases; however, the duration of fever appeared to be shorter (mean duration, 1.5 days [range, 1–4 days]) among the outbreak cases than that previously reported among patients from the general hospital (mean duration, 2.8 days) [4]. For 1 particular outbreak, we observed an even shorter duration of fever (median duration, 16 h [range, 8–30 h]) among 10 laboratory confirmed cases that occurred in patients who received a seasonal influenza vaccination 1 month prior to infection, compared with other cases from the same outbreak. Human pre- and postvaccination serum samples revealed that neutralizing antibodies against recently circulating human H1N1 viruses do not react with pandemic H1N1 isolates [5]; however, partial immunity resulting from prior exposure to conventional human strains may blunt the impact of pandemic H1N1 viruses in the human population, according to a recent publication [6]. This might explain in part our current findings.

3. Nucleotide sequencing of the full hemagglutinin gene and neuraminidase gene on representative isolates from different generations in 2 outbreaks disclosed ~100% homologousness, thus displaying a stable genetic makeup among strains from different generations in 1 outbreak.

4. Postexposure prophylaxis with oseltamivir administered within 48 h from first exposure proved to be effective for 90 of the 112 patients having had close contacts. When no symptoms were observed thereafter, however, the genetic material of pandemic influenza H1N1 virus was detected in 10 asymptomatic cases occurring in patients with close contacts after 4 days.
of prophylaxis. The control measures for these patients with close contacts should be based on whether or not there was detection of nucleotide acid.

5. From 83 paired serum samples collected from patients from 3 outbreaks, only 65 (78.3%) had a seroconversion of hemagglutination inhibition (HI) antibody against pandemic H1N1 influenza 1 month after patients received a diagnosis of infection. In addition, 13 (25.5%) of 51 individuals who had negative nucleotide acid results presented with detectable HI antibody against pandemic H1N1 during convalescence. This finding indicated the existence of an atypical clinical presentation during the outbreaks. If the criteria for screening of probable cases had been followed, then >20% of cases with H1N1 influenza would not have been diagnosed. Clinicians should consider H1N1 influenza in the differential diagnosis of influenza-like illness, even if diagnostic criteria are not completely met, especially during outbreaks.

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Wei Liu,¹ a Fang Tang,² a Zeng-De Li,² Hong Yang,¹ and Wu-Chun Cao¹

¹State Key Laboratory of Pathogen and Biosafety, Beijing Institute of Microbiology and Epidemiology, and ²Center for Diseases Control and Prevention of Chinese People’s Armed Police Forces, Beijing, China

References


Reprints or correspondence: Dr Wu-Chun Cao, State Key Laboratory of Pathogen and Biosafety, Beijing Institute of Microbiology and Epidemiology, 20 Dong-Da St, Fengtai District, Beijing 100071, China (caowc@nic.bmi.ac.cn).

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