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Fatalities Associated with the 2009 H1N1 Influenza A Virus in New York City

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Background. When the 2009 H1N1 influenza A virus emerged in the United States, epidemiologic and clinical information about severe and fatal cases was limited. We report the first 47 fatal cases of 2009 H1N1 influenza in New York City.

Methods. The New York City Department of Health and Mental Hygiene conducted enhanced surveillance for hospitalizations and deaths associated with 2009 H1N1 influenza A virus. We collected basic demographic and clinical information for all patients who died and compared abstracted data from medical records for a sample of hospitalized patients who died and hospitalized patients who survived.

Results. From 24 April through 1 July 2009, 47 confirmed fatal cases of 2009 H1N1 influenza were reported to the New York City Department of Health and Mental Hygiene. Most decedents (60%) were ages 18–49 years, and only 4% were aged ⩾65 years. Many (79%) had underlying risk conditions for severe seasonal influenza, and 58% were obese according to their body mass index. Thirteen (28%) had evidence of invasive bacterial coinfection. Approximately 50% of the decedents had developed acute respiratory distress syndrome. Among all hospitalized patients, decedents had presented for hospitalization later (median, 3 vs 2 days after illness onset; $P<.05$) and received oseltamivir later (median, 6.5 vs 3 days; $P<.01$) than surviving patients. Hospitalized patients who died were less likely to have received oseltamivir within 2 days of hospitalization than hospitalized patients who survived (61% vs 96%; $P<.01$).

Conclusions. With community-wide transmission of 2009 H1N1 influenza A virus, timely medical care and antiviral therapy should be considered for patients with severe influenza-like illness or with underlying risk conditions for complications from influenza.

Before the emergence of the 2009 H1N1 influenza A virus, descriptions of clinical illness due to human infection with swine-origin influenza viruses had been limited to sporadic cases and reports from a 1976 military fort outbreak [1, 2]. As the 2009 H1N1 influenza outbreak evolved in North America, illness due to infection with the virus was described as mostly mild, although with a clinical spectrum comparable to seasonal influenza [3, 4]. With seasonal influenza, risks for complications and fatal outcome are highest for young children, adults aged ⩾65 years, and individuals with certain chronic medical conditions, such as asthma [5, 6]. Information on risk factors for severe illness and death from infection with 2009 H1N1 influenza has been sparse until recently, with reports describing severe and fatal illness now emerging [3, 4, 7–13].

In April 2009, an outbreak of 2009 H1N1 influenza at a New York City high school signaled the spread of the virus beyond Texas and California, the 2 states in the country with confirmed cases at the time [14]. By early July, the number of deaths among New York City residents with 2009 H1N1 influenza represented a large proportion of the total number of deaths reported nationwide [15]. We describe the epidemiologic and clinical characteristics of the first 47 individuals in New York City who died after infection with 2009 H1N1 influenza.

METHODS

Case definition. A fatal case was defined as death in a New York City resident with a clinically compatible illness and confirmed 2009 H1N1 influenza A virus infection; there could be no period of recovery to baseline health status before death and no other recognized alternate cause of death. Laboratory confirmation required testing by real-time reverse-transcriptase polymerase chain reaction [16].

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Surveillance. Enhanced surveillance for hospitalizations and deaths associated with 2009 H1N1 influenza occurred from 24 April 2009, when the outbreak was first recognized in New York City, to 8 July 2009; this report includes fatal cases reported from 24 April to 1 July 2009. The New York City Health Code mandates that health care professionals report cases of influenza caused by a novel strain with pandemic potential and that laboratories report electronically all positive influenza test results. Before the 2009 H1N1 influenza A virus emerged, influenza-related deaths in New York City were reportable to the Department of Health and Mental Hygiene (DOHMH) only if they occurred in children aged <18 years. From 24 April to 8 July 2009, the DOHMH requested reporting of all hospitalized patients diagnosed as having influenza A and all critically ill patients with a febrile respiratory illness. For these patients, the DOHMH attempted to retrieve and submit diagnostic specimens for confirmation. The DOHMH also expanded reporting of influenza-related deaths to include all deaths among individuals with confirmed 2009 H1N1 influenza, regardless of age, and all unexplained deaths involving a febrile respiratory illness. Health care professionals informed the New York City Office of Chief Medical Examiner (OCME) and notified the DOHMH of fatalities. Medical examiners collected postmortem nasopharyngeal swabs, performed autopsies, and reported to the DOHMH cases under investigation for 2009 H1N1 influenza, including individuals with a history of a febrile respiratory illness who died suddenly outside a medically attended setting.

To ascertain fatal cases not identified through reporting, confirmed 2009 H1N1 influenza cases were matched with the death certificate registry maintained by the New York City Office of Vital Statistics. The DOHMH added a prompt to the electronic system for registering death certificates in New York City, reminding health care professionals to include influenza if suspected or confirmed as a cause of death. To further enhance case ascertainment, the Office of Vital Statistics regularly searched the death certificate registry for decedents with "influenza" listed as a cause of death.

Clinical investigation. The DOHMH staff completed an initial triage data collection form for reported cases of hospitalized patients with influenza A and critically ill patients with a febrile respiratory illness. For fatal cases, the DOHMH interviewed clinicians and hospital infection control practitioners using a standardized case report form, which collected information about demographic characteristics, underlying conditions, clinical presentation, and laboratory findings. For 31 fatalities among patients who presented to a hospital and for the first 95 hospitalized patients with confirmed 2009 H1N1 influenza, admitted 11–25 May 2009, staff conducted medical record reviews using a standardized abstraction form, which collected additional information on medical history and clinical course.

Thirty-one decedents underwent autopsy; the DOHMH staff discussed preliminary findings with the OCME and reviewed reports from the Centers for Disease Control and Prevention's Infectious Diseases Pathology Branch, which evaluated pulmonary specimens sent by the OCME using immunohistochemical analysis and polymerase chain reaction [17]. If no clinical information was available, next of kin was contacted. Death certificates were reviewed for patient demographic characteristics and cause of death.

Variable definition. Underlying conditions recognized as increasing the risk for severe seasonal influenza, such as asthma and diabetes mellitus, were characterized as risk conditions; data for other chronic medical conditions are presented separately [5, 6]. A chronic medical condition detected after death, such as atherosclerotic cardiovascular disease, was considered a risk condition even if it had not been diagnosed before death. Data on obesity were collected from hospital records and medical examiners. Overweight was defined as a body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) of 25–29.9, obese as a BMI of 30–39.9, and extreme obesity as a BMI $\geq$40 [18]. Invasive bacterial coinfection was defined as isolation of bacteria from a normally sterile site or molecular detection of pathogenic bacteria in postmortem lung tissue.

Statistical analysis. We described all fatal cases and compared the demographic characteristics of the decedents with 2007 New York City Census estimates to determine whether certain demographic groups were overrepresented among fatalities [19]. The BMI was calculated for all patients except children aged <2 years and pregnant women (because antepartum weight was unavailable). For vital signs and laboratory tests, reference values from standard texts were used [20, 21]. We compared clinical and treatment characteristics for hospitalized patients who died and hospitalized patients who did not die using case report form and medical record abstraction data, except where otherwise noted. For these comparisons, no diagnoses identified only after death were included. We used SUDAAN (RTI International) to calculate marginal estimates for adjusted probabilities and SAS software, version 9.1 (SAS Institute), for all other analyses. To identify significant differences, we used the Fisher exact test for categorical variables and the Kruskal-Wallis test for continuous variables.

RESULTS

Overview. During the surveillance period, the DOHMH received reports of 47 fatal cases. Demographic data of the decedents are presented in Table 1. Two of the decedents were aged <6 months. More than one-half (28 [60%]) of the decedents were aged 18–49 years, and 30% were aged 50–64 years; these age categories represent 48% and 17%, respectively, of the general New York City population [19]. Although considered at risk for complications and death from seasonal influ-

Table 1. Characteristics of 47 Patients Who Died of 2009 H1N1 Influenza in New York City, 24 April to 1 July 2009

Characteristic	No. (%) of patients	No. (%) of New York City population
Age group, years[a]		
0–4	2 (4.3)	565,649 (6.8)
5–17	1 (2.1)	1,330,691 (16.1)
18–49	28 (59.6)	3,979,785 (48.1)
50–64	14 (29.8)	1,385,357 (16.7)
⩾65	2 (4.3)	1,013,045 (12.2)
Total	47	8,274,527
Male sex	22 (46.8)	3,949,043 (47.7)
Race/ethnicity		
White	9 (19.1)	2,928,832 (35.4)
Black or African American	17 (36.2)	1,979,191 (23.9)
Asian	5 (10.6)	971,412 (11.7)
Hispanic	14 (29.8)	2,269,971 (27.4)
Not specified or other	2 (4.3)	125,121 (1.5)

[a] The median age of the patients was 43 years (range, 7 weeks to 82 years).

enza, only 4% of the decedents were aged ⩾65 years, in contrast to 12% of New York City residents [22]. At least 36% of all the decedents were African American, compared with 24% of the general New York City population [19].

Most patients (37 [77%]) had disease onset in the 3 weeks from 18 May to 7 June 2009, with the peak in the number of fatal cases occurring 2 weeks after the peak in illness onset (Figure 1). Median time from illness onset to death was 9 days (range, 1–44 days). Eight individuals (17%) died at home, 11 (23%) died ⩽24 h after arriving at a hospital, and 28 (60%) died after >24 h in a hospital. Decedents who died at home or ⩽24 h after arrival at a hospital did not differ significantly from decedents who died after >24 h in a hospital when comparing age, race, socioeconomic status, or timing from illness onset to initial clinical encounter.

Underlying conditions. Among the decedents, 37 (79%) had underlying conditions associated with increased risk of influenza complications, including age; of the remaining 10 patients with no risk conditions, 8 (17%) were obese (Table 2). Chronic pulmonary disease and diabetes were most common (32% each). Of the 9 patients with asthma for whom medical record review data were available, 4 reported taking inhaled corticosteroids at home and 7 were treated with bronchodilators during their hospitalization. One patient characterized as immunosuppressed received an infusion of infliximab, an antagonist of tumor necrosis factor, for severe psoriasis; the patient developed difficulty breathing 7 days later, did not seek medical care for the respiratory symptoms, and died at home the next day.

More than half (25 [58%]) of the decedents were obese or extremely obese, and an additional 7 (16%) were overweight, compared with 22% obesity and 35% overweight among the New York City general population (Table 2) [23]. Twelve (80%) of the extremely obese and 5 (50%) of the obese decedents had underlying risk conditions; 8 (19%) of all obese decedents had no risk condition. Among the 11 decedents of normal weight, 9 (82%) had risk conditions.

Symptoms. The most common presenting symptoms among the decedents were subjective fever (83%), cough (77%), shortness of breath (70%), and fatigue and weakness (43%). Fever was reported by all hospitalized decedents. Vomiting and diarrhea were reported by 30% and 17% of all decedents, respectively.

Radiologic and laboratory findings. Among the 28 decedents with radiologic results, 75% had abnormalities, most commonly multilobar infiltrates (38%). Most decedents (39 [83%]) had antemortem diagnostic testing for influenza A; 11 (35%) of 31 decedents tested by enzyme immunoassay had

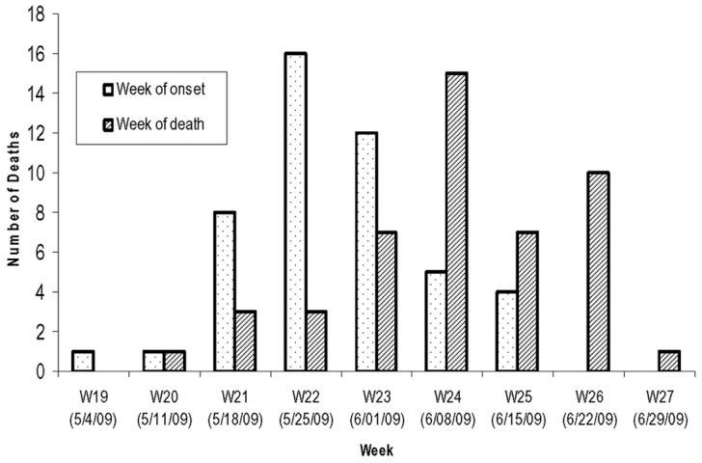

Figure 1. Week of onset and death for 47 patients who died of 2009 H1N1 influenza in New York City, 24 April through 1 July 2009.

Table 2. Underlying Medical Conditions of 47 Patients Who Died of 2009 H1N1 Influenza, New York City, 24 April to 1 July 2009

Medical condition	No. (%) of patients
Underlying risk condition[a]	
Chronic pulmonary disease[a]	15 (31.9)
Asthma	10 (21.3)
Chronic obstructive pulmonary disease	8 (17.0)
Chronic metabolic disorder	18 (38.3)
Diabetes mellitus	15 (31.9)
Chronic cardiovascular disease (excluding hypertension)	14 (29.8)
Immunosuppressive condition[a]	13 (27.7)
Human immunodeficiency virus infection	7 (14.9)
Cancer	4 (8.5)
Immune-suppressing medication	5 (10.6)
Renal disease	5 (10.6)
Chronic liver disease (eg, hepatitis C)	4 (8.5)
Neuromuscular disorders	3 (6.4)
Cognitive dysfunction	2 (4.3)
Hemoglobinopathy (eg, sickle cell disease)	2 (4.3)
Pregnancy	2 (4.3)
Age <2 years	2 (4.3)
Age ⩾65 years	2 (4.3)
Multiple underlying risk conditions	27 (57.4)
No underlying risk condition	10 (21.3)
Other diagnoses	
Psychiatric diagnoses	6 (12.8)
Current smoker	13 (27.7)
Substance abuse	7 (14.9)
Weight by BMI[b]	
Normal weight or underweight (BMI, <25)	11 (25.6)
Overweight (BMI, 25–29.9)	7 (16.3)
Obese or extremely obese (BMI, ⩾30)	25 (58.1)
Obese (BMI, 30–39.9)	10 (23.3)
Extremely obese (BMI, ⩾40)	15 (34.9)

NOTE. BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters).

[a] Underlying conditions are not mutually exclusive.

[b] BMI was not calculated for infants and pregnant women ($n = 43$).

positive results, 2 (25%) of 8 tested by direct fluorescent antibody had positive results, and 2 (40%) of 5 tested by viral culture had positive results.

Thirteen decedents (28%) had evidence of invasive bacterial coinfection; 7 coinfections were identified only after death by immunohistochemical analysis or polymerase chain reaction testing of lung and bronchial tissue [17]. *Streptococcus pneumoniae* was most commonly identified (8 patients [17%]), followed by *Streptococcus pyogenes* (3 patients [6%]); 1 pediatric case patient had postmortem evidence of both bacteria. *S. pneumoniae* was identified after death in an infant who reportedly had had an upper respiratory tract infection, was found unresponsive at home, and had a failed resuscitation attempt.

Medical care. All 28 decedents who died after >24 h in the hospital were admitted to the intensive care unit, and all but 1 received mechanical ventilation. An additional 9 case patients (19%) were intubated emergently and died in the emergency department. Almost one-half (49%) of all decedents developed acute respiratory distress syndrome, and among 31 decedents with medical record review data, 14 (45%) developed renal failure, with 8 (27%) requiring dialysis (Table 3).

For decedents who sought medical care, median time to initial clinical encounter and also to hospitalization was 3 days (range, 0–12 and 0–8, respectively) (Table 4). Decedents with risk conditions presented earlier for hospitalization than decedents without risk conditions (median, 3 vs 5.5 days; $P <$ .50). Three decedents who died outside a hospital never sought medical care for influenza-like illness (ILI) and were diagnosed after death as having influenza. There were 2 nosocomial cases; a case patient with multiple myeloma developed ILI 4 days after hospitalization for a bone marrow transplantation, whereas another case patient with diabetes and obesity developed ILI after more than a month in the hospital for treatment of osteomyelitis.

Thirty-two decedents (68%) were treated with oseltamivir. Median time from symptom onset to oseltamivir therapy was 5.5 days (range, 0–17 days); no significant difference was observed between those with and without risk conditions. Oseltamivir was administered at 75 mg twice daily for decedents, with the exception of 1 case patient with human immunodeficiency virus and 1 postpartum case patient, both of whom received 150 mg twice daily. Among the 15 decedents who did

Table 3. Indicators of Illness Severity from Medical Record Reviews for 31 Patients Who Died of 2009 H1N1 Influenza, New York City, 24 April to 1 July 2009

Indicator of severity	No. (%) of patients
Mechanical ventilation	25 (80.6)
Acute respiratory distress syndrome[a]	23 (48.9)
Vasopressor medications	21 (67.7)
Renal failure	14 (45.2)
Dialysis	8 (25.8)
Liver impairment	12 (38.7)
Sepsis	12 (38.7)
Shock	11 (35.5)
Myocardial dysfunction	9 (29.0)
Myocardial infarct	2 (6.5)
Disseminated intravascular coagulopathy	1 (3.2)
Encephalitis or encephalopathy	0 (0.0)
Other	5 (16.1)

NOTE. The length of the intensive care unit stay was as follows: mean, 3.7 days; median, 2 days; and range, 0–16 days.

[a] Data available for all 47 patients.

Table 4. Time to Medical Care and Antiviral Therapy for Hospitalized Patients with Fatal and Nonfatal Cases of 2009 H1N1 Influenza, New York City, 24 April to 1 July 2009

Time to treatment	Hospitalized patients, median (range)		
	Fatal cases (n = 28)[a]	Nonfatal cases (n = 95)	P[b]
Time from illness onset to initial clinical encounter, days	3 (0–8)	2 (0–14)	.06
Time from illness onset to hospitalization, days	3 (0–8)	2 (0–14)	.03
Time from illness onset to oseltamivir administration, days	6.5 (0–17)	3 (0–15)	<.01
Time from hospitalization to oseltamivir administration, days	1.5 (0–12)	0 (0–10)	<.01

[a] Fatal cases are limited to patients who were in the hospital for >24 h.
[b] Determined by Kruskal-Wallis test.

not receive antiviral therapy, 1 was hospitalized for ILI for >24 h, whereas 9 died ⩽24 h after arrival at a hospital.

Comparison of hospitalized patients who died and survived. Comparing the 28 hospitalized patients who died with the 95 hospitalized patients who survived, decedents had significantly higher proportions of diabetes (43% vs 11%), cardiovascular disease (25% vs 4%), and immunosuppression (29% vs 3%; P<.05 for each). Obesity was not significantly more common among the patients who died versus the patients who survived (54% vs 33%; P = .09). Decedents reported shortness of breath (75% vs 32%; P<.01) and fatigue or weakness (57% vs 25%; P<.01) more often on presentation.

Comparing laboratory abnormalities, hospitalized patients who died were more likely to have an elevated hematocrit (56% vs 19%), thrombocytopenia (33% vs 9%), an elevated serum creatinine level (50% vs 11%), an elevated level of transaminases (61% vs 18%), and an elevated creatine kinase level (39% vs 4%) in univariate analysis (P<.05 for each). After controlling for age, sex, and other laboratory tests, an elevated creatinine level remained more common among hospitalized patients who died than hospitalized patients who survived (47% vs 6%; P <.05).

Decedents presented for hospitalization later than survivors (median, 3 vs 2 days; P<.05). Hospitalized patients who died also received oseltamivir later than hospitalized patients who survived (median, 6.5 vs 3 days; P<.01). Among case patients receiving oseltamivir, decedents were less likely than survivors to receive oseltamivir within 2 days of illness onset (15% vs 49%; P<.01). Similarly, decedents were less likely than survivors to receive oseltamivir ⩽2 days after hospitalization (61% vs 96%; P<.01).

DISCUSSION

We describe a series of fatalities during the spring outbreak of 2009 H1N1 influenza in New York City. Unlike seasonal influenza, deaths occurred predominately among individuals 18–64 years old and were less common among elderly individuals; our data support other findings about the age distribution of fatal 2009 H1N1 influenza cases [13, 22, 24]. It is possible that older persons have some immunity to 2009 H1N1 influenza because of preexisting cross-reactive antibodies [25]. In our case series, most decedents had comorbidities recognized as risk conditions for severe influenza.

Obesity was common and, for ~20% of decedents, the only known chronic medical condition. We did not, however, observe a significant difference in obesity among hospitalized patients who died and survived. Recent reports have described high proportions of severe or fatal 2009 H1N1 influenza cases among obese patients [7–9]. Although not recognized to increase risk for severe seasonal influenza, obesity is associated with other conditions, such as diabetes, that are risk factors for complications or fatal outcomes. Although obesity has been linked to higher all-cause mortality in large epidemiologic studies [26, 27], some studies in critical care settings have demonstrated no correlation between BMI and fatal outcome [28]. The pathophysiology of severe 2009 H1N1 influenza in obese individuals is unknown, and further research is needed to elucidate the role of obesity in influenza mortality.

Some decedents described in this series were not diagnosed before death as having influenza, despite having a medical encounter before death. Health care professionals should consider influenza, particularly in the context of a community outbreak, for patients with a clinically compatible respiratory illness. Of note, 17% of decedents did not present with fever, suggesting the need to consider influenza based on a constellation of symptoms that may not include fever. As noted in other published reports, more than one-third of decedents in our series reported vomiting and/or diarrhea—symptoms not commonly associated with seasonal influenza [3, 7]. In the presence of community-wide influenza transmission and given the limited sensitivity of rapid diagnostic tests, a negative enzyme immunoassay, direct fluorescent antibody, or viral culture result should not discourage clinicians from empirically treating a patient with ILI, when risk factors indicate [5, 6, 29]. One case patient hospitalized for respiratory distress had a negative influenza result by enzyme immunoassay, did not have confirmatory testing before death, and received no antiviral medication. The

patient developed acute respiratory distress syndrome and died 4 days after admission.

Decedents presented for hospitalization later than survivors. Similar to findings from a national sample of hospitalized patients with 2009 H1N1 influenza, decedents in New York City were less likely to receive oseltamivir within the recommended 48 h of illness onset than survivors [7]. As with seasonal influenza, timely medical care and antiviral therapy are critical for treatment of severe illness with 2009 H1N1 influenza or illness among individuals with a known risk condition.

Bacterial coinfections were common among the case patients who died of 2009 H1N1 influenza in New York City. This finding supports our understanding of the role of bacterial pneumonia in past pandemics and underscores the importance of concurrent antibiotic treatment for secondary bacterial pneumonia in patients with severe influenza [30, 31]. Because more than one-half of the invasive bacterial coinfections in this series were detected after death, empirical antibiotic treatment should be considered for patients with worsening influenza. The finding of pneumococcal coinfection in 17% of decedents highlights the potential for pneumococcal vaccination to modify influenza morbidity and mortality. Many of the risk conditions for severe seasonal influenza are indications for seasonal influenza vaccination and vaccination with either the 23-valent polysaccharide or the 7-valent conjugate pneumococcal vaccine [32, 33].

Postmortem examinations revealed previously undiagnosed acute coinfections and chronic comorbid conditions among patients who died of 2009 H1N1 influenza and have contributed to our overall understanding of this newly emerged pathogen. Collaboration with the OCME also enhanced DOHMH surveillance because medical examiners identified cases that were not or could not be reported because of death occurring outside medically attended settings. To understand the epidemiology of and clinical spectrum due to this influenza strain, we adopted a surveillance approach for fatal cases that was more comprehensive than during the usual influenza season. Consequently, we cannot compare data from the outbreak with data from previous influenza seasons in New York City. In the event of a pandemic with a higher case-fatality rate, maintaining such an intensive surveillance approach is likely unfeasible.

Limitations of our investigation include the possibility of missed fatal cases of 2009 H1N1 influenza among individuals who were afebrile, who did not meet laboratory criteria for testing, or who were not reported. Also, our convenience sample of the first 95 hospitalized patients who survived may not represent the full 2009 H1N1 influenza clinical spectrum, and comparisons between these cases and the universe of known fatal cases should be interpreted with caution. Last, the relationship we describe between timely antiviral administration and fatal outcome is limited by the observational nature of this study.

Although differences were noted between hospitalized patients with 2009 H1N1 influenza who died and those who survived, additional studies are needed to characterize risk factors for fatal outcome. Data collected from New York City and the rest of the United States were used to inform the Advisory Committee on Immunization Practices and its 2009 H1N1 influenza vaccination strategy for 2009–2010 [34]. The recommendations prioritized vaccination for individuals ages 6 months to 24 years and for individuals 25–64 years of age with underlying conditions, reflecting the epidemiology of severe and fatal cases. When indicated, timely medical care, antiviral medication, antibiotic treatment, and vaccination are critical for controlling morbidity and mortality associated with 2009 H1N1 influenza.

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