CONCISE COMMUNICATION

Nosocomial Influenza in a Pediatric Hospital: Comparison of Rates of Seasonal and Pandemic 2009 Influenza A/H1N1 Infection

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The rates of nosocomial seasonal (January 2008 to March 2009) and 2009 A/H1N1 (April 2009 to December 2010) influenza infections in a children's hospital were compared. Droplet precautions were used. The rates were similar during both periods, suggesting that use of droplet precautions did not result in a higher rate of influenza A/H1N1 infection.

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Nosocomial influenza infection may result in significant morbidity and mortality in pediatric patients.[1] During the epidemic of the 2009 influenza A/H1N1 infection and a highly susceptible population, there was a possibility of large numbers of nosocomial cases. Herein, we report and compare the incidence of nosocomial 2009 A/H1N1 and nosocomial seasonal influenza infection in a children's hospital where droplet precautions were the primary strategy for prevention of transmission.

PATIENTS AND METHODS

We performed a retrospective review of inpatients with community-acquired and nosocomial influenza infection at the Steven and Alexandra Cohen Children's Medical Center of New York (formerly Schneider Children's Hospital) of the North Shore-LIJ Health System, a hospital with 154 inpatient beds. All inpatients were included except those admitted to the neonatal intensive care unit and newborn nursery. Other than the pediatric intensive care unit and the stem cell transplant unit that consist of single bedded rooms, most rooms had 2 beds. In addition, there were 5 rooms with 4 beds and 14 single-bed isolation rooms, 10 with negative pressure ventilation.

Cases were identified using a medical records search and data from the Infection Control Division, derived from virology laboratory reports. A nosocomial case was defined as a patient with fever and/or cough and a positive influenza test performed at least 48 hours after admission who did not have fever, cough, sore throat, or rhinorrhea within the first 48 hours of hospitalization. Diagnostic modalities for influenza testing varied. During 2008, patients were diagnosed with influenza infection using a rapid antigen detection test (Becton Dickinson, Directigen A+B, BD and Co) or by direct fluorescent antibody testing (using D3 Respiratory Reagents, Diagnostic Hybrids), or viral culture. In March 2009 the rapid antigen detection test was changed to the 3M Rapid Detection Flu A+B Test (3MA+B; 3M Medical Diagnostics), and all specimens were tested using polymerase chain reaction assay (Luminex Respiratory Viral Panel, Luminex Molecular Diagnostics). Patients who tested positive for influenza A via a rapid antigen detection test prior to April 1, 2009, were classified as seasonal influenza infection cases.[2] Although rapid antigen tests for influenza generally exhibit high specificity, at our center the rapid antigen test for influenza B exhibited poor specificity (unpublished observations), and influenza B cases identified solely via a rapid antigen detection test were excluded.[2]

inpatients were divided into 2 cohorts: prepandemic (admitted to hospital from January 1, 2008, through March 31, 2009) and pandemic (admitted to hospital from April 1, 2009, through December 31, 2010). Comparisons of nosocomial influenza infection rates were performed by using the incidence density ratio method with inpatient days as a measure for exposure.[3] Inpatient days at risk were calculated as the length of stay in excess of 2 days, because patients could not be diagnosed with nosocomial influenza during the first 2 days of their stay. Data on individual length of stay for inpatients with influenza were not available, but length of stay was estimated to be a median of 3 days on the basis of a previous study in this population.[2] Therefore, a second comparison of rates was carried out by removing 1 day of stay for each inpatient with influenza, since these patients were not at risk of being diagnosed with nosocomial influenza.

The association between source of infection (nosocomial, community acquired) and period was compared using the Fisher's exact test. The study was approved by the Institutional Review Board of the North Shore-LIJ Health System.

RESULTS

The difference in rates of nosocomial infection between the prepandemic and pandemic cohorts was not statistically significant ($P = .91$; Table 1). The results of a secondary analysis removing 1 additional day of stay for each influenza patient from the patient-days at risk (3 day median duration of hospitalization minus 2 days not at risk) did not differ qualitatively from the initial analysis (not shown).

A summary of the number of inpatients with influenza infection and the type and subtype of strains of influenza in

TABLE 1. Rates of Nosocomial Influenza Infection: Comparison between the Prepandemic and Pandemic Cohorts

Period	No. of patient-days[a]	No. of cases[b]	Rate of infection[c]	*P*
Prepandemic	23,943	4	0.167	.912
Pandemic	27,951	5	0.179	

[a] Patient-days at risk of nosocomial influenza infection.
[b] Cases of nosocomial influenza infection.
[c] Rate of nosocomial influenza infection per 1000 patient-days.

TABLE 2. Hospitalized Patients with Influenza Infection: Nosocomial Cases and Comparison between the Prepandemic and Pandemic Cohorts

	Prepandemic period			Pandemic period		
Influenza infection cases	Community	Nosocomial	Total	Community	Nosocomial	Total
All	50	4 (7.4%)[a]	54	198	5 (2.4%)[a]	203
Influenza A, seasonal	42	2	44	6	2	8
Influenza 2009 A/H1N1	0	0	0	190	2	192
Influenza B	8	2	10	2	1	3

[a] $P = .095$, Fisher's exact test.

each cohort (Table 2) shows that during the pandemic period, 95% of cases were caused by 2009 A/H1N1 strains. During the pandemic period, the number of hospital admissions with influenza increased, but the proportion of influenza-infected inpatients with nosocomial influenza infection was lower than that during the prepandemic period. However, the difference was not significant ($P = .09$). As described earlier, cases of influenza B that were diagnosed solely using a rapid antigen test were excluded from analysis. However, when we repeated the analyses including these cases, the differences in rates still were not significant.

DISCUSSION

The proportion of pediatric inpatients with seasonal influenza that were nosocomial cases, 7.4%, was within the range (3%–15%) reported in other studies.[1,4,5] During the pandemic influenza period, we experienced a higher volume of inpatients with influenza, but nosocomial cases occurred at a lower rate and comprised a smaller proportion of cases (2.4%) than seasonal influenza infections during the prepandemic period. Others have reported a higher proportion of nosocomial cases and a higher nosocomial rate.[6,7]

During both periods, our infection control precautions for patients with suspected or proven influenza infection included droplet precautions rather than use of a N95 or equivalent respirator and airborne precautions. Using these precautions, our rate and proportion of nosocomial influenza infection cases were similar or lower during the pandemic period despite an increase in the number of influenza-infected patients. Although our numbers of nosocomial cases were low, these findings suggest that the use of droplet precautions did not result in a higher transmission rate of 2009 A/H1N1 to patients. However, it is plausible that because of heightened awareness of the general public and healthcare personnel during the pandemic, there was an increased adherence to infection control precautions that contributed to a lower rate of nosocomial transmission. Furthermore, the greatest potential impact on mask type is on transmission to healthcare personnel, and we do not have data on transmission to healthcare personnel.[8]

There are limitations to our study. During the course of the study period, the laboratory diagnostic methods were changed to a more sensitive nucleic acid amplification-based assay, possibly resulting in detection of more influenza cases during the pandemic period. Our case definition of nosocomial influenza may have included cases of community-acquired infection that had an incubation period of longer than 2 days. Inpatients with influenza infection were not the only source of nosocomial influenza infection; visitors and healthcare personnel were also potential sources. In this study and in most of the studies on nosocomial influenza infection, the impact of these potential sources of nosocomial influenza infection was not evaluated. It is plausible that during the risk period when the 2009 A/H1N1 strain was highly prevalent in our community, inpatients were at higher risk from this source of virus than during periods when seasonal influenza was prevalent. However, comparison between the prepandemic and pandemic periods did not show a significant difference in rates of nosocomial cases. It is also plausible that adherence to infection control practices differed during the prepandemic and pandemic periods; we were unable to assess adherence to infection control practices in this retrospective study. Finally, we may have missed nosocomial cases that manifested clinically following hospital discharge, because our study design did not include a postdischarge review.

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REFERENCES

1. Slinger R, Dennis P. Nosocomial influenza at a Canadian pediatric hospital from 1995 to 1999: opportunities for prevention. *Infect Control Hosp Epidemiol* 2002;23:627–629.
2. Ostovar G, Rubin L. Comparison of the clinical features of children hospitalized with pandemic 2009 A:H1N1 and seasonal influenza. *Clin Pediatr* 2011;50:348–354.
3. Kleinbaum G, Kupper L, Morgenstern H. *Epidemiologic Research: Principles and Quantitative Methods.* 1st ed. New York: Wiley, 1982:117–140.
4. Evans M, Hall K. Influenza control in acute care hospitals. *Am J Infect Control* 1997;25:357–362.
5. Serwint J, Miller R. Why diagnose influenza infections in hospitalized pediatric patients? *Pediatr Infect Dis J* 1993;12:200–204.
6. Fanella S, Pinto M. Pandemic (H1N1) 2009 influenza in hospitalized children in Manitoba: nosocomial transmission and lessons learned from the first wave. *Infect Control Hosp Epidemiol* 2011;32(5):435–443.
7. Enstone J, Myles P. Nosocomial pandemic (H1N1) 2009, United Kingdom, 2009–2010. *Emerg Infect Dis* 2011;17(4):592–597.
8. Gralton J, McLaws M. Protecting healthcare workers from pandemic influenza: N95 or surgical masks? *Crit Care Med* 2010;38: 657–667.