DOI:10.1111/irv.12016
www.influenzajournal.com

Short Article

Seroprevalence of 2009 H1N1 Virus Infection and Self-Reported Infection Control Practices Among Healthcare Professionals Following the First Outbreak in Bangkok, Thailand

Kulkanya Chokephaibulkit,[a] Susan Assanasen,[b]Anucha Apisarnthanarak,[c] Yong Rongrungruang,[b] Kanchana Kachintorn,[d] Yuwadee Tuntiwattanapibul,[d] Tepnimitr Judaeng,[d] Pilaipan Puthavathana[e]

[a]Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. [b]Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. [c]Division of Infectious Diseases, Faculty of Medicine, Thammasat University Hospital, Bangkok, Thailand. [d]Division of Center for Nosocomial Infection Control, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. [e]Department of Microbiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand
Correspondence: Kulkanya Chokephaibulkit, Division of Infectious Diseases, Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand.
E-mail: kulkanya.cho@mahidol.ac.th

Accepted 10 August 2012. Published online 8 October 2012.

A serologic study with simultaneous self-administered questionnaire regarding infection control (IC) practices and other risks of influenza A (H1N1) pdm09 (2009 H1N1) infection was performed approximately 1 month after the first outbreak among frontline healthcare professionals (HCPs). Of 256 HCPs, 33 (13%) were infected. Self-reported adherence to IC practices in >90% of exposure events was 82·1%, 73·8%, and 53·5% for use of hand hygiene, masks, and gloves, respectively. Visiting crowded public places during the outbreak was associated with acquiring infection (OR 3·1, $P = 0·019$). Amongst nurses, exposure to HCPs with influenza-like illness during the outbreak without wearing a mask was the only identified risk factor for infection (OR = 2·3, $P = 0·039$).

Keywords 2009 H1N1 pandemic, Thailand, healthcare workers, seroprevalence, hemagglutination assay

Please cite this paper as: Chokephaibulkit *et al.* (2013) Seroprevalence of 2009 H1N1 Virus Infection and Self-Reported Infection Control Practices Among Healthcare Professionals Following the First Outbreak in Bangkok, Thailand. Influenza and Other Respiratory Viruses 7(3), 359–363.

Introduction

The outbreak of influenza A (H1N1) pdm09 (2009 H1N1) pandemic in Thailand started in early June, peaked around July to August, and waned in September.[1] Healthcare professionals (HCPs) are at risk of exposure to infection from the community as well as in the workplace. However, they also have the greatest access to personal protective equipment (PPE) and are likely to receive the annual seasonal vaccine that may provide some protection against 2009 H1N1 infection.[2] The 2009 H1N1outbreak was an opportunity to evaluate the effectiveness of infection control (IC) practices in HCPs.

We conducted a serologic study of 2009 H1N1infection among frontline HCPs who were involved in the care of 2009 H1N1 patients during the first outbreak in two large public hospitals in Bangkok, Thailand. The aim of the study was to understand the magnitude of acquisition of infections among HCPs in relation to self-reported IC practices and the factors associated with infection during the outbreak. This information may be useful for future outbreak control and vaccination strategies.

Methods

HCPs who worked during the peak of the 2009 H1N1 outbreak (June–August, 2009) on the wards that cared for patients with influenza and at emergency rooms of Siriraj Hospital and Thammasat Hospital, two large public tertiary care centers in Bangkok, were randomly invited to participate in the study. These two hospitals served adults and children patients. The wards at which the HCPs in this study were working were 3 ERs, four pediatric wards, three adult wards, and three intensive care units. Immunocompromised patients were being cared for in some of the wards participating the study. Approximately, a third of HCPs in each of these wards were invited and 97% accepted. The study was conducted from October 1 to 19,

2009, approximately 1 month after the end of the outbreak. An anonymous self-administered questionnaire was administered prior to a single blood draw for assessment of hemagglutination inhibition (HI) titer. The questionnaire consisted of demographic information, medical history, and factors that may be associated with community-acquired and occupational-acquired influenza. Adherence to hand hygiene (alcohol-based hand rub and/or hand washing with water) and using mask and gloves when in contact (defined as having activities or procedures that potentially resulted in contact or droplet transmission) with patients with suspected 2009 H1N1 were categorized as: every time or >90–100%, mostly or 70–90%, and <60%, respectively. The wards were classified as isolation wards (1–three patients in a room, PPE practice for airborne and contact precautions when entering the patients' room), semi-open ward (share up to 12 patients in a room, PPE practice for airborne and contact precaution when entering patients' area), and open ward or emergency room (large ward hold up to 24 beds or walk-in patients, PPE practice for contact and droplet precaution as needed). There was no verification of the accuracy of responses in the questionnaire.

HI assay was performed using the protocol previously described.[3,4] We defined an HI titer ≥40 as seropositive and a marker of acquiring recent infection assuming that none of the HCPs had been infected with the 2009 H1N1 virus prior to the outbreak and that pre-existing HI antibody to 2009 H1N1 was uncommon.

Statistical analysis

Descriptive analysis was performed on the demographic and other variables associated with risk of influenza infection. Univariate analysis was performed using a binomial test. Multiple logistic regression analysis was used for multivariate analysis of self-reported factors including the IC practices associated with an HI titer ≥40. Stata (Version 9.2, College Station, TX, USA) was used for the data analysis.

Results

There were 256 HCPs, 93% female, who participated in this study. The median (range) age was 34 (20–61) years. The majority (81·3%) were nurses and nurse assistants. Of the 198 HCPs working in specific wards, 72 (36·4%) were working in isolation wards, 57 (28·8%) in semi-open wards, and 69 (34·8%) in open wards or emergency rooms. The majority (82%) had received seasonal influenza vaccines (Southern Hemisphere strain 2008) between April 2008 and May 2009, at least 1–2 months before the outbreak started. Adherence to IC practices in >90% of the exposure events when in contact with patients with suspected 2009 H1N1 infection was 82·1%, 73·8%, and 53·5% with hand hygiene, using a mask, and using gloves, respectively. One hundred and twenty (47·1%) HCPs reported a history of respiratory tract infection (RTI) during the outbreak.

Thirty-three (13%) HCPs had a serum HI titer ≥40 suggesting acquisition of 2009 H1N1 infection during the outbreak. The proportions seropositive in HCPs aged <25, 25–49, and ≥50 years were 20·9%, 12·9%, and 2·8%, respectively. HCPs with a history of RTI during the outbreak tended to have a higher proportion seropositive than those who had no history of RTI (16·7% versus 8·9%, $P = 0·06$). The proportions seropositive among nurses and nurse assistants in different patient care areas were not different: 12·5% versus 19·3% versus 11·6% ($P = 0·411$) in isolation, semi-open, and open wards, respectively.

The univariate analysis revealed that a younger age (<25 years) and visiting crowded public places during the outbreak were associated with acquiring infection. In multivariate analysis, the only risk factor was visiting crowded public places during the outbreak (odds ratio = 3·1, 95% CI = 1·2-8·1, $P = 0·019$, Table 1). The rates of adherence to IC practices were not associated with acquisition of infection. The HCPs with adherence to hand hygiene and mask use at 70–90% of exposure events had a similar rate of infection to those with adherence >90–100%. In a subgroup analysis of 198 nurses and nurse assistants, there was a weak association between acquisition of 2009 H1N1 infection and close contact with HCPs with influenza-like illness (ILI) during the outbreak without wearing a mask (odds ratio = 2·3, 95% CI 0·9–5·6, $P = 0·039$).

Discussion

This study revealed a rate of 2009 H1N1 infection among frontline HCPs of 13%, indicated by a serum HI titer ≥40, after the first outbreak in Bangkok. This result was consistent with a previous report of 18% of HCPs in a large hospital in Bangkok who became sick with the 2009 H1N1 infection during the same outbreak,[5] and higher than the 7% reported among healthy blood donors in Bangkok around the same period of this study,[6] suggesting that HCPs had a higher risk of getting 2009 H1N1 infection than the general population.

A report of the 2009 H1N1 outbreak in England found that 42%, 20%, and 6% of the general population age 5–14, 15–24, and 25–44 years in London and West Midlands had an HI titer against 2009 H1N1 of ≥32.[7] This evidence of higher prevalence in a younger age group correlated well with a large study of the 2009 H1N1 outbreak in the US in which 40% and 35% of the patients were in the age groups 10–18 and 19–50 years, respectively, and only 5% occurred at age >50 years.[8] This age bias was probably due to the increased number of social or institutional gathering

Table 1. Factors associated with serologic evidence of recent infection defined by hemagglutination inhibition (HI) titer ≥40

Characteristics	No. (%)	No. (%) Cases with HI ≥ 40	No. (%)Cases with HI <40	Crude odds ratio (95% CI)	*P*-value	Adjusted odds ratio (95% CI)	*P*-value
Age (years)	(*n* = 256)	(*n* = 33)	(*n* = 223)				
<25	43 (16·8)	9 (27·3)	34 (15·2)	9·0 (1·1–405·8)	0·0198*	5·7 (0·7-49·0)	0·111
25–49	178 (69·5)	23 (69·7)	155 (69·5)	5·0 (0·8–213·8)	0·14*	4·3 (0·6-33·6)	0·163
50–64	35 (13·7)	1 (3)	34 (15·3)	1			
Body Mass Index	(*n* = 254)	(*n* = 33)	(*n* = 221)				
≥25	44 (17·3)	4 (12·1)	40 (18·1)	0·6 (0·2–1·9)	0·47*		
<25	210 (82·7)	29 (87·9)	181 (81·9)	1			
Career	(*n* = 256)	(*n* = 33)	(*n* = 223)				
Physician	30 (11·7)	3 (9·1)	27 (12·1)	1			
Nurse or nurse assistant	208 (81·3)	28 (84·8)	180 (80·7)	1·4 (0·4–7·7)	0·78*		
Others	18 (7)	2 (6·1)	16 (7·2)	1·1 (0·1–10·9)	1·00*		
Received the seasonal influenza vaccine between April 2008 and May 2009	(*n* = 250)	(*n* = 32)	(*n* = 218)				
Yes	205 (82)	27 (84·4)	178 (81·7)	1·2 (0·4–4·3)	0·71		
No	45 (18)	5 (15·6)	40 (18·3)	1			
Living with children younger than 5 years	(*n* = 255)	(*n* = 33)	(*n* = 222)				
Yes	52 (20·4)	8 (24·2)	44 (19·8)	1·3 (0·5–3·2)	0·56		
No	203 (79·6)	25 (75·8)	178 (80·2)	1			
Living with children age 5–15 years	(*n* = 253)	(*n* = 33)	(*n* = 220)				
Yes	59 (23·3)	4 (12·1)	55 (25·0)	0·4 (0·1–1·3)	0·12*		
No	194 (76·7)	29 (87·9)	165 (75·0)	1			
Having household member sick with respiratory tract infection during the outbreak	(*n* = 253)	(*n* = 33)	(*n* = 220)				
Yes	117 (46·2)	12 (36·4)	105 (47·7)	0·6 (0·3–1·4)	0·22		
No	136 (53·8)	21 (63·6)	115 (52·3)	1			
Visiting crowded public places during the outbreak	(*n* = 256)	(*n* = 33)	(*n* = 223)				
Yes	149 (58·2)	27 (81·8)	122 (54·7)	3·7 (1·4–11·4)	0·0032	3·1 (1·2–8·1)	0·019
No	107 (41·8)	6 (18·2)	101 (45·3)	1			
Mask type used when caring for patients with suspected/confirmed 2009 H1N1	(*n* = 239)	(*n* = 30)	(*n* = 209)				
N95 respirator	142 (59·4)	16 (53·3)	126 (60·3)	1			
Surgical Mask	78 (32·6)	10 (33·3)	68 (32·5)	1·2 (0·4–2·9)	0·73		
Use either mask	19 (8)	4 (13·4)	15 (7·2)	2·1 (0·4–7·7)	0·26*		
Frequency of hand hygiene before and after caring for patients with suspected/confirmed 2009 H1N1	(*n* = 246)	(*n* = 32)	(*n* = 214)				
All the time (>90–100%)	202 (82·1)	27 (84·4)	175 (81·8)	1·2 (0·4–4·1)	0·76		
Most of the time (70–90%)	43 (17·5)	5 (15·6)	38 (17·8)	1			
Sometimes (<60%)	1 (0·4)	0	1 (0·4)	–			
Frequency of mask use when caring for patients with suspected/confirmed 2009 H1N1	(*n* = 244)	(*n* = 32)	(*n* = 212)				
All the time (>90–100%)	180 (73·8)	24 (75·0)	156 (73·6)	0·9 (0·4–2·5)	0·86		
Most of the time (70–90%)	56 (23)	8 (25)	48 (22·6)	1			
Sometimes (<60%)	8	0	8 (3·8)	–			
Frequency of glove use when caring for patients with suspected/confirmed 2009 H1N1	(*n* = 245)	(*n* = 32)	(*n* = 213)				
All the time (>90–100%)	131 (53·5)	19 (59·4)	112 (52·6)	1·4 (0·4–4·9)	0·57		
Most of the time (70–90%)	69 (28·2)	8 (25)	61 (28·6)	1·0 (0·3–4·4)	0·94		
Sometimes (<60%)	45 (18·3)	5 (15·6)	40 (18·8)	1			
Having respiratory tract infection during the outbreak	(*n* = 255)	(*n* = 32)	(*n* = 223)				
Yes	120 (47·1)	20 (62·5)	100 (44·8)	2·1 (0·9–4·8)	0·06	1·8 (0·8-4·0)	0·132
No	135 (52·9)	12 (37·5)	123 (55·2)	1			

*Fisher's exact test.

activities and less pre-existing cross-protective antibody in the younger compared with the older participants.[9] In our study, we also found that having an HI titer ≥40 was associated with younger age and visiting crowded public places, suggesting that the outbreak spread more widely in a younger population, probably from gathering in public places.

Conducting a serologic study immediately after the first outbreak of 2009 H1N1 infection was a unique opportunity to look at the effectiveness of IC practices. Assuming from a previous study that approximately 1.4% of healthy people had an HI antibody titer to 2009 H1N1 ≥ 40 before the outbreak,[6] we can expect that the serologic evidence reported is primarily due to the acquisition of infection during the outbreak. The HCPs who participated in this study were the frontline personnel at highest risk of exposure to 2009 H1N1-infected patients and were well trained in using PPE and IC practices. Ideally, these HCPs should not acquire 2009 H1N1 infection from patient care. However, our set-up was not ideal. Aerosol-generating procedures were performed in many of the patient care areas and none of the wards had the perfect negative pressure needed to limit aerosol spread. Moreover, adherence to IC practices was not perfect in real life.

A previous report revealed that frontline HCPs were actually less likely to contract 2009 H1N1 than other HCPs, probably because of good adherence to IC practices and use of PPE.[10] However, we found that only about half of our HCPs reported perfect adherence (>90% of exposure events) to wearing of gloves, and around 70–80% perfectly adhered to mask use and hand hygiene. Despite this imperfect adherence, it seemed that the HCPs in our study acquired infection from the community rather than from patient care. A report from the US also found acquisition from the community as a major route of infection in HCPs.[11] We did not find the levels of adherence to hand hygiene and mask use (70–90% versus 90–100%) to be associated with infection. This is in line with a previous study that revealed a protective effect of >75% adherence to hand hygiene.[10]

The hospital environment is the setting where HCPs spend much of their time and may be the source of infection, in addition to patient care activities. Environmental contamination in household settings of patients with influenza was well documented.[12] A study in Singapore reported that contact with H1N1-infected colleagues was associated with 2009 H1N1infection in hospital staff.[13] We found that exposure to HCPs with ILI was associated with 2009 H1N1 infection in a subgroup analysis of nurses and nurse assistants, but not in the whole cohort analysis. Of note, almost half of the HCPs in this study had an RTI during the outbreak but only 16·7% of these RTI episodes appeared to be associated with HI seroconversion to 2009 H1N1 suggesting that other respiratory viruses co-circulated during the outbreak, and this may have masked the effect of ILI contact in the whole cohort. Hospital policies to monitor ill HCPs and prevent transmission of infection from ill HCPs are needed.

There are several limitations of the study. First of all, our sample size was not large. Inherent self-report survey-based research compared with observer-based research, in particular when reporting hand hygiene and PPE habits, is that over-reporting of the habit cannot be excluded. We do not have verification of the accuracy of the answers to the questionnaire and we did not monitor for adherence to IC practices in our study.

In conclusion, we found that of 2009 H1N1 infection among frontline HCPs was somewhat higher than in the general population. The risk of infection was found to be associated with community exposure risk, particularly in the young, as well as exposure to other HCPs with ILI without protection.

Acknowledgements

We thank Varaporn Pumsuwan, M.Sc., Natcha Viruchkul, M.N.S, Wanatpreeya Phongsamart, MD, Orasri Wittawatmongkol, MD, Alan Maleesatharn, MBA, Amy Downing, MD, and Hatairat Lerdsamran, M.Sc., Thanarak Plipat, MD, and Tim R Cressey, Ph.D for their contribution to the study. We thank all the participants for their volunteerism to join the study and response to the questionnaire. This study was supported by the National Science and Technology Development Agency and the Thailand Research Fund for Senior Research Scholar and The National Research University Project of the Thailand Office of Higher Education Commission (A.A.).

References

1 WHO. Pandemic H1N1 2009- Thailand 31st March 2010. Available at http://www.searo.who.int/EN/Section10/Section2562_15043.htm (Accessed 22 February 2011).
2 Garcia-Garcia L, Valdespino-Gomez JL, Lazcano-Ponce E *et al.* Partial protection of seasonal trivalent inactivated vaccine against novel pandemic influenza A/H1N1 2009: case-control study in Mexico City. BMJ 2009; 339:b3928.
3 Louisirirotchanakul S, Lerdsamran H, Wiriyarat W *et al.* Erythrocyte binding preference of avian influenza H5N1 viruses. J Clin Microbiol 2007; 45:2284–2286.
4 Kitphati R, Pooruk P, Lerdsamran H *et al.* Kinetics and longevity of antibody response to influenza A H5N1 virus infection in humans. Clin Vaccine Immunol 2009; 16:978–981.
5 Kiertiburanakul S, Apivanich S, Muntajit T *et al.* H1N1 2009 influenza among healthcare workers in a tertiary care hospital in Thailand. J Hosp Infect 2010; 74:300–302.
6 Lerdsamran H, Pittayawonganon C, Pooruk P *et al.* Serological Response to the 2009 Pandemic Influenza A (H1N1) Virus for Disease Diagnosis and Estimating the Infection Rate in Thai Population. PLoS ONE 2011; 6:e16164.

7 Miller E, Hoschler K, Hardelid P, Stanford E, Andrews N, Zambon M. Incidence of 2009 pandemic influenza A H1N1 infection in England: a cross-sectional serological study. Lancet 2010; 375:1100–1108.

8 Dawood FS, Jain S, Finelli L *et al.* Emergence of a novel swine-origin influenza A (H1N1) virus in humans. N Engl J Med 2009; 360:2605–2615.

9 Serum cross-reactive antibody response to a novel influenza A (H1N1) virus after vaccination with seasonal influenza vaccine. MMWR Morb Mortal Wkly Rep 2009;58:521–524.

10 Apisarnthanarak A, Mundy LM. Factors associated with health care-associated 2009 influenza a (H1N1) virus infection among Thai health care workers. Clin Infect Dis 2010; 51:368–369.

11 Novel influenza A (H1N1) virus infections among health-care personnel - United States, April-May 2009. MMWR Morb Mortal Wkly Rep 2009;58:641–645.

12 Simmerman JM, Suntarattiwong P, Levy J *et al.* Influenza virus contamination of common household surfaces during the 2009 influenza A (H1N1) pandemic in Bangkok, Thailand: implications for contact transmission. Clin Infect Dis 2010; 51:1053–1061.

13 Chen MI, Lee VJ, Barr I *et al.* Risk factors for pandemic (H1N1) 2009 virus seroconversion among hospital staff, Singapore. Emerg Infect Dis 2010; 16:1554–1561.