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Labor Awareness to Virus A (H5N1) in Hong Kong's Livestock Industry

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Abstract

18 confirmed human cases of H5N1 sickness coincided with outbreaks of highly pathogenic influenza A (H5N1) in poultry in 1997. Although exposure to live chickens was linked to human sickness, no cases among poultry employees were recorded (PWs). A cohort study involving 293 Hong Kong government employees (GWs) who took part in a poultry culling operation and 1525 PWs was carried out to assess the possibility of H5N1 transmission from birds to humans. When anti-H5 antibodies were detected by both micro neutralization and Western blot analysis, paired serum samples from GWs and single serum samples from PWs were deemed to be anti-H5 antibody positive. One documented seroconversion and 3% of GWs were seropositive. 10% of PWs exhibited anti-H5 antibodies. Anti-H5 antibodies were linked to more intensive chicken exposure, such as butchering and exposure to sick poultry. These data imply that work exposure increases the likelihood of contracting avian influenza.

Keywords: Pathogenic influenza; Anti-H5 antibodies; Sick poultry; Micro neutralization

Introduction

Hong Kong residents reported 18 instances of avian influenza A (H5N1) infection in 1997; all of the case patients required hospitalization, and six of them passed away [1]. The epidemic happened concurrently with outbreaks of highly pathogenic avian influenza (HPAI) H5N1 among chickens on 3 Hong Kong farms from March through May (1 human case), and among chickens in wholesale and retail markets from November through December (17 human cases). This was the first instance where a subtype of avian influenza A virus was linked to human respiratory illnesses.

According to gene sequence data, the avian H5N1 viruses that were recovered from chickens and people in Hong Kong in 1997 had genomes that were closely related to one another and had a multibasic amino acid motif with HPAI at the location where the HAl and HA2 genes cleave. A case-control study carried out in January 1998 suggested that the main risk factor for human H5N1 infection was exposure to chicken in retail markets [2]. The genetic and epidemiologic evidence taken together suggested that the human H5N1 infections were caused by numerous, separate poultry-to-human H5N1 viral transmissions [3, 4]. The Hong Kong government recruited staff from a number of agencies to help with the slaughter of chickens and other poultry across the entire territory of Hong Kong due to the possibility of increased human-to-poultry H5N1 virus transmission in the poultry markets. A total of 1.5 million chickens and many hundred thousand additional domestic birds were killed during this 4-day operation that started on December 29. Following the slaughter of the birds, no new human H5N1 cases were found [1].

Prior to the depopulation of poultry, surveillance in Hong Kong's live bird markets revealed that 20% of hens tested from those markets was H5N1 virus positive [5]. However, among poultry workers (PWs), a group anticipated to have the highest amount of contact to H5N1-infected birds no H5N1 case patients was found. By conducting a retrospective cohort study among PWs and government employees (GWs) involved in the slaughter of poultry, we evaluated the rates of and risk factors for H5N1 infection among people exposed to poultry in order to better understand the potential for the H5N1 viruses to spread to humans.

Materials and Methods

Enrollment PWs and GWs working in the slaughter of poultry were invited to visit any of the 14 Hong Kong Government outpatient clinics and take part in a study on H5N1 infection in people who had been exposed to poultry. Participating From December 29 to January 15, 1998, GWs and PWs were recruited for the study. All participants had to fill out a self-administered questionnaire that was initially created in English and then translated into Chinese. Participants' age, sex, type of poultry employment, exposure to H5N1 patients (the "bird flu"), respiratory ailments they had since November 1, 1997, and exposure to H5N1 patients were all questioned. PWs were also asked if, since November 1, 1997, they had dealt with poultry, and if so, whether they had noticed a mortality rate of more than 10%.

Collect blood PWs were requested to submit a single serum sample because it was unable to determine with precision when they first came into contact with potentially H5N1-infected poultry. GWs were asked to submit paired serum samples, with the first sample being taken 0–7 days after the end of the culling operations and the second sample being taken two weeks after the first sample. The dates of the culling operation characterized the GWs' period of intense poultry exposure. Blood samples were divided into serum and kept at - 20°C until testing.

Testing for antibodies despite being often used to identify influenza antibodies in human serum, the hem agglutination inhibition (HI) assay is not sensitive enough to identify antibodies to the H5N1 virus [6]. In order to identify H5-specific antibody in the current work, a micro neutralization technique was utilized first, followed by validation using a Western blot experiment. These assays' sensitivity and specificity for detecting anti-H5 HA antibodies in patients under the age of 15 have been previously documented [6]. At the Government Virus Unit, Hong

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Kong Department of Health, serum samples were examined using the micro neutralization assay using a nonpathogenic avian H5N3 virus A/Duck/Singapore/-Q/F119-3/97 (Duck/Singapore). By using the micro neutralization technique, serum samples were deemed to be positive if anti-H5 titers of less than 80 were obtained.

At the Centers for Disease Control and Prevention (CDC), blood samples that tested positive in the micro neutralization assay underwent a confirmatory Western blot assay [7]. In order to identify IgG antibodies in serum diluted 1:100, this experiment utilized a highly pure baculovirus-expressed HA protein from the A/Hong Kong/156/97 virus (kindly donated by Bethanie Wilkinson, Protein Sciences, Meriden, CT). Anti-H5 antibody was thought to be present in serum samples that tested positive in the micro neutralization assay and Western blot [6].

Statistical analysis

Not all PW serum samples that were positive in the micro neutralization testing at the CDC could be examined by Western blot due to a lack of resources. So, a random sample of micro neutralization-positive serum from the 15–29, 30-44, and 45–59 age groups was chosen for Western blot analysis. We used a nested unmatched case control study to evaluate the risk factors for H5 infection. All PWs who tested positive for both micro neutralization and Western blot included as case patients. All PWs who tested negative by the micro neutralization assay as well as those who tested positive by the assay but negative by the Western blot were included as control subjects. PWs who tested positive by micro neutralization but whose serum samples were not analyzed by Western blot were excluded from the nested case-control study. Based on the proportion of PWs who tested positive by micro neutralization and the proportion of those predicted to test positive by Western blot, weighted by age group, we estimated the seroprevalence of H5 among PWs. All serum samples that tested positive for micro neutralization in GWs were Western blot examined, and the results for this population were analyzed using a cohort research methodology. Data was examined using SAS for Windows (version 8.01; SAS Institute). The micro neutralization and Western blot assays were found to be less specific for individuals 60 and 14 years old, thus they were omitted from the analysis along with those who did not indicate their age.

Results

Seroprevalence of H5N1 among PWs. 1525 PWs aged 15-59 were enrolled in the research between December 29 and January 15; of these, 142 (9%) were aged 15-29, 806 (53%) were aged 30-44, and 577 (38%) were aged 45-59. 444 (29.1%) of all PWs were positive for H5 using the micro neutralization technique. 231 (52%) of these 444 PWs underwent a Western blot test, and 81 (35%) of them confirmed positive. The age group-weighted H5-seropositive rate estimate was 10% based on the percentages of positive micro neutralization samples that were also Western blot positive.

PWs case-control nested analysis. 81 (6%) of the 1312 PWs in the nested case-control analysis were antibody positive, including 2% (3/134) of those aged 15 to 29 years old, 8% (56/705) of those aged 30-44, and 5% (22/473) of those aged 45 to 59. Working in retail poultry operations as opposed to wholesale or a poultry hatchery or farm, reporting mortality of more than 10% among the poultry they worked with, butchering and feeding poultry, and preparing poultry for restaurants, which typically entails butchering chickens, were factors statistically associated with H5 seropositivity in a stratified analysis that controlled for age group. As more reported exposure types were reported, the chance of harboring H5 antibodies increased. Numerous PWs reported performing more than one task connected to poultry, and significant collinearity between the several types of exposures looked at prevented the use of logistic Modelling. However, stratified analysis revealed that the exposures most strongly linked with H5 seropositivity were slaughtering poultry and exposure to poultry with >10% mortality. After adjusting for slaughtering, the effect of feeding poultry was insignificant. Smoking and anti-H5 antibody were not related.

Traits of the participating GWs a questionnaire and a blood sample were provided by the 293 GWs who participated in the poultry culling operation and were over the age of 60. 85% of the population was male, the median age was 41, and 22.5% of people smoked. Overall, 229 out of 293 serum samples were paired. Eight people had paired serum samples, and two of those samples were obtained more than 14 days after the culling procedure. Blood was taken from 52 (81%) of the 64 individuals who provided a single serum sample within 14 days after the culling operation.

Discussion

The findings of this study show that those who work with domestic poultry are more likely to contract the H5N1 virus. For the purpose of assessing the dangers of human infection with animal influenza viruses, occupationally exposed individuals may represent significant populations.

Comparing the reported H5 seroprevalence rate of 10% among PWs to the other Hong Kong cohorts studied in 1997 using analogous antibody testing techniques, this rate is considerable. Seroprevalence rates were 0% among adult blood donors [7] and 0.7% among healthcare professionals [8] among groups not exposed to infected human case patients and with assumed low levels of poultry exposure. These low background rates contrast with rates of 3.7% among medical personnel who treated H5N1 case patients [8] and 3.8% among those who travelled with a case patient who was symptomatic [9], which are rates comparable to those seen among GWs. A group likely to have had similar environmental exposures as the case patients, including contact with contaminated poultry, in addition to case-patient exposure, the seroprevalence rate among PWs was comparable to the 12% rate identified among household contacts of case patients [9]. The higher H5 seroprevalence rate among PWs, of whom only one, a seronegative person, reported knowing a case patient, and evidence from a case-control study [10] suggest that poultry-to-human transmission was the main source of human infection, despite epidemiologic evidence of human-to-human transmission being found in the study of health care workers [8].

Short ridge [11] reported human incidence rates for antibody to the H5 virus ranging from 0% in an urban Hong Kong population to 2%–7% in rural populations in southern China, where H5 viruses were isolated from 4% of domestic ducks tested, using the single radial hemolysis (SRH) assay to detect antibody to various avian virus subtypes. These rates might have been overestimated since the SRH assay may also identify subtype cross-reactive antibodies to the internal nucleoprotein in addition to subtype-specific antibodies to influenza surface glycoproteins. No antibodies against the HAs of the avian viruses (HIIN2, H4N4, H7N4, and H3N8) isolated from duck feaces on these farms were discovered in a subsequent serosurvey of farm households that produced ducks and pigs [12]. However, it's possible that the study's use of the HI test to find avian virus antibodies reduced its sensitivity of detection. When looking for human antibodies against

H5N1 viruses, the HI test is less accurate than the micro neutralization assay [6]. When looking for antibodies in additional mammalian species infected with an avian H2N2 virus [13] obtained a similar finding.

Experimentally infected hens succumbed to a quickly progressing, lethal sickness brought on by the H5N1 HPAI viruses that were isolated from both humans and chickens in Hong Kong [2]. The heart's myocytes, myeloid inflammatory cells, and vascular endothelial cells are where the HPAI H5N1 viruses reproduce most frequently. The findings of our study, which discovered a connection between harboring anti-H5 antibodies and slaughtering and preparing poultry for restaurants, tasks that require very close contact with poultry, are supported by the pathophysiology of HPAI.

The PW study had the drawback of just having one serum sample taken. This means that the exact moment of H5 virus infection cannot be predicted. It's probable that past exposure to a related H5 virus led to the anti-H5 antibody found in at least some PWs. Prior to the depopulation of poultry, viral surveillance in retail markets detected H5N1 viruses from 20% of chickens tested and from 2.4% and 2.5% of ducks and geese, respectively [5]. The possibility still exists that PWs may have been shielded from subsequent infection or severe disease with the HPAI H5N1 virus during the 1997–1998 pandemic by an earlier subclinical or undetected H5 virus infection.

The investigation of GWs gave researchers a rare chance to look into a group that had experienced brief but extensive exposure to birds afflicted with the H5N1 virus. One person had a seroconversion that was recorded. First serum samples from eight additional GWs had anti-H5 antibody results that were favourable. After the first day of the chicken depopulation, 8 to 11 days later, the first serum samples were taken. Although it would seem early for a primary antibody response to a new virus, seroconversion was found in 1 adult H5N1 case patient as early as 7 days after the onset of symptoms [9]. However, seroconversion that occurred less than 14 days following the onset of symptoms was more typical. Four of the eight GWs were absent in the second serum samples that were taken 14 days after the first samples. In one instance, a modest and transitory antibody response to infection was indicated by the second sample's neutralizing antibody titer of 80. The micro neutralization titers of the second serum samples for the other 3 people were comparable to those of the corresponding first samples; however the second serum samples failed the confirmatory Western blot experiment. Unlike the Western blot, which only found IgG, the micro neutralization assay may have picked up IgG, IgM, or IgA antibodies. In the second serum sample, which was taken three to four weeks after virus exposure, a subclinical viral infection may cause weak and temporary IgG responses and low levels of antigen expression. Because several GWs with a single blood sample did not donate a later convalescent specimen for testing, the seroprevalence rate of 3% for the entire cohort may have been underestimated.

In GWs but not PWs, smoking was discovered to be a risk factor for H5 antibody. Smoking has been linked to higher rates of seroconversion and clinical influenza disease in several investigations of young adults [14, 15]. Smoking however only seemed to raise the risk in people who had no antibody titers beforehand. Because PWs already had antibodies from past exposures to H5 avian viruses, the same risk factor may not have been present in those individuals.

Our study's findings are in contrast to those of a small serological survey of people who had contact with infected poultry or people who had been exposed to the virus during the Italian chicken outbreak caused by the HPAI H5N2 virus in 1997 [16]. No anti-H5 antibodies were found in blood samples from exposed people when a micro neutralization assay and the SRH test were utilized, and no human cases of H5N2 respiratory illness were found. Similarly, no human infections were found during HPAI H5N2 virus epidemics that plagued hens in the northeastern United States in the early 1980s, causing significant morbidity and mortality. However, no virus was identified from swabs taken 12 hours after those involved in the depopulation of hens departed the contaminated chicken houses, indicating that people were not vulnerable to this HPAI H5N2 virus [17]. It is unknown if the H5N1 viruses from Hong Kong are distinct from other H5 HPAI viruses in terms of their capacity to infect people. It's likely that they had a particular gene combination that allowed for a little amount of virus transmission from poultry to humans. Two children with respiratory illnesses were found to have avian H9N2 viruses in Hong Kong in 1999, which had internal genes that were quite similar to those of the H5N1 viruses.

Another HPAI H5N1 outbreak was discovered in Hong Kong's poultry during May 2001, and it was contained by the culling of birds. No signs of human cases were found. This H5 virus is different from the H5N1 virus that was responsible for the pandemic in 1997. (Yi Guan, personal communication). However, the 1999 H9N2 virus infection that led to human sickness and the 2001 HPAI H5N1 outbreak among poultry show the necessity for ongoing surveillance of both humans and animals.

Conclusion

Further proving the ability of avian influenza viruses to infect humans is the serologic data for infections in PWs and GWs given here. These results emphasize the necessity of conducting more seroprevalence investigations in communities of people in Asia and abroad who are exposed to domestic poultry through live bird markets. These investigations will increase our knowledge of the potential for avian influenza viruses to cause epizootic and may reveal candidate subtypes of avian influenza with pandemic optical.

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