Editorial

Avian influenza (Bird flu)

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Avian influenza (Bird flu) is an infectious disease of birds caused by different subtypes of influenza A virus. These subtypes differ because of changes in haemagglutinin and neuraminidase proteins on the surface of the virus[1]. Natural carriers of the bird flu virus are migratory wildfowl of which wild ducks form the largest group[2]. However, domestic birds are particularly susceptible to the bird flu virus. Wildfowl and other migratory birds store the bird flu virus in their intestines and pass it out in their faeces. Dried faeces can become pulverized and transported in the wind where it can contaminate and infect other birds and humans[2].

Infected birds shed the bird flu virus in their saliva, nasal secretions, and faeces. Susceptible birds become infected when they have contact with contaminated secretions or excretions or with surfaces that are contaminated with secretions or excretions from infected birds[1]. Domesticated birds may become infected with avian influenza virus through direct contact with infected waterfowl or other infected poultry, or through contact with surfaces (such as dirt or cages) or materials (such as water or feed) that have been contaminated with the virus[1].

Infection with avian influenza viruses in domestic poultry causes two main forms of disease that are distinguished by low and high extremes of virulence[1]. The "low pathogenic" form may go undetected and usually causes only mild symptoms such as ruffled feathers and a drop in egg production. However, the highly pathogenic form spreads more rapidly through flocks of poultry. This form may cause disease that affects multiple internal organs and has a mortality rate that can reach 90-100% often within 48 hours[1]. To date, all outbreaks of the highly pathogenic form have been caused by influenza A viruses of subtypes H5 and H7[2].

The first documented infection of humans with an avian influenza virus occurred in Hong Kong in 1997, when the H5N1 strain caused severe respiratory disease in 18 humans, of whom 6 died[2]. In 1997, exposure to live poultry within a week before the onset of illness was associated with disease in humans, whereas there was no significant risk related to eating or preparing poultry products or exposure to persons with influenza A (H5N1) disease[3]. Exposure to ill poultry and butchering of birds were associated with seropositivity for influenza A (H5N1)[4]. Recently, most patients have had a history of direct contact with poultry, although not those who were involved in mass culling of poultry. Plucking and preparing of diseased birds; handling fighting cocks; playing with poultry, particularly asymptomatic infected ducks; and consumption of duck's blood or possibly undercooked poultry have all been implicated.

Human-to-human transmission of influenza A (H5N1) has been suggested in several household clusters[5] and in one case of apparent child-to-mother transmission[6]. Intimate contact without the use of precautions was implicated, and so far no case of human-to-human transmission by small-particle aerosols has been identified. In 1997, human-to-human transmission did not apparently occur through social contact[7] and serologic studies of exposed health care workers indicated that transmission was inefficient[8]. To date, the risk of nosocomial transmission to health care workers has been low, even when appropriate isolation measures were not used[9]. So far, the spread of H5N1 virus from person to person has been limited and has not continued beyond one person[1]. Nonetheless, because all influenza viruses have the ability to change, scientists are concerned that the H5N1 virus would one day be able to infect humans and spread easily from one person to another. Because these viruses do not commonly infect humans, there is little or no immune protection against them in the human population. If H5N1 virus was to gain the capacity to spread easily from person to person, an influenza pandemic could begin[1].

The incubation period of avian influenza A (H5N1) may be longer than for other known human influenzas. In 1997, most cases occurred within two to four days after exposure with ranges of up to eight days[12]. Symptoms of avian influenza in humans have ranged from typical human influenza-like symptoms (e.g., fever, cough, sore throat, and muscle aches) to

eye infections, pneumonia, severe respiratory diseases (such as acute respiratory distress), and other severe and life-threatening complications[2]. Progression to respiratory failure has been associated with diffuse, bilateral, ground-glass infiltrates and manifestations of the acute respiratory distress syndrome[13]. Multiorgan failure with signs of renal dysfunction and sometimes cardiac compromise, including cardiac dilatation and supraventricular tachyarrhythmias, have been common[13]. Other complications have included ventilator-associated pneumonia, pulmonary haemorrhage, pneumothorax, pancytopenia, Reye syndrome, and sepsis syndrome without documented bacteraemia[13]. The clinical spectrum of influenza A (H5N1) in humans is based on descriptions of hospitalized patients. The frequencies of milder illnesses, subclinical infections, and atypical presentations (e.g., encephalopathy and gastroenteritis) have not been determined, but case reports[10] indicate that each occurs. Most patients have been previously healthy young children or adults[11]. In contrast to 1997, when most deaths occurred among patients older than 13 years of age, recent avian influenza A (H5N1) infections have caused high rates of death among infants and young children[13]. The case fatality rate was 89 percent among those younger than 15 years of age in Thailand. Death has occurred an average of 9 or 10 days after the onset of illness (range 6 to 30), and most patients have died of progressive respiratory failure[13]. It is remarkable that in 1998, for the first time in Sri Lanka, multiorgan involvement associated with influenza A virus was documented, although this epidemic was attributed to the H_3N_2 strain[14].

Common laboratory findings have been leucopenia, particularly lymphopenia; mild-to-moderate thrombocytopenia; and slightly or moderately elevated aminotransferase levels. Marked hyperglycaemia, perhaps related to corticosteroid use, and elevated creatinine levels also occur[5]. In Thailand[13], an increased risk of death was associated with decreased leucocyte, platelet, and particularly, lymphocyte counts at the time of admission.

Antemortem diagnosis of influenza A (H5N1) has been confirmed by viral isolation, the detection of H5-specific RNA, or both methods. Commercial rapid antigen tests are less sensitive in detecting influenza A (H5N1) infections than are RT-PCR assays[13].

Most hospitalized patients with avian influenza A (H5N1) have required ventilatory support within 48 hours after admission[5,13], as well as intensive care for multiorgan failure and sometimes hypotension. In addition to empirical treatment with broad-spectrum antibiotics, antiviral agents, alone or with corticosteroids, have been used in most patients, although their effects have not been rigorously assessed. The institution of these interventions late in the course of the disease has not been associated with an apparent decrease in the overall mortality rate, although early initiation of antiviral agents appears to be beneficial.[13] Cultivable virus generally disappears within two or three days after the initiation of oseltamivir among survivors, but clinical progression despite early therapy with oseltamivir and a lack of reductions in pharyngeal viral load have been described in patients who have died.

Patients with suspected influenza A (H5N1) should promptly receive a neuraminidase inhibitor pending the results of diagnostic laboratory testing. The optimal dose and duration of treatment with neuraminidase inhibitors are uncertain, and currently approved regimens likely represent the minimum required. These viruses are susceptible in vitro to oseltamivir and zanamivir[15]. Early treatment will provide the greatest clinical benefit[13] although the use of therapy is reasonable when there is a likelihood of ongoing viral replication.

Influenza is a recognized nosocomial pathogen. Current recommendations are based on efforts to reduce transmission to health care workers and other patients in a non-pandemic situation and on the interventions used to contain SARS[16]. The efficiency of surgical masks, even multiple ones, is much less than that of N-95 masks, but they could be used if the latter are not available. Chemoprophylaxis with 75 mg of oseltamivir once daily for 7 to 10 days is warranted for persons who have had a possible unprotected exposure[17].

Several measures may be implemented to prevent the spread of H5N1[16]:

- Culling of infected birds to prevent the transmission of the virus to humans.
- Quarantine and treat infected human promptly with antiviral drugs such as oseltamivir or zanamivir.
- Pens should be protected from wildfowl and migratory birds.

- People who are engaged in cleaning, slaughtering and processing of poultry should take the necessary precautionary measures to prevent themselves from being infected and minimize the spread of infection.

- Monitoring of the migratory patterns of wild birds should provide early alerts of the arrival of infected birds which could then be targeted on arrival.

- People who eat poultry are not at risk for the H5N1 strain, but precautionary measures such as cooking all meat to a temperature of at least 70 ºC should be done. Eggs should be thoroughly cooked.

- Poultry imports from foreign countries should be accompanied by the necessary certification from the relevant competent authority in the country of origin indicating that the poultry carcasses are free from disease and fit for human consumption.

There currently is no commercially available vaccine to protect humans against H5N1 virus that is being seen in Asia and Europe[1]. However, vaccine development efforts are taking place. Research studies to test a vaccine to protect humans against H5N1 virus began in April 2005, and a series of clinical trials is under way[1].

Subsequent to the detection of bird flu in neighbouring India, there is growing concern about the outbreak of the disease in Sri Lanka. However, so far, no poultry birds suffering from the avian flu virus have been diagnosed in Sri Lanka. Furthermore, the migrating period of birds in Sri Lanka is over and the birds are now flying back. However, it should be remembered that the migratory season begins again around September and continues till March.

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