A Concise Introduction of Avian Influenza and Its Pathogen: the Requirement of Public Health

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Abstract: In the late 1990s, types of H5N1 and H9N2 avian influenza viruses (AIV) have infected respiratory tract in humans in Hong Kong through the domestic poultry to the potential contact with human being, which highlights the status of public health of veterinary pathogen. Particularly, the recent outbreak of avian influenza (AI) together with the temporary SARS prevalence and the natural disaster caused by tsunami in Asia, impacting on the economic and society significantly. Therefore, we emphasize here the importance of AIV and depict the etiology, molecular biology, clinical signs, diagnosis and prevention of AI in order to update the scientific literatures, increase the understanding to AIV, improve the prevention consciousness in public. [Nature and Science. 2005;3(2):92-94].

Key words: avian influenza; public health; pathogen; prevention

1 Why Avian Influenza Viruses Become the Spotlight in Public?

Influenza A viruses own 15 hemagglutinin (HA) and 9 neuraminidase (NA) subtypes, existing in waterfowl extensively, is the source of influenza A viruses that infect mammals. Historically, only influenza A H1N1, H2N2 and H3N2 viruses have caused the pandemics in Human (1918, 1957, and 1968 respectively). Normally, pandemic strains possessing novel HA derived from avian or animal influenza viruses, regardless of other accompanying avian virus genes, sporadically emerge in humans and have the potential to cause a pandemic of influenza if the virus is capable of transmitting among a human population that lacks immunity to the novel HA [1]. Generally, avian influenza viruses were not considered to be able to directly infect humans and cause influenza-like respiratory disease due to the discrepancy of specific receptor and a potential barrier within species. However, A high-pathogenicity avian H5N1 influenza viruses circulated among poultry in farms and retail markets in 1997, in Hong Kong (The H5N1 viruses were subsequently transmitted to humans, causing 18 documented cases of respiratory disease, including 6 deaths). Likewise, in 1998–99, a second influenza A virus subtype, H9N2, was isolated from humans with respiratory disease. More recently, some lethal cases have been reported in Asian [2]. Now, there are less doubt that these avian influenza viruses should be considered a risk to public health and social economy.

2 Etiology

The AIV belong to the virus family orthomyxoviridae. The virus particle has an envelope with glycoprotein projections with hemagglutinating and neuraminidase activity. These two surface antigens, HA and NA, are the serologic taxonomy basis of the influenza viruses using the letters H and N with the appropriate numbers in the virus designation e.g., H7N2.
Currently there are 15 hemagglutinin and 9 neuraminidase antigens described among the Type A influenza viruses. The type designation (A, B, or C) is based upon the antigenic character of the M protein of the virus envelope and the nucleoprotein within the virus particle. All influenza viruses affecting domestic animals (equine, swine, avian) belong to Type A, and Type A influenza virus is the most common type producing serious epidemics in humans. Types B and C do not affect domestic animals [3].

3 Molecular Basis of Pathogenicity

Influenza A viruses the hemagglutinin glycoprotein is produced as a precursor, HA0, which requires posttranslational cleavage by host proteases before it is functional and virus particles are infectious. The HA0 precursor proteins of low-pathogenicity avian influenza (LPAI) have a single arginine at the cleavage site and another basic amino acid at position -3 or -4. These viruses are limited to cleavage by extracellular host proteases such as trypsin-like enzymes and thus restricted to replication at sites in the host where such enzymes are found, i.e., the respiratory and intestinal tracts. High-pathogenicity avian influenza (HPAI) viruses possess multiple basic amino acids (arginine and lysine) at their HA0 cleavage sites either as a result of apparent insertion or substitution and appear to be cleavable by a specific protease(s), probably one or more proprotein-processing subtilisin-related endoproteases of which furin is the primary candidate. HPAI viruses can replicate throughout the bird, damaging vital organs and tissues, which result in disease and death [4].

4 Epidemiology

Most of all, AI outbreaks probably start with direct or indirect contact of domestic poultry with waterbirds. Many strains that circulate in wild birds are either non-pathogenic or mildly pathogenic for poultry. However, a virulent strain may emerge either by genetic mutation or by reassortment of less virulent strains. Swine appear to be important in the epidemiology of infection of turkeys with swine influenza virus when they are in close proximity. Other mammals do not appear to be involved in the epidemiology of HPAI. The infection of humans with an H5 avian influenza virus in Hong Kong in 1997 has resulted in a reconsideration of the role of the avian species in the epidemiology of human influenza.

Once AI is established in domestic poultry, it is a highly contagious disease and wild birds are no longer an essential ingredient for spread. Infected birds excrete virus in high concentration in their faeces and also in nasal and ocular discharges. When introduced into a flock, the virus is spread from flock to flock by the usual methods involving the movement of infected birds, contaminated equipment, egg flats, feed trucks, and service crews, to mention a few. The disease generally spreads rapidly in a flock by direct contact, but on occasions spread is erratic.

Airborne transmission may occur if birds are in close proximity and with appropriate air movement. Birds are readily infected via instillation of virus into the conjunctival sac, nares, or the trachea. Some evidences indicate that virus can be recovered from the yolk and albumen of eggs laid by hens at the height of the disease. The possibility of vertical transmission is unresolved. In addition, the hatching of eggs from a diseased flock would likely be associated with considerable risk [5].

5 Clinical Features

Compared with the LPAI, the clinical signs of HPAI are severe and result in high mortality rates (can be 100%) in many species of birds, especially domestic fowl. Typically, after 3-7 days incubation time, the following clinical signs will emerge: Sudden death; Severe depression with ruffled feathers; Inappetence; Drastic decline in egg production; Edema of head and neck; Swollen and cyanotic combs and wattles; Excessive thirst; Watery diarrhea that begins as bright green and progresses to white; Swollen and congested conjunctiva with occasional hemorrhage; Diffuse hemorrhage between hocks and feet; Respiratory signs are dependent on tracheal involvement; Nasal and ocular discharge; Mucus accumulation (varies); Lack of energy; Coughing/sneezing; Incoordination; Nervous system signs such as paralysis. Death may occur prior to any symptoms or as late as a week after symptoms, though it is frequently within 48 hours [6].

6 Diagnosis

According to the clinical manifestations and the characteristics of epidemiology, the preliminary
diagnosis can be easily performed. However, the final conclusion cannot be drawn without the further Laboratory Diagnosis, for example, the isolation and identification of the causative virus. Commercially available type A influenza antigen-capture enzyme linked immunosorbent assay kits designed for use in human influenza have recently shown promise as a possible rapid diagnostic test for poultry. Furthermore, the molecular biological technique, i.e., PCR and molecular pathology will be useful for common and Differential Diagnosis. However, all the diagnosis procedures should be performed by specialists and in special institutions due to the potential risk.

7 Prevention and Treatment

From the views of epidemic angle, to control the source of infection; to cut the transmission pathway; and to protect the susceptible groups are still very important and efficient for AI prevention.

Inactivated oil-emulsion vaccines and viable vaccines prepared using naturally avirulent or attenuated strains can be vaccine candidates, the former is safer that the latter, even though the former is more expensive that the latter in cost. Some drugs, such as amantadine hydrochloride can be administered in drinking water to reduce disease losses, but drug-resistant viruses quickly emerged, negating the initial beneficial effects to some extent [3]. Other prevention policies and treatments remain under investigated, and we are looking forward to hearing good news as soon as possible.

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