Clinical signs and pathologic lesions of highly pathogenic avian influenza in Indonesia: A threat to Indonesian poultry

R. Wasito¹*, Hastari Wuryastuti¹, Rachmat Pamudy² and Roger K. Maes³

¹Faculty of Veterinary Medicine, Gadjah Mada University, Yogyakarta, Indonesia
²Faculty of Animal Husbandry, Institut Pertanian, Bogor, Indonesia
³Virology Section, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, E. Lansing, MI, USA

*Corresponding Author’s E-mail: wst_ugm@yahoo.com

We have studied the clinical signs and pathological lesions responsible for the property characteristic of primary avian influenza type A H5N1 subtype during the 2003 to 2005 outbreaks of the H5N1 influenza viruses in Indonesia. The highest number affected flocks were in layer chickens, with only limited number of quail flock, backyard chicken flock, ducks flock and live-bird markets infected. Those poultry originated from provinces in East Java, West Java, Central Java and Yogyakarta. Depression and droopiness, loss of appetite, sudden drop in egg production, neurologic dysfunction, respiratory distress and diarrhoea were common clinical signs manifestations of the avian influenza infection. Grossly, the most severe and consistent lesions included cyanosis (purplish-blue coloring) of wattles and comb, edema and swelling of head, eyelids, comb, and hemorrhages in the skeletal muscles and feet. Interestingly enough that the quails had only a severely decreased in the general condition and food intakes, and also showed neurologic disorders and diarrhoea. Grossly, the quail lacked significant gross lesions as those seen in the chickens. The clinical signs and pathologic anatomic lesions were analogous to those experimentally induced with other highly pathogenic avian influenza viruses in domestic poultry.

Keywords: Avian influenza, H5N1, clinical signs, pathological lesions, Indonesian poultry

INTRODUCTION

Avian influenza virus is classified within the genus Influenza virus A of the family of Orthomyxoviridae (Lamb and Krug, 1996). There are many strains of avian influenza virus that can cause varying amounts of clinical signs and pathological lesions in poultry. AI viruses can be classified into low pathogenic (LPAI) and highly pathogenic (HPAI) forms based on the severity of the diseases they cause. Most AI virus strains are LPAI and typically cause little or no clinical signs in infected poultry. However, some LPAI virus strain are capable of mutating under field conditions into HPAI viruses. HPAI is an extremely infectious and fatal form of the disease. HPAI can strike poultry quickly without any infection warning signs. Once established, the disease can spread rapidly from flock to flock (Anonymous, 2002). All 16 known influenza A virus hemagglutinin (HA) subtypes can be isolated in avian species. Only the H5 and H7 HA subtypes are considered to be highly pathogenic in avian species, having shown pantropic dissemination within the host (Bosch et al., 1981; Kawaoka et al., 1987). AI viruses can infect chickens, turkeys, pheasants, quail, ducks, geese and guinea fowl, as well as a wide variety of other birds. Migratory waterfowls have proved to be the natural reservoir for this disease. Pigs are permissive to
Both human and avian influenza viruses and have been proposed to be an intermediate host for the genesis of pandemic influenza viruses through re-assortment or adaptation of avian viruses (Peiris et al., 2001).

Prospective research carried out between June to December 2005 in the Laboratory of Immunology and Molecular Biology, The Animal Hospital, Faculty of Veterinary Medicine, Gadjah Mada University, Yogyakarta, Indonesia, provides the first evidence of flies (Musca domestica) had the avian influenza virus H5N1 (Wuryastuti et al., 2005). However, direct evidence that flies may play a critical role on the transmission of avian H5N1 viruses to poultry and even to human is still lacking.

Morbidity and mortality rates are as variable as the signs and are dependent on the species and virus, as well as age, environment, and concurrent infections (Easterday et al., 1997). Clinical signs vary greatly and depend on many factors including the age and species of poultry affected, husbandry practices, and the inherent pathogenicity of the influenza virus strain. Clinical signs may include ruffled feathers, soft-shelled eggs, depression and droopiness, sudden drop in egg production, loss of appetite, cyanosis (purplish-blue coloring) of wattles and comb, edema and swelling of head, eyelids, comb, wattles and hocks, diarrhea, blood-tined discharge from nostrils, incoordination, including loss of ability to walk and stand, pin-point hemorrhages (most easily seen on the feet and shanks), respiratory distress and increased death in a flock. The clinical signs of avian influenza are similar to those of other avian diseases. Avian influenza may be confused with infectious bronchitis, infectious laryngotracheitis, fowl cholera, and the various forms of Newcastle disease (Jacob et al., 2003).

Postmortem lesions vary greatly depending on pathogenicity of the virus, age of the birds, and type of poultry. Lesions may include swelling of the face and area below the beak. Removing skin from the carcass will show a clear straw-colored fluid in the subcutaneous tissues. Blood vessels are usually engorged. Hemorrhage may be seen in the trachea, proventriculus, beneath the lining of the gizzard, and throughout the intestines. The lining of the gizzard may be easily removed. Other areas likely to show swelling and hemorrhages include the muscle along the breast bone as well as in the heart, gizzard fat and abdominal fat (Jacob et al., 2003).

MATERIALS AND METHODS

Sampling of poultry

Sampling was carried out in the Laboratory Pathology and Animal Hospital, Faculty of Veterinary Medicine, Gadjah Mada University, Yogyakarta, Indonesia. The poultry collected between October 2003 to December 2005 on a daily basis from poultry submitted at the Faculty of Veterinary Medicine, Gadjah Mada University. The total sample incorporated about 100 farms consisted of layer chicken (80%), quail (14%), backyard chicken (5%) and ducks and live-bird markets (1%). Those samples originated from provinces in East Java, West Java, Central Java and Yogyakarta.

RESULTS

In the present study, mostly only severe changes in the clinical signs appearances of the poultry were observed. The typical clinical signs manifested by poultry infected with highly pathogenic avian influenza viruses include respiratory signs, rales, edema of the head and face, nervous system disorders (Figures 1-2). Cyanosis of unfeathered skin (especially the combs and wattles), decreased egg production, diarrhea, excessive lacrimation and sinusitis were also observed. In layer
chickens, the ovary may be hemorrhagic.

The swollen and petechial hemorrhages may be seen in the lungs (Figures 3-4) in all poultry examined.

**DISCUSSION**

Avian influenza is an infectious disease of birds caused by type A strains of the influenza virus disease, which was first identified in Italy more than 100 years ago, occurs worldwide (Webster and Kawaoka, 1988; Easterday et al., 1997). Many domestic and wild poultry species are infected with influenza viruses. These include chicken, turkey, ducks, guinea fowl, domestic geese, quail, pheasant, partridge, mynah birds, passerines, psittacine, budgerigars, gulls, shorebirds, seabirds and emu (Easterday et al., 1997; Webster and Kawaoka, 1988). Avian influenza A viruses produce an array of syndrome in poultry, ranging from asymptomatic to mild upper respiratory infections to loss of egg production to rapidly fatal systemic disease (Easterday et al., 1997). Some infected birds show symptoms of avian influenza, while others do not. The types of presenting clinical signs depend on the species and age of the poultry, the strain of AI virus, and accompany bacterial infections (Webster and Kawaoka, 1988; Easterday et al., 1997). Occasionally, the poultry will die without showing any clinical signs of illness (Campbell et al., 1970; Alexander et al., 1993).

In layer chickens, the ovary may be hemorrhagic. The general condition of the poultry and food intake were dramatically changed in all poultry studied. These findings are consistent with those of previous study (Jacob et al., 2003) and may suggest that species adaption and maximal pathogenicity of avian influenza infection for their corresponding species of the poultry (Alexander et al., 1978; Tashiro et al., 1987).

The pathological anatomy lesions in the infected AI chicken (layer and backyard chickens) are quite similar. Some of the gross lesions differences observed among studied poultry may reflect differences in species of the poultry. Swelling of the head and face, cyanosis of unfeathered skin (especially the combs and wattles), and swollen and petechial hemorrhages of the foot and muscles, especially breast and thigh muscles, abdominal fats and proventriculus were only observed in the chicken, but not in the quails. The quails had a severely decreased in the general condition and food intakes.

It was reported that systemic replication of avian influenza virus in chicken and turkey is largely defined by the presence of multiple basic amino acids at the hemagglutinin cleavage site (Senne et al., 1996, Wood et al., 1993; Klenk and Rott, 1988). Despite this molecular conformity among H5 and H7 high pathogenic avian viruses, considerable variation has been observed in the pathogenicity and transmissibility of these viruses in different avian species, even within the avian order Galliformes. For example, the A/turkey/Ontario/7732/66 (H5N9) influenza virus is moderately pathogenic for the chickens and quail, highly pathogenic in turkeys, and apathogenic in pheasants, pigeons, and ducks (Slemons and Easterday, 1972). Therefore, because of the differences in virulence avian influenza viruses demonstrate in domesticated avian species, it is important to consider possible ramifications of interspecies transmission of influenza viruses when different poultry species are commingled.

**CONCLUSION**

The highly pathogenic avian influenza is suspected with any flock where sudden deaths follow severe depression, in acceptance, and a drastic decline in egg production. The present of facial edema, swollen and cyanotic unfeathered skin, especially combs and wattles, and petechial hemorrhages on the internal membrane surfaces, foot and muscles, especially breast and thigh
muscles increases the likelihood that the disease is highly pathogenic avian influenza. Highly pathogenic avian influenza H5N1 infection is easily confused with primarily Newcastle disease because the disease clinical signs (torticolis and curled-toe paralysis) and postmortem lesions (petechial hemorrhages of the internal organs) are similar. However, in an area where AI is endemic, such as during an outbreak, sound presumptive diagnoses can be made by flock history, clinical signs, and gross lesions.

REFERENCES