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Epidemiology and Pathogenesis of Avian Influenza Type a Virus Infection in Poultry, A Review

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Abstract

Avian influenza (Bird flu) is a highly infectious disease caused by an Orthomyxovirus of the family Orthomyxoviridae. Avian influenza viruses are classified in the genus influenza virus A that infects wide variety of domestic and wild animal species. Aquatic birds are the natural hosts of avian influenza viruses as asymptomatic carriers. Mode of transmission of this virus in poultry flock is through ingestion, inhalation and mechanical transmissions and its clinical signs are variable. Avian influenza is distributed worldwide. According to disease severity avian influenza is classified into two forms: highly pathogenic avian influenza (HPAI) also known as fowl plague, and low pathogenic avian influenza (LPAI). HPAI viruses are highly virulent representing the greatest concern for public health and economic importance and categorized as List a disease by OIE. Diagnosis mainly depends on isolation and identification of viruses. Serological tests and RT-PCR are also used. The disease is not treatable except trials for secondary microbial complications. Biosecurity is the most useful method for prevention and control.

Keywords: Avian; Influenza; Pathogen; Virus

Abbreviations: HPAI: Highly Pathogenic Avian Influenza; LPAI: Low Pathogenic Avian Influenza; HPAIV: Highly Pathogenic Avian Influenza Virus; HP: High Pathogenicity; LP: Low Pathogenicity; HA: Hemagglutinin; NP: Nucleoprotein; M: Matrix; HA: Hemagglutinin; NA: Neuraminidase; CNS: Central Nervous System; AI: Avian Influenza.

Introduction

Avian influenza, which is caused by influenza a viruses, can affect variety of domestic and wild bird species. Avian influenza viruses are the causative agents of periodically occurring disease outbreaks in birds with high damage to the poultry industry. In addition, these viruses provide the genetic pool from which human and other mammalian influenza viruses emerge. Avian influenza has therefore a high impact on both animal and human health.

The best known disease caused by an avian influenza virus is fowl plague. Fowl plague was first reported in 1978 and in 1901, causing severe loses in poultry, and in 1955 it was identified as avian influenza virus. From the 1970s onward surveillance indicates the ubiquitous presence of avian influenza virus in water fowl and the risk these birds posed to commercial chicken industries. In 1997 a highly pathogenic avian influenza virus (HPAIV) emerged in Hong Kong that had killed close to 150 million birds in Asia up to the beginning of 2005 [1]. Type an influenza viruses are notorious for their ability to undergo antigenic change with time and to give rise to new viral subtypes. When different

subtypes infect the same animal host genetic recombinants may occur: Some of the genes from one virus strain may replace those within another strain, giving rise to a new viral subtype.

Many combinations of hemagglutinin and neuraminidase antigens in influenza A viruses are represented in isolates from avian species, particularly from water fowl. Influenza sub types are distributed worldwide and are frequently recovered from clinically normal birds. Outbreaks of severe clinical disease, usually caused by subtypes expressing H5 and H7 determinants, occur periodically in chicken and turkeys. In these species acute infection is often referred to as fowl plague or highly pathogenic avian influenza and categorized as List a disease by OIE [2].

Avian influenza is classified by the World Organization for Animal Health (OIE) in to two forms, High pathogenicity (HP) and low pathogenicity (LP), based on virulence in chicken. H7 is one of the two economically important avian influenza virus subtypes because historically all highly pathogenic avian influenza viruses have been either H7 or H5 subtype and it is among the most common subtype in commercial poultry in the world. In numerous cases the high pathogenic form mutated from a low pathogenic H7 or H5 virus that was circulating in chickens or turkeys. However, not all H7 LPAI viruses become high pathogenic [3]. Influenza A viruses are highly contagious pathogens that have been isolated from a wide variety of animals including man, birds, swine, horses, minks, seals and most recently from cats and dogs. Influenza A viruses are rarely known to cross species barriers, however their interspecies transmission has always been a major concern. Although determinants of inter species transmission are still not fully identified, many studies showed that the compatibility between the hemagglutinin (HA) protein of the virus and its corresponding receptor on the host cell is essential for establishment of an infection in a specific host [4].

The objective of this paper is:

- To review the epidemiology and pathogenesis of avian influenza type A virus infection in poultry.

A General Overview of Avian Influenza Type a Virus Infection In Poultry

Etiology

Avian influenza is caused by an orthomythovirus of the family orthomythoviridae. An orthomythoviridae contains 5 genera, 3 of which contain influenza viruses. Influenza virus A is the main cause of avian influenza in poultry [5].

Classification: Based on antigenicity, there are three antigenically distinct types of influenza viruses. These are Type A, Type B and Type C influenza viruses. The type specificity is determined by antigenic nature of nucleoprotein (NP) and matrix (M) antigens which are closely related among all influenza [6].

Type an influenza viruses are further divided into subtypes based on the antigenic relationships of the surface glyco proteins, hemagglutinin (HA) and neuraminidase (NA). To date, 16HA subtypes (H1-H16) and 9NA subtypes (N1-N9) have been recognized. Each virus has one HA and one NA antigen apparently in any combination. All influenza subtypes in the majority of possible combinations have been isolated from avian species. Thus far, only virus of H7, H5 and H10 subtypes have been shown to cause highly pathogenic avian influenza (HPAI) in susceptible species [7]. Based on pathotype (pathogenicity), avian influenza viruses from poultry are classified into two pathotypes, highly pathogenic and low pathogenic avian influenza. These terminologies are originally based on lethality in experimentally inoculated chicken [8].

To date, apart from the H10 isolates, only viruses of H5 and H7 subtypes have been shown to cause HPAI. It appears that most of these viruses arose by mutation after wild bird reservoir. Highly Pathogenic avian influenza viruses arose by mutation after wild bird reservoir. Most HPAI viruses appear to have arisen as a result of spontaneous duplication of purine triplets, which is the insertion of basic amino acids at the cleavage site. This most likely occurred due to transcription error by the polymerase complex [9]. The factors that bring about mutation from low pathogenic to high pathogenic avian influenza are not known. However, it can be reasonably assumed that the wider the circulation of LPAI in poultry, the higher the chance that there will be a mutation to HPAI. In some cases, low pathogenic avian influenza (LPAI) viruses of the H5 or H7 subtypes circulated for very long periods of time without mutating to highly pathogenic form Sfakianos et al., [10].

Morphology and Chemical Composition of Avian Influenza Viruses: Virions are typically spherical to pleomorphic but can be filamentous. Individual virions range in diameter from 80-120 nm, but the filamentous forms can have lengths up to several hundreds of nm [6]. A helical nucleocapsid is enclosed within the viral envelope. The surface is covered by two types of glycoprotein projections, rod shaped trimer of HA and mushroom shaped trimers of NA. The virions may be disintegrated with detergents, resulting in the release of spikes, which retain their respective activities. The HA is responsible for the attachment of the virion to cell surface receptors (Sialyl oligosaccharides) and is responsible for the hem agglutinating activity of the virus. Neuraminidase enzyme activity is responsible for the release of new virus

from the cell by its action on the neuromeric acid in the receptors [7].

All influenza viruses have eight different gene segments that encode at least 10 different viral proteins. The structural proteins in the mature virion can be divided into the surface proteins that include hemagglutinin (HA), neuraminidase (NA), membrane ion channel proteins and the internal proteins including nucleoprotein (NP), matrix protein and polymerase complex composed of the polymerase basic protein 1, polymerase basic protein 2 and polymerase acidic protein. Two additional proteins produced by influenza viruses are nonstructural protein 1 and nonstructural protein 2, which is also known as the nuclear export protein. The nonstructural protein 2 is primarily found in host cells, but some protein can be found in the virion [11].

Influenza virions are composed of 0.8-1.1% RNA, 70-75% protein, 5-8% carbohydrates and 20-24% lipids. The lipids are located in the viral membrane; most are phospholipids with smaller amount of cholesterol and glycolipids. Several carbohydrates including ribose, galactose, mannose and glucosamine are present in the virion mainly as glycoprotein or glycolipids. The composition of the lipid and carbohydrate chains linked to glycoproteins or glycolipids of the viral membrane are determined by the host cell [7].

Antigenic Variation: Influenza A viruses can undergo antigenic variation in two ways, antigenic drift and antigenic shift. Antigenic drift involve minor point mutations resulting in RNA replication errors. The mutations will be expressed in the new virions produced. Although such mutations may be detrimental, on occasion one might have a subtype change in shape (that is the structural shape has "drifted") so they are not recognized by the host's immune system [12].

Antigenic shift occurs only among influenza A viruses in two ways. In the first, influenza A viruses of two different subtypes simultaneously infect the same host allowing reassortment, of exchange of viral RNA segments in the host's cells. The second possible mechanism involves the direct transmission of influenza virus from avian or other animal species to humans with subsequent adaptation by mutation to the new human host. Genetic reassortment between human and avian viruses is suggested as the mechanism by which new human pandemic strains arise [13].

Epidemiology of the Disease

Incidence and Distribution: Influenza viruses are distributed throughout the world in many domestic birds, including turkeys, chicken, guinea fowl, quail, pheasants, geese and ducks and in wild bird species. However, incidence and distribution of avian influenza varies greatly with geographic region, country, species and age of birds, time of year and the environment or agricultural system occupied [7].

The primary introduction of low pathogenic avian influenza viruses into a poultry population is a result of wild bird activity, usually water fowl, but gull and sore birds have also been implicated. Similarly backyards reared in the Italy have been shown to harbor the same viruses isolated from water fowl during the same period. In considering prevalence and distribution of influenza viruses in avian species, it becomes clear that many viruses circulate in birds throughout the world [7].

Most often the wild birds that are host to the virus do not get sick, but they can spread influenza to other birds. Infection with certain avian influenza A viruses (for example, some H5 and H7 strains) can cause wide spread disease and disease among some species of domestic birds (Center for Disease Control and Prevention; http://www.cdc.gov). Over the past two decades, the incidence of avian subtypes of influenza a virus directly infecting humans has dramatically increased. Land use changes, cultural practices and worldwide outbreaks have contributed this increase [14].

Host Range and Reservoir Host: The natural reservoir of influenza a viruses is aquatic birds, in which the viruses appear to have achieved an optimal level of host adaptation and do not cause disease. The exception is the H5N1 strain which had already crossed to humans in 1997 (in Hongkong) and those crossing have gradually increased in frequency and distribution in recent years. Avian influenza viruses have been shown to infect birds and mammals. Generally speaking the former are infected more readily and efficiently than the later and the interspecies and interspecies transmission with in the class Mammalia. One of the main factors that influence susceptibility to infection is the receptor conformation on the host cells [15].

Influenza viruses have been shown to infect great variety of birds and highly pathogenic avian influenza was a disease of domesticated birds and those wild birds usually only harbored the low pathogenic avian influenza. It seems likely that part of influenza gene pool is maintained in shore birds and from which the predominant number of isolated influenza viruses are of subtype different from those isolated from ducks. Pigs have an important role in the ecology and epidemiology of influenza viruses and are regarded as "mixing vessel" for the introduction of reassorted viruses into the human population, primarily because of their susceptibility to human and avian viruses [16].

Risk Factors: There are several recognized risk factors for the introduction of influenza A viruses into a domestic

poultry. These include 1) direct access of poultry to wild birds infected with avian influenza viruses, especially wild ducks. Outbreaks of multiple subtypes of avian influenza occurred routinely in the fall when infected ducks had the opportunity to contact with turkeys. Once the virus was introduced to a turkey farm, the virus could become adapted to turkeys and spread to other turkey farms by the movement of infected birds and contaminated materials. 2) Infection through avian influenza virus contaminated drinking water. 3) Exposure of turkeys to pigs infected with the swine influenza virus. Turkeys are susceptible to swine influenza viruses and having turkey farm and swine farm in a close proximity is a risk factor for the introduction of swine influenza to turkeys. 4) Live bird marketing system is a risk factor for the introduction of avian influenza virus into commercial poultry. Domestic water fowl, primarily ducks are often raised on ponds where exposure to wild birds, including ducks is common. This provides high risk for domestic ducks to be infected with avian influenza [17].

Modes of Transmission: Influenza Type a viruses normally seen in one species across over and cause illness in another species. It is transmitted by direct contact between infected and susceptible birds or indirect contact through aerosol droplets or exposure to virus contaminated fomites. Thus avian influenza viruses are readily transported to other premises by people (contaminated shoes and clothing) and equipments shared in production, live haul and live bird marketing [18].

Source of infection for the initial introduction of influenza viruses in to commercial poultry flocks include other species of domestic poultry, exotic captive birds, wild birds and other animals. Brocken contaminated eggs may infect chicks in the incubator. HPAI viruses have been recovered from the egg shell and internal egg contents. Some avian influenza virus strains can be transmitted to mammals by direct or indirect contact; close contact with dead or sick birds seems to be the principal means of transmission to humans [19].

Immunity: Immunity to influenza occurs through a number of steps; initially, a large cytokine response occurs and characterized predominantly by IL-2, IL-6 and interferon gamma production. This leads to extensive local inflammation with neutrophils and macrophages infiltrating the sub epithelium of the respiratory tract. Within the alveolar macrophages and pneumocytes, MHC-I up regulation leads to antigen presentation of the hemagglutinin and other sub capsular proteins. This eventually leads to natural killer cell destruction of infected cells and the development of neutralizing antibodies largely against HA by day 14 of infection [14].

Method to produce resistance to avian influenza viruses is through active or passive immunity. Principally against avian influenza virus HA, and to a lesser extent, the NA, but such protection for the first one to three weeks post hatching, while active immunity was effective for longer periods of time [11].

Active immunity develops via infection with avian influenza virus as well as immunization with vaccines that elicits a humeral antibody response at both systemic and mucosal level. This includes a systemic IgM response by 5 days post infection, followed shortly by an IgG response. The intensity of antibody response against surface proteins varies with bird species. While passive immunity studies on the protection by maternal antibody to homologous HA or NA have not been reported. Based on evidence available for other avian pathogens, protection against clinical signs and death from homologous avian influenza viral challenge is probable for the first two weeks after hatching [20].

Pathogenesis

The pathogenesis of avian influenza virus varies widely depending on strain of virus, age and species infected concurrent infections and husbandry. Avian influenza viruses can infect wide range of domestic and wild birds including chicken, ducks, turkeys, geese, quail, pheasants and migratory birds. In these natural hosts, influenza viruses replicate in the gastro intestinal tract and are secreted in large amounts in to the feces [21].

Avian influenza virus hemagglutinin (HA) adsorbs to host cell receptors containing sialic acid bound to glycoproteins, thus initiating receptor mediated endocytosis. In these endosomes, low pH dependent fusion occurs via HA mediated fusion of viral envelope with the endosome membrane. Proteolytic cleavage of HA into HA1 and HA2 is an essential pre-requisite for fusion and infectivity. The viral nucleocapsid is transported to the nucleus where the viral transcriptase complex synthesizes mRNA [22].

Determinants of Pathogenicity of Avian Influenza Viruses:

- **Viral Factors:** Abroad tissue tropism and the ability to replicate systemically are hall marks of these viruses. The most important and well-studied molecular correlate of these properties reside in the cleavability of the HA precursors of glycoprotein [23]. The viral factors that determine the pathogenesis and virulence of influenza viruses include ability to bind to host cells, ability of virus shedding, escape from immunosurveilance by evolution of antigenic variation or recombination with different virus strains from zoonotic disease and modulation of the immune response to attenuate effective host defense mechanism (Influenza report; http://www.

influenzareport.com).

- **Host Factors**: The virulence of highly pathogenic avian influenza viruses are clearly influenced by the specific host. Two variants of H5N1/97 virus one of which was isolated from a human patient with mild respiratory illness and the other form a fatal human case, displayed similar differential pathogenicity in mice [20].

Virions with uncleaved hemagglutinin are noninfectious. Hemagglutinin cleavability is dependent on its primary structure that the site where cleavage occurs and the presence of the right proteases in the target tissues that can carry out that cleavage. In epithelial cells lining the respiratory and intestinal tracts, the hemagglutinin of all incoming avian influenza viruses are cleaved by host proteases, there by activating its fusion activity and allowing its entry. However, in other tissues, only hemagglutinins of virulent viruses are cleaved, leading to systemic disease and death [24].

The pathogenesis of avian influenza is quite different from that in mammals. Infection with the most virulent strains is characterized by viremia and multifocal lymphoid and visceral necrosis, leading to pancreatitis [24]. The host factors that determine the pathogenesis and virulence of influenza viruses include presence of target receptors on host cells, availability of enzymes in host cells which are essential for viral entry and replication, state of immunocompetence of the individual host and ability of the immune system to control the viral replication effectively without causing serious collateral damage for the host by its inflammatory response (Influenza report; http://www.influenzareport.com).

Persistence Infection: In poultry, first, the process begins by inhalation or ingestion of infective mild pathogenic or highly pathogenic avian influenza (HPAI) virions. Multiple replication cycle occur in respiratory and/or intestinal tract with release of infectious virions. In poultry the nasal cavity is the major site of initial replication. Second, the virions invade sub mucosa entering capillaries. The virus replicates with in endothelial cells and spread via the vascular/lymphatic systems to infect and replicate in variety of cell types in visceral organs, brain and skin. Clinical signs and death are due to multiple organ failure. Damage caused by avian influenza virus is the result of one of the following three processes such as direct virus replication in cells, tissue and organs, indirect effects from production of cellular mediators (cytokines) and ischemia from vascular thrombosis. Third, for the mild pathogenic avian influenza viruses, replication usually limited to the respiratory or intestinal tracts. Illness or death is most often from respiratory damage, especially if accompanied by secondary bacterial infections. Occasionally, the mild pathogenic avian influenza viruses spread systemically, replicating and causing damage in kidney tubules, pancreatic acinar epithelium and other organs with epithelial cells having trypsin-like enzyme [11].

Clinical Findings and Lesions

Clinical signs, severity of disease and mortality rates vary depending on avian influenza virus strain and host species. Low pathogenic avian influenza (LPAI) viruses typically produce respiratory signs such as ocular and nasal discharge & swollen infra orbital sinuses. Lesions in the respiratory tract include congestion and inflammation of trachea and lungs. In layers and breeders there may be decreased egg production or fertility, ova rupture and mucosal edema. High pathogenic avian influenza (HPAI) viruses cause severe systemic disease with high mortality in chicken, turkey and other gallinaceous birds. In per-acute cases, clinical signs or gross lesions may be lacking before death. However, in acute cases, lesions may include cyanosis and edema of head, comb, wattle; edema and discoloration of shanks and feet due to subcutaneous echymotic hemorrhages; petechial hemorrhages on visceral organs and in muscles; and blood tinged oral and nasal discharges. In severely affected birds, greenish diarrhea is common. The location and severity of microscopic lesions are highly variable and may consist of edema, hemorrhage, necrosis in parenchyma cells of multiple visceral organs, skin and central nervous system (CNS) [25].

Diagnosis

Clinical diagnosis is usually not possible except in an epidemic. Virus isolation is essential not only to establish the cause of an outbreak but also to assess objectively the virulence of the causative virus [24]. A definitive diagnosis requires viral isolation and identification or the demonstration rising antibody titer by viral neutralization tests, soluble antigen fluorescent antibody test, agar gel precipitation or ELISA technique [18].

Methods for virus isolation include 1) Candle 9- to 11-day-old embryonated fowl's eggs to check embryo viability. Mark the shell of the egg with a pen to delimit the air sac. With a manual or electric device, drill a small hole just above the air sac. 2) Record the identification number of the sample, the passage number (1° or 2°), the kind of sample (lung, cloacal swabs, etc.) and the date of inoculation on five eggs. 3) Inoculate 0.1–0.2 ml of clarified supernatant obtained from the tracheal and cloacal swabs or from the organ homogenate into the allantoic cavity of each of the five 9- to 11-day-old embryonated fowl's eggs. 4) Seal the eggs with glue or wax. 5) Incubate the inoculated eggs at 37°C for 7 days. Candle the inoculated eggs daily to check embryo viability. 6) Test the allantoic fluid of eggs containing dead embryos for haemagglutinating (HA) activity. 7) If HA activity is detected, identify the HA agents by means of the haemagglutinating

inhibition (HI) test. 8) After 7 days, chill the remaining eggs in a refrigerator (4°C) to end the first passage. The following day, open the eggs using sterile techniques under a laminar flow cabinet. Collect approximately 10 ml of the allantoic fluid. 9) Test the allantoic fluid for the presence of HA activity by the "rapid HA test". 10) If bacteria are present, the fluids must be passed through a 450-nm membrane filter. 11) A sample giving a positive result in the rapid HA test must be tested further [5].

The use of diagnostic methods based on molecular technology has improved substantially over the last decade; consequently, laboratory tests for the identification and characterization of avian influenza (AI) viruses have become available. Such tests may be used to detect the AI viral genome directly from clinical specimens as well as to generate data on the molecular characteristics of an isolate or of viral RNA present in a sample collected from an infected animal. In addition, the current definitions of highly pathogenic AI are based on the results of conventional and molecular techniques, thus, the latter are among the official methods used to identify virulence factors and to confirm the presence of HPAI viruses in laboratory specimens. Such as RT-PCR, polymerase chain reaction and real-time PCR techniques [5].

As differential diagnosis, Low pathogenic avian influenza must be differentiated from other respiratory diseases or causes of decreased egg production including acute to sub-acute viral diseases such as infectious bronchitis, infectious laryngotracheits, lentogenic New castle disease, and infections with other paramyxoviruses; bacterial diseases such as mycoplasmosis, infectious coryza and respiratory form of fowl cholera and fungal diseases such as aspergilosis. Highly pathogenic avian influenza must be differentiated from other causes of high mortality such as velogenic new castle disease, and sever water depression [25].

Treatment

Avian influenza (Bird flu) control activities there is no specific treatment. Broad spectrum antibiotics used to control secondary bacterial invaders and increasing house temperature may help to reduce mortality in poultry. Vaccination with autogenous virus or a virus of the same hemagglutin in type used as prophylaxis [26]. As with any viral infections, no chemotherapeutic agent can cure influenza in human beings. Amantadine hydrochloride is used as prophylactic treatment for some high risk individuals such as elderly or persons with little natural defenses [27].

Control and Prevention

Avian influenza (Bird flu) control activities operate at international, national and local levels. At the international level, countries must be willing to report disease out breaks. The fowl plague form of avian influenza appears in list A of the International Animal Health Code of the Office International des Epizootics; the disease is there by notifiable and restrictions apply to the movement of birds or avian products. At the national level, many countries have regulations aimed at preventing the introduction and spread of virus; these are often primarily concerned with new castle disease. Policies usually involve trade embargoes to guard against importation of infected birds or avian products from countries not declared "virus free". At the local farm based level, efforts are aimed at preventing virus introduction into chicken and turkey flocks from wild birds. Today, commercial chicken facilities are always made wild bird proof [24].

There are six basic tools used to control and prevent avian influenza. These include farm biosecurity, stamping out, cleaning and disinfection, movement management and vaccination. None of these measures used alone is likely to lead to elimination of infection. Control and eradication of AI also depend on a fully functional surveillance system that allows early detection of infection and disease. This requires a well-resourced and trained veterinary service. Control programs should be backed by public education and behavioral change campaigns to provide accurate and timely information on the nature of the disease to groups at risk. It is important that programs fully engage all stakeholders to ensure success [12].

Public Health and Economic Importance

Avian influenza (Bird flu) mainly infects birds, but is concern to humans, who have no immunity against it. The virus that causes this infection in birds can mutate and easily infect humans and potentially start a deadly worldwide epidemic. Pandemic influenza virus has its origin in avian influenza viruses. The highly pathogenic avian influenza virus subtype H5N1 is already panzootic in poultry, with attendant economic consequences. It continues to cross species barriers to infect humans and other mammals, often with fatal outcomes. Therefore, H5N1 virus has rightly received attention as a potential pandemic threat [28].

Avian influenza virus which was responsible for fatal human disease has the genetic formula H5N1. Meanwhile, a second avian influenza virus (H9N2) has been isolated from humans. Both viruses are genetically linked to influenza viruses found in quail. A third avian virus, H7N7, can also infect humans [29].

The biggest threat resulting from demonstrations of direct natural infections of humans with avian influenza viruses is that pandemic viruses could emerge as a result without an intermediate host. There are two mechanisms

by which this could occur: by genetic reassortment or by progressive adaptation the first cause could occur if a person was simultaneously infected with avian influenza virus and human influenza virus. The second mechanism by which the generation of a pandemic virus may occur is through progressive adaptation of virus entirely of avian origin. Recent studies on the genome of H1N1"Spanish flu" influenza virus, which affected a human beings at the beginning of 20th century, have resulted in the speculation that this virus was entirely of avian origin and not generated by re-assortment [30].

Variations in cell receptor specificity on respiratory tract epithelium may account for some of the differences in influenza virus transmission and efficient replication. Avian respiratory epithelium has predominantly α- 2, 3 linkage while human respiratory epithelium has predominately α-2, 6 linkage. Two cases are exceptions with presumed exposure through consumption of raw duck blood and organs, and defeathering H5N1 infected dead swans [9].

The highly pathogenic strain of the H5N1 avian influenza virus was first isolated and characterized in a domestic goose in the southern Guangdong of china in 1996. The following year, the first H5N1 HPAI outbreak occurred in domestic poultry in Hongkong, resulting in the culling of 1.5 million chickens in an effort to control and eliminate the disease [31]. It is likely that H5N1 virus infection among domestic poultry has become endemic in certain areas and that sporadic human infections resulting from direct contact with infected poultry and/or wild birds will continue to occur (Center for Disease Control and prevention; http://www.cdc.gov) [32].

The full name of H5N1 avian influenza virus is more revealing of the virus that was initially isolated by scientists. The circulating H5N1 virus that is the current source of fear throughout the world is officially named A/Goose/Guangdong/1/96(H5N1). The deadly characteristics of this virus in humans lead to the categorization of the virus into the highly pathogenic category of influenza virus [10].

In addition to the human deaths that have occurred due to H5N1 virus and largely as a result of that, the highly pathogenic avian influenza panzootic caused three notable economic and social impacts. These are market shocks, negative consequences for lively hood as a result of the disease and the control process applied to control it and changes to the structure of the poultry market chains [33].

Conclusion and Recommendations

Avian influenza (Bird flu) is a public health threat that has the potential to cause serious illness and death in poultry as well as in humans. Understanding its epidemiology, pathogenesis, transmission and clinical features and preparing for the prevention and management of its outbreak will help to avoid its potential devastating consequences [34-37].

Based on the above conclusion, the following recommendations are forwarded:

- Authorities should be notified immediately of any suspicious cases of Highly Pathogenic avian influenza (HPAI).
- While waiting for the authority or a confirmed diagnosis, all suspect animals should be quarantined.
- Should highly pathogenic avian influenza (HPAI) be confirmed by diagnosis?
- Individual and organizational awareness creation about economically devastating nature of the disease is required.
- To control outbreak of HPAI the premises must be thoroughly cleaned and disinfected and there should be proper destruction of all exposed cadavers, litter and animal products.

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