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Avian Influenza: A Repeatedly Re-emerging Havoc

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Abstract

Avian influenza virus, a member of genus influenza A virus within the family Orthomyxoviridae, which is also called as Fowl plague as causes epidemic leading to high mortality. The disease primarily affects domestic poultry and wild birds but can occasionally cause disease in other animals and humans. This document is an attempt to briefly describe the important aspects of the disease viz. the virus, transmission, clinical signs, diagnosis, vaccination, prevention and control.

Introduction

Avian influenza commonly known as bird flu or fowl plague, is a viral disease of birds, other animals and humans. Disease is caused by influenza A virus. Virus capable of causing disease is of 2 types: Virus with low virulence called Low pathogenic strain and with high virulence called High pathogenic strain inflicting up to 100% mortality. The disease is notifiable to World Organization for Animal Health (WOAH).

Etiology

Avian influenza belongs to Orthomyxoviridae family, is an enveloped RNA virus (negative sense), composed of 8 segments of ssRNA proteins associated with RNA genome to form nucleoprotein RNA-polymerase complex. Virions are spherical in shape. The matrix surrounding genome complex is enveloped in lipid membrane, covered by surface projections/spikes. Surface spikes are glycoproteins, have hemagglutinin (HA) and neuraminidase (NA) activity. Hemagglutinin is a rod-shaped trimer while NA is a mushroom shaped tetramer. Hemagglutinin is responsible for attachment of virion to cell surface receptors while NA is responsible for release of new virus from the cell. Type A influenza viruses have been isolated from birds only.

Host

Domestic birds including chicken, turkey, duck, geese, guinea fowl, pheasant, quail and feral birds. Ducks yielded more viruses than any other group (they don't suffer from clinical signs), instead they act as reservoirs and carriers of AI and spread it to chickens and turkeys which suffer clinically.

Transmission

In field conditions, influenza viruses are released in nasal secretions and feces of infected birds i.e. mode of transmission includes both direct and indirect contact. Feco-oral route is the main route of spread.

Pathogenesis

Avian influenza virus adsorbs to glycoprotein receptors containing sialic acid on cell surface. The virus enters the cell by receptor-mediated endocytosis. The tissue tropism of a virus is involved in the pathogenicity. The basis for tissue tropism of a virus is receptor specificity. Receptor recognition by virus is important factor in both tissue tropism and pathogenesis. Since no cellular receptors have been identified for influenza virus, role of virus receptor interactions in the disease is unclear. Recent studies at molecular level reveal that infectivity depends on post translational cleavage of HA molecule. The cleavage is brought by host proteases and takes place at the cleavage site. The susceptibility of HA molecule to cleavage by host proteases depends on number of basic amino acids at the cleavage site. The ability of proteases in host cell to achieve cleavage of HA molecule is important in deciding the extent of virus replication and in the production of infective virus particles. The HA of low/moderately virulent influenza viruses have only a single amino acid Arginine at cleavage site. Hence these viruses are cleaved in tissues where trypsin like enzymes are found i.e. respiratory and digestive tract and pathogenicity limited to these systems only. Highly pathogenic viruses possess HA with multiple basic amino acids at the cleavage site. Therefore, their HA can be cleaved by proteases found throughout the body, generalized disease and death. Other factors may also influence virulence e.g. LP virus isolates possess a glycoprotein site in the cleavage region and it blocks efficient cleavage. A single mutation removing this glycosylation site has resulted in HP strain. Concurrent bacterial infections also play a major role in aggravating effect of LP strain viruses.

Clinical Signs

Highly virulent strains cause sudden death without prodromal symptoms. If the birds survive for more than 48 hrs, there is cessation of egg laying, respiratory distress, lacrimation, sinusitis, diarrhoea, edema of head, face and neck and cyanosis of unfeathered skin particularly of comb and wattles. Less virulent strains cause considerable loss particularly in turkeys because of anorexia, decreased egg production, respiratory distress and sinusitis

Lesions

With less pathogenic viruses, mild lesions observed in the sinuses characterized by catarrhal, fibrinous, serofibrinous, mucopurulent or caseous inflammation. In case of highly pathogenic viruses, no prominent lesions seen because birds die very quickly before gross lesions can develop. Histopathological lesions include edema, congestion, hemorrhages and foci of perivascular lymphoid cuffing mainly in the myocardium, spleen, lungs, brain, wattles, liver and kidneys. However, none of the lesions are pathognomonic for the disease.

Diagnosis

Clinical diagnosis is usually not possible except in an epidemic because of the variability of clinical signs. Virus isolation is essential to establish the cause of an outbreak and also to assess virulence of the causative virus. Virus is best isolated from cloacal swabs. Specimens are inoculated into the allantoic cavity of 8-10 day old embryonated eggs and presence of virus is indicated by hemagglutinating activity using allantoic fluid and chicken RBCs. The cleavability of HA of isolates is assessed by the production of plaques in the cell cultures that are permissive for virulent viruses but not permissive for avirulent viruses. Isolates are typed/subtyped using HA and NA inhibition assays. To assess the virulence of the isolates, intracerebral and intravenous pathogenicity

indices are determined using 1day old chicks and 6-week-old chickens, respectively.

Treatment

Presently, no practical, specific treatment exists for avian influenza virus infections in commercial poultry. Amantidine has been shown experimentally to be effective in reducing mortality, but the drug is not approved for food animals and its use rapidly gives rise to amantadine-resistant viruses which compromise public health. Supportive care and antibiotic treatment have been employed to reduce the effects of concurrent bacterial infections.

Vaccination

Not currently available in India. India follows a strict "Stamping-out policy" i.e. culling of infected and in-contact birds, disinfection, movement control and surveillance.

Control

Avian influenza control activities operate at international, national and local levels.

1. At International level, countries must be willing to report disease outbreaks. Fowl plague form of avian influenza appears in list-A of International Animal Health Code of the World Organization for Animal Health. The disease is thereby notifiable and restrictions apply to movement of birds or avian products.

2. At the national level, many countries have regulations aimed at preventing the introduction and spread of virus. Policy usually involves trade embargoes to guard against importation of infected birds or avian products from countries not declared virus free. To minimize secondary spread, strict hygienic measures are required which include cleaning and disinfection, an interval between slaughter and repopulation and controlled movement of humans and animals.
3. At the local farm-based level, efforts are aimed at preventing virus introduction into chicken and turkey flocks from wild birds.

Zoonotic Potential

Avian influenza can transmit from birds to humans, though it is relatively rare. Certain strains like H5N1 and H7N9 are known to infect humans and can cause severe respiratory disease with high mortality. Transmission to humans occurs through direct contact with infected poultry or their excretions, exposure to contaminated environments, inhalation of aerosolized virus particles. Human to human transmission is rare and limited but the risk exists if the virus undergoes genetic changes. People at higher risk include poultry workers, Veterinarians and slaughter house workers. Human cases have occurred in India but they are very rare and sporadic. Prevention includes use of personal protective equipment (PPE), proper cooking of poultry products, surveillance and control in birds.

Conclusion

Avian influenza is a highly contagious viral disease with significant impacts on poultry health, economy and public health. Effective control relies on strict biosecurity, surveillance, early diagnosis, culling of infected flocks and regulated vaccination strategies. Due to its zoonotic potential, avian influenza remains a major concern under the One Health approach, requiring coordinated efforts between Veterinary and Human health sectors to prevent outbreaks and minimize risks to humans.