

Review Article

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A Review on Infectious Bursal Disease - Major Concern in Poultry

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ABSTRACT

Infectious bursal disease is an acute and highly contagious viral disease of mostly young chickens caused by a non-envelope double stranded RNA virus belonging to a family Birnaviridae and is the second considerable threat among infectious poultry diseases. The enormous economic losses associated with the disease is due to high mortality, decreased performance, immune suppression that leads to increase susceptibility to other diseases and decrease response to vaccination. The target organ of infectious bursal disease virus (IBDV) is the bursa of Fabricius at its maximum development, which is a specific source for B lymphocytes in avian species. The observed clinical signs during IBD infection include high mortality, unsteady gait, ruffled feathers, urate containing diarrhoea and sudden death. The post-mortem finding includes haemorrhages in the leg and breast muscles, enlarged, edematous and hyperaemic bursa with bloody or mucoid contents. RT-PCR/RFLP is a very useful and rapid method for characterization and identification of existing and evolving strains of IBDV. Only supportive therapy, immunobooster medicines with vaccination and maintaining bio securities may combat mortalities.

Keywords

Infectious bursal disease,
Birnaviridae,
Haemorrhage, B lymphocyte

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Introduction

The term poultry refers to rearing of chickens, ducks, geese, turkeys and domestic fowls. They are reared for their flesh, eggs and feathers. Poultry farming have been gaining much importance due to growing demand for poultry products as having high food values in terms of rich sources of protein, vitamins and

minerals. Among the poultry products, chicken is the widely accepted meat in India and in the world and being used by the people regularly as a supplementary food.

Between 1970 and 2005 poultry meat and eggs increased faster than beef, veal and pig meat (Windhorst, 2006). Apart from lot of opportunities in this field there are some

barriers as well in terms of climatic hazards, outbreak of bird flu, outbreak of different kinds of bacterial and viral diseases, vaccine failure if not proper maintaining cool chain, availability of good qualities food etc. Among these barriers one of the viral disease namely Infectious Bursal Disease (IBD) is havoc for poultry men (Siddique *et al.*, 1987). This disease was discovered in town Gumboro (Cosgrove, 1962). IBD is the second considerable threat among infectious poultry diseases (Alexander, 1996). This disease is extremely contagious in infected flocks, morbidity is high, with up to 100 percent seroconversion after infection, whilst mortality is variable (van den Berg *et al.*, 2000). The enormous economic losses associated with the disease is due to high mortality, decreased performance, immune suppression that leads to increase susceptibility to other diseases and decrease response to vaccination (Abdu *et al.*, 2001; Khan *et al.*, 2007). Experts documented that IBD was one of the disease which was not affected by weather (Yunus *et al.*, 2009). The incidence of IBD decreased with increase in flock size. This threat is at peak during third to fourth week of age (Sarfraz *et al.*, 2017; Sultana *et al.*, 2009; Yunus *et al.*, 2008) and also up to sixth week, at which age the bursa of fabricius is at its peak of development where follicles are filled with immature lymphocytes (Mahgoub, 2012; Sharma *et al.*, 2000; Baxandale, 1981; Khan *et al.*, 2007).

Etiology

Infectious bursal disease otherwise known as Gumboro disease is an acute and highly contagious viral disease of mostly young chickens cause by a non-envelope double stranded RNA virus belonging to a family Birnaviridae (Hair-Bejo *et al.*, 2004; Lukert and Saif, 1997). There are two distinct serotypes, namely 1 and 2 of infectious bursal disease virus (IBDV) have been reported

(Lukert and Saif, 1997), out of which only serotypes 1 has been associated with clinical disease, thereby all commercial vaccines available have been prepared against serotype 1 (OIE, 2008). The mortality is determined by multiple factors including the virulence of IBDV, the dose of infection, the age and breed of chickens and the passive immunity as well (Ingrao *et al.*, 2013). Out of four existing pathotypes of serotype 1, very virulent IBD (vvIBDV) have been incriminated for most vaccination failure (Lukert and Saif, 1997), and causes mortality from 50% to 100% in young chickens (Annamalai *et al.*, 2016; Stoute *et al.*, 2013).

Transmission and pathogenesis

The selected host of the virus is young chickens where a clinical disease occurs, while in older birds the infection is essentially subclinical. It was found that light breeds showed higher mortality than the heavier breeds (Bumstead *et al.*, 1993; Nielson *et al.*, 1998). Moreover, the layer type chickens were more susceptible to vvIBDV than broiler-type chickens, both in conventional status and specific-pathogen-free status (Sa *et al.*, 2016; Tippenhauer *et al.*, 2013). Sultana *et al.*, (2008) stated that this disease prevails around the year however stress factors enhances its occurrence in birds. In winter months the sheds were kept air tight to maintain temperature so the virus load increases inwards when the fresh air moves through shed ventilation system it becomes the vital source of aerosol infection in nearby sheds.

The target organ of IBDV is the bursa of fabricius at its maximum development, which is a specific source for B lymphocytes in avian species. After oral infection or inhalation, the virus replicates primarily in the lymphocytes and macrophages of the gut-associated tissues. Then virus travels to bursa

through blood stream, where replication occurs. After 13 hours of post-inoculation most follicles are positive for virus and by 16 hours of same pronounced viraemia occurs with secondary replication in other organs leading to disease and death (Muller *et al.*, 1979). IBDV also invades and replicates in the cells of monocytes-macrophage lineage in a persistent manner (Inoue *et al.*, 1992), which impedes the phagocytic activity of macrophages and facilitates virus dissemination (Lam, 1998). Apart from these the virus can infect chicken bone marrow-derived dendritic cells (Liang *et al.*, 2015).

Diagnosis and histopathology

The observed clinical signs during IBD infection include high mortality, unsteady gait, ruffled feathers, urate containing diarrhoea and sudden death (Islam and Sabad, 2004; Rakibul Hasan *et al.*, 2010). The post-mortem finding includes haemorrhages in the leg and breast muscles, enlarged, edematous and hyperaemic bursa with bloody or mucoid contents or atrophic in chronic cases and haemorrhage in the junction between gizzard and proventriculus. Presence of gelatinous exudates in the serosa of bursa and occasionally atrophied bursa which sometimes contained cheesy mass in the lumen was also noticed. In addition dehydration and nephrosis with swollen kidneys are common (Islam and Sabad, 2004; Chowdhury *et al.*, 1996; Okoye, 1984).

Severe depletion of lymphoid cells is observed not only in the bursa of fabricius but also in the non-bursa lymphoid tissues (Thierry, 2000). Using various immuno staining methods, a higher frequency of antigen-positive cells could be demonstrated after infection of birds with vvIBDV compared with other strains, in the thymus (Inoue *et al.*, 1999; Nunoya *et al.*, 1992; Sharma *et al.*, 1993) the spleen and the bone

marrow (Inoue *et al.*, 1999; Tanimura *et al.*, 1995). Sharma *et al.*, (1993) also investigated that atrophy of the thymus has been associated with the acute phase of the disease and might be indicative of the virulence of the isolate. Thrombocytes also represent a target for IBDV, and acute disease is characterized by disseminated haemorrhages probably related to an impairment of the clotting mechanism (Skeeles *et al.*, 1980). Laboratory diagnosis of IBDV by inoculation in avian embryo through chorio-allantoic membrane (CAM) route is the effective tool where thickened CAM, congested and haemorrhagic dead embryos along the feather tracts, toe and cerebral area is noticed (Rakibul Hasan *et al.*, 2010; Takase *et al.*, 1996). The positive results has been obtained by using molecular methods for detection of IBDV through Agar gel immune diffusion test (AGIDT) by (Gupa *et al.*, 2001; Karunakaran *et al.*, 1993; Muhammad *et al.*, 1996; Shekaro, 2015) and RT-PCR using IBD virus specific primers by (Banda *et al.*, 2001; Hernandez *et al.*, 2006). RT-PCR/RFLP is a very useful and rapid method for characterization and identification of existing and evolving strains of IBDV (Zahoor *et al.*, 2011).

Control and treatment

As it is a viral disease, there is no effective remedy as such but through control measures supportive therapy mortality can be reduced. Haddad *et al.*, (1997) stated that 1-day-of-age-administered IBDV-BDA complex vaccine can induce active immunity and protection against a standard IBDV challenge in the face of variable levels of maternal IBDV immunity. Similarly the role of cell-mediated immunity in pathogenesis of IBD was stated by Yeh *et al.*, (2002) and they concluded that under normal conditions, IBDV induces a protective antibody response; however, in the absence of antibody, CMI alone is adequate in protecting birds against

virulent IBDV. Thee multivitamin therapy along with vaccination showed boosting effects in protection. IBD strain D-78 caused low stress with good protective titre in the birds (Khan *et al.*, 2003). The comparative immune stimulatory studies of two available market products i-e Livol (herbal supplement) and immuno tone (selenium and vit E) by ELISA were noted. Livol showed more encouraging results than immunotone to minimize the adverse effect of IBDV vaccine. Different immunogenic products were tried to select the better one available in commercial markets (Qayyum *et al.*, 2012; Saleem and Mehmood, 2013; Zahid *et al.*, 2015). It promoted immune status against IBD and result in better feed conversion ratio (Malik *et al.*, 2006). VP2 from a virulent IBDV strain 52/70 expressed as a β -galactosidase fusion protein in a recombinant fowlpox virus, fpIBD 1, provided protection against mortality, but not against damage to the bursa of fabricious (Bayliss *et al.*, 1991). A novel complex IBDV vaccine containing a mixture of IBDV with viral antibodies (bursal disease antibody; BDA) has been evaluated for safety and protection of chicks following subcutaneous administration (Whitfill *et al.*, 1995). Apart from these some preventive measures includes:

The premises and equipment should be cleaned and disinfected regularly.

Dead birds need to be well disposed

Wild birds and rodents should be excluded in and around the farm.

Arrangement of foot bath at the entry of farm.

Day to day morbidity and mortality records should be maintained.

Infectious bursal disease commonly observed in chicks during 3-6 weeks of age hampering immune status of the host. The main reason for occurrence is improper vaccination administration, poor vaccine handling, poor

management and bio security in the farm etc which counts for economic losses. Only supportive therapy, immunobooster medicines with vaccination may combat mortalities along with good bio security measures can improve economic status of farmers by reducing morbidity and mortality costs.

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