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A review on infectious bursal disease in chickens (review article)

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Abstract
Infectious bursal disease is an acute, highly contagious and immuno-suppressive viral disease of young chickens caused by a double stranded non-enveloped RNA virus belonging to genus Avibirnavirus and family Birnaviridae. The extensive economic losses is due to high mortality especially in 3-6 week age birds, immune suppression and decreased performance that leads to increase susceptibility to infectious diseases and decrease response to immunization. The target organ of infectious bursal disease virus (IBDV) is the bursa of fabricius which is a specific source for B lymphocytes. Even though turkeys, guinea-fowls, ostriches and ducks are infected with infectious bursal disease but clinical disease appears only in young chickens. The noticed clinical symptoms of IBD include ruffled feathers, whitish watery diarrohoea, unsteady gait and vent feathers soils with urates. The post-mortem lesions includes enlarged, edematous and hyperaemic bursa of Fabricius with bloody or mucoid contents and hemorrhages in the breast and thigh muscles. RT-PCR is the rapid diagnostic method for identification and characterization of existing and evolving IBDV strains. Prevention and control of IBDV is by vaccination and maintaining bio security measures, supportive therapy and immune-booster medicines may control the mortalities.

Keywords: Avibirnavirus, bursa fabricius, chickens, infectious bursal disease

Introduction
Infectious bursal disease (IBD) is an acute, highly contagious and infectious viral disease in young chickens (Nascimento *et al.,* 2017) [4]. The etiological agent was first isolated in Gumboro, Delawer (in United States of America), and the disease was originally known as Gumboro disease (Quinn *et al.,* 2002) [16]. IBD is a huge problem to the global poultry industry (Van den Berg *et al.,* 2000) [20]. Local chicken contributes about 99% of the total poultry population but, however losses due to chicken mortality is very high 61% (Zelleke *et al.,* 2005) [22]. Transmission of virus is through feed, water and faeco-oral route (Sun *et al.,* 2001) [18]. The massive economic losses is due to high mortality, decreased performance, immune supression that leads to increase susceptibility to other diseases and decrease response to immunization (Abdu *et al.,* 2001; Khan *et al.,* 2007) [1]. The major predilection sites of viruses are bursa, thymus (Lymphoid tissue), spleen and bonemarrow (Jones 2008) [7]. The viral disease is characterized by edematous, hyperaemic bursa in acute phase (3-4 days of post infection) and severe atrophy of bursa Fabricius in later phase. Clinical manifestations include whitish watery diarrohoea, deposition of urates in urinary tract (Saif and Barnes 2003) [17]. Pathological changes observed are pin point haemorrhages in thigh muscles and edematous kidney filled with urate deposits (OIE 2008) [14]. Diagnosis is based on history, clinical symptoms and post-mortem findings (Muller *et al.,* 1992) [12]. Viral antigens can be expressed by Agar gel precipitation assay (AGPT) or Antigen capture enzyme linked immune sorbent aassay (AC-ELISA) (Dwight *et al.,* 2004) [5]. Prevention and control measures include vaccination, maintainence of hygiene and biosecurity measures (Kaufer and Weissi 2005; Van den berg *et al.,* 2004) [22, 20].

Etiology
The causative agent of IBDV belongs to genus Avibirna virus of Family Birnaviridae (OIE 2008; Van den berg *et al.,* 2004; Teshome *et al.,* 2015) [14, 20]. The replication occurs in cytoplasm of host cell and involves a virion-associated RNA dependent RNA polymerase. The virus of the family *Birnaviridae* contains the genera which affects chicken, insects and fish (Quinn and Markey 2003) [15].

Transmission: Transmission is via ingestion, inhalation and conjunctiva (Horizontal transmission).

The infected bird excretes the virus in faeces after 48 hours of post-infection and transmits the disease by contact within 16 days of infection. The houses in which outbreak has been occurred the virus remains infectious even for 54-122 days (Van den berg *et al.,* 2004) [20]. The virus is not transmitted vertically, but can survive on the eggshell surface (Maclach and Dubovi 2001) [11].

Pathogenesis

The virus enters through inhalation and faeco-oral route. Four to five hours after oral infection the virus replicates in gut, macrophages, lymphoid cells and kuppfer cells of liver which results in primary viraemia. Then virus spreads to Bursa Fabricius after eleven hours of post infection and replicates in Bursa follicles and B- cells which results in secondary viraemia. Finally the virus reaches muscles and kidney resulting in pathognomic lesions and death (Jordan *et al.,* 2002) [8].

Clinical signs

The incubation period of disease varies from 2-3 days. The earliest sign of IBDV is the tendency of bird to engage in vent pecking. In acute stage the clinical symptoms includes whitish watery diarrohoea, vent feathers soils with urate depositions, prostration, ruffled feathers, unsteady gait, tremors and anorexia. In severe cases dehydration and sub normal temperature followed by death (Zeleke *et al.,* 2005) [22]. In sub-clinical form the chickens experience permanent and severe immunosuppression due to destruction of immature lymphocytes in Bursa Fabricius, spleen and thymus (Jordan *et al.,* 2002) [8].

Gross lesions

The post mortem lesions include haemorrhages in breast and thigh muscles, dark and discolouration of pectoral muscles, pale and edematous kidney with urate deposits (Weissi and Kauffer 1994) [21]. Presence of slimy exudates in the serosa of bursa and occasionally atrophied bursa which contains cheesy exudates in the lumen (Islam and Samad 2004) [6]. Bursa, the target organ of virus undergoes series of changes after post infection is as follows. On 3rd day edematous bursa due to accumulation of fluid and by 4th day bursa becomes double the normal weight and size and on 8 th day it reduces to one-third of its normal weight (Musa *et al.,* 2012) [13]. Spleenomegaly with greyish foci evenly distributed on the surface of spleen. Petechial haemorrhages on mucosa at the junction of proventriculus and gizzard (Ashraf *et al.,* 2006) [2].

Microscopic lesions:

Necrosis and degeneration of B- lymphocytes in the bursal follicles, lymphocytic depletion is replaced by heterophil and hyperplasic reticulo-endothelial (RE) cells (Saif and Barnes 2003) [17].

Diagnosis

Diagnosis is based on flock history, clinical symptoms and post-mortem findings (Muller *et al.,* 1992) [12]. Pathological changes observed at bursa is characteristic and histopathological conclusions combined with immune histochemistry confirm IBDV infection. Viral antigens demonstrated by Agar gel precipitation test (AGPT) and Antigen capture enzyme linked immune sorbent assay (AC-ELISA) (Dwight *et al.,* 2004) [5]. RT-PCR in combination with restriction enzyme analysis is a rapid diagnostic method for identification and characterization of IBDV strains (Zeinberge *et al.,* 2000).

Prevention and control

Prevention and control of IBDV is by immunization, practice of All-in All-out management and bio security measures, supportive therapy and immune-booster medicines may control the mortalities (Jordan *et al.,* 2002) [8]. Apart from these preventive and control measures include –cleaning and disinfecting of premises and equipments, proper disposal of dead birds, placing of foot dips at the entry of farm, maintainence of day-to-day morbidity and mortality records. Entry of vehicles, visitors and equipments are strictly restricted into the farm.

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