Infectious Bursal Disease

Zeinab M. S. Amin Girh[1], Nagwa S. Rabie[1] and Mona S. Zaki[2]

[1]Department of Poultry Diseases, National Research Centre, Dokki, Giza, Egypt.
[2]Hydrobiology Department, National Research Centre, Dokki, Giza Egypt
drmonazaki@yahoo.com

Abstract: Infectious Bursal Disease (IBD), or Gumboro disease, is an acute, highly contagious viral infection of chickens. Infectious Bursal Disease continues to be a potential threat to the poultry industry. Infectious Bursal Disease is a highly contagious, globally occurring acute viral poultry disease caused by a bisegmented, double stranded RNA virus that belongs to the genus *Avibirinavirus* family *Birnaviridae*. The disease is economically significant to the commercial poultry industry through the mortality, reduced weight gain and condemnation carcass due to marked haemorrhage in the skeletal muscle as well as immunosuppression. The re-emergence of IBD in variant or highly virulent form in different parts of the world during the last couple of decades, have demanded further research efforts in understanding the added complexity of the disease process and the means to control it. Control of the disease has been through exclusion or eradication of chickens via all-in/all-out procedure and genetic selection of chickens resistant to the disease. At present, the disease is controlled by the combined use of live virus and inactivated oil emulsion vaccines. But these vaccines are not always safe as they may not contain the required immunogens present in the variant strains prevailing in that area. Thus, new technologies and second-generation vaccines including rationally designed recombinant and subunit vaccines have been developed.
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Introduction

Infectious Bursal Disease (**IBD**), or Gumborodisease, is a highly contagious disease of young chickens caused by infectious bursal disease virus (**IBDV**). Infectious bursal disease causes severe economic losses to the poultry industry worldwide by bringing about high mortality and immunosuppression that lead to increased susceptibility to opportunistic infections and poor response to widely used vaccines against other pathogens **(Sharma et al., 2000; Muller et al., 2003).** The IBDV is highly resistant to physical and chemical inactivation (**Enterradossiand Saif, 2008)**. The disease was first reported by Cosgrove, who in 1962 observed a disease, affecting chickens on farms in the neighbourhood of Gumboro, Delaware, USA **(Cosgrove, 1962)**. Thus, Gumboro disease became synonymous for the condition. The virus causing IBD suppresses the immune system of affected birds by damaging organs of primarily the humoral cell defence, particularly bursa of Fabricus (BF), hence alternatively named **(Cullen, 1982).** Infectious bursal disease has worldwide distribution, and the effects of the disease are economically significant to the commercial poultry industry **(Muller et al., 2012)**. Through the mortality, reduced weight gain and condemnation carcass due to marked haemorrhage in the skeletal muscle **(Tesfaheywet et al., 2012)**. The domesticated hen (*Gallus gallus*) is the only species for which IBD virus (IBDV) has been reported to induce clinical disease. However, some reports in serological surveys in wild birds **(Ogawa et al., 1998)**. Suggest their role as a reservoir. Until 1987, the field strains of IBDV were of low virulence and caused only 1% to 2% mortality **(van den Berg et al., 2000).** However, new IBDV strains emerged and able to cause up to 5% specific mortality in USA **(Rosenberger and Cloud.1986).** Meanwhile, IBD outbreaks that caused high mortality of 50% to 60% in the laying hens and 25% to 30% in the broilers were reported in Europe and Japan, respectively. These outbreaks were caused by the highly pathogenic field isolates that was also known as very virulent strains (vvIBDV) and capable of causing up to 100% mortality in specific-pathogen-free (SPF) chickens **(van den Berge and Gonzea., 1991)**. The incubation period of IBD is about two to three days. The infected chickens will then be having watery diarrhea and become exhausted, prostrated, dehydrated, and ruffled feathers **(Ley et al., 1983)**. Usually death follows at three days post infection. The flock mortality rate reaches the peak at day four, but will rapidly drop. The survivals, despite having the destructed bursas, will

recover by five to seven days post infection **(van den Berg et al.,., 2000)**. Nevertheless, because their bursa had been destructed by the virus, the recovered birds became immunosuppressed and susceptible to any opportunistic infection. Outbreaks of IBD may not always be noticeable; particularly when the flocks' maternal antibodies were present or the involved IBDV strains were of low pathogenicity **(van den Berg et al., 2000)**. The infected chickens, though may appear healthy, were indeed immunosuppressed and unresponsive to the costly vaccination programmes. Subclinical IBDV infection is not uncommon in the field and may prevail especially after the decline of passive immunity **(van den Berg et al., 2000).** On the other hand, acute outbreaks with high flock mortality rate usually suggested that vvIBDV strains are involved **(van den Berg et al., 2000)**. And should the virus persist in the farm premises and transmitted to the successive flocks, the clinical signs will appear earlier and gradually replaced by the subclinical forms **(van den Berg., 2000)**. However, the farm still suffers from episodes of acute IBD outbreaks. The causative agent is a bisegmented, double stranded RNA virus that belongs to the family Birnaviridae. IBDV is a non-enveloped icosahedral virus, approximately 58-60 nm in diameter (van den Berg, 2000). IBDV is endemic in most poultry producing areas of the world. The virus is highly stable and has a tendency to persist in the environment despite thorough cleaning and disinfections. The virus is highly stable to chemical and physical agents **(Ley et al., 1983)**. In poultry houses the virus can remain viable for up to 60 days in the litter **(Vindevogel et al, 1976)**. There are two serotypes of IBDV: serotype 1 and 2. All viruses capable of causing disease in chickens belong to serotype 1; serotype 2 viruses may infect chickens and turkeys and are considered non-pathogenic for both species **(Jackwoodet al., 1982)**. Viruses of both serotypes of IBDV share common group antigens that can be detected by fluorescent antibody test (FAT) and enzyme linked immunosorbent assay (ELISA) **(Hair-Bejo, 2006)**. The caIBDV was The predominant strain until early 1980s **(van den Berg et al., 2000)**. The vaIBDV which is antigenically different from the caIBDV was reported in the mid-1980s in USA **(Snyder *et al.*, 1994)** pointed out that this Variant strain is also different from the classical strain in that it results in severe bursal atrophy and also the vaccine produced from classical strain did not give full protection against the vaIBDV. In late 1980s; however, vvIBDV which caused an acute IBDV was reported from Europe with a high mortality in young chickens ranging from 21 to 35 days of age **(van den Berg and Meulemans,.1991)**100% mortality was reported in susceptible chickens **(van den Berg,2000)**.

Aetiology and natural hosts

Infectious bursal disease virus (IBDV) belongs to the genus *Avibirnaviurs* of the family *Birnaviridae*. There are two recognized serotypes of IBDV. Serotype 1 is pathogenic only too young chickens, and can be divided into Classical virulent, attenuated, antigenic virulent and very virulent strains**.** Serotype 2 viruses are naturally A virulent and do not cause clinical disease. The natural hosts of IBDV are chickens (serotype 1) and turkeys (Serotype 2). Antibodies or virus are sometimes found wild birds including ducks, guinea fowl, quail, Pheasants and ostrich but no signs of infection are seen. There is no evidence that IBD virus can infect other animals **(OIE, 2016).**

Epidemiology

In chickens, severe acute disease, usually in 3 to 6 week-old birds, is associated with high mortality, but less Acute or subclinical infections are common earlier in life. IBDV multiplies rapidly in developing B lymphocytes in the bursa of Fabricius leading to immunosuppression, increased susceptibility to other diseases, and clinical disease. IBD causes lymphoid depletion in wild birds but infection is generally sub-clinical. The incubation period is about 2-3 days; virus shedding can last up to two weeks. The virus is highly Contagious and highly resistant to heat and chemicals. It can persist in faeces, bedding, and contaminated feed. and water for up to four months and can spread through the movement of poultry products, equipment, feed, bags, vehicles and people, and to a lesser extent, through dust aerosols. There is no evidence that IBD can be transmitted in embryos or semen **(OIE 2016).** The role that wild birds may play in the transmission of IBDV remains uncertain **(Animal Health Australia 2009).**

An epidemiological link has been identified between wild birds, (including waterfowl) and domestic chickens. Evidence suggests that wild birds may transmit the virus between poultry flocks, and May also serve as a reservoir for the virus (Ogawa et al. 1998; Jeon et al. 2008; Kasanga et al. 2008). There is At least one documented case (in South Korea) of IBDV transmission from wild birds to commercial poultry (Jeon et al. 2008). Clinical signs in chickens, acute infection cause depression, debilitation, dehydration, watery diarrhea and Swollen, blood-stained vents. Mortality rates vary with virulence of strain and dose as well as the host's ability to mount an effective immune response. Infection with less virulent strains may not result in overt clinical Signs but chickens may develop fibrotic or cystic bursa of Fabricus that can prematurely atrophy (before six months of age) and they may succumb to other infections due to immunosuppression. IBDV infection

in wild birds is believed to be subclinical with no associated signs. In artificially inoculated wild birds, there was no evidence of disease, changes in behavior, or mortality **(Van den Berg et al. 2001)**.

Diagnosis

Clinical and differential diagnosis

The clinical diagnosis of the acute forms of IBD is based on disease evolution (a mortality peak followed by recovery in five to seven days), and relies on the observation of the symptoms and post-mortem examination of the pathognomonic lesions, in particular of the bursa of Fabricius.

The conditions most liable to be clinically mistaken for IBD are avian coccidiosis, Newcastle disease in some visceral forms, stunting syndrome, chicken infectious anaemia, mycotoxicoses and nephropathogenic forms of infectious bronchitis. In all acute cases, the presence of bursal lesions allows for a diagnosis of IBD. In subclinical cases, an atrophy of the bursa may be confused with other diseases such as Marek's disease or infectious anaemia. A histological examination of the bursa will allow differentiation between these diseases **(Lukert and Saif, 1997).**

Histological diagnosis

Histological diagnosis is based on the detection of modifications occurring in the bursa (see the sub-section entitled 'Clinical signs'). The ability to cause histological lesions in the non-bursal lymphoid organs, such as the thymus **(Inoue et al.,1994)**, the spleen or bone marrow **(Inoue et al.,1999)**

Serological diagnosis

In areas contaminated by IBDV, most broiler flocks have anti-IBDV antibodies when leaving the farm. Current serological tests cannot distinguish between the antibodies induced by pathogenic IBDV and those induced by attenuated vaccine viruses, so serological diagnosis is of little interest in endemic zones. Nonetheless, the quantification of IBDV-induced antibodies is important for the medical prophylaxis of the disease in young animals, in order to measure the titre of passive antibodies and determine the appropriate date for vaccination **(DeWit, 1999)** or in laying hens to verify success of vaccination (Meulemans et al.,1987)

The most widely used quantitative tests are the detection of precipitating antibodies by agar gel immunodiffusion (AGID) **(Cullen and Wyeth,1975)**, enzyme-linked immunosorbent assay (ELISA) **(Marquardt et al.,1980)**, and SN in cell culture **(Weisman and Hitchner,1978).**

Agar gel immunodiffusion is the simplest, but least sensitive technique. Results are obtained after an incubation period of48 h. Variability in results may be due to the investigator, as well as the nature of the viral strain used as an antigen **(Wood et al.,1984)** Serum neutralisation presents the disadvantages that specialised equipment and five days incubation are required.

The technique is much more sensitive than AGID and correlates better with the level of protection of the subjects tested **(Roney and Freund.,1988)**.

The ELISA is the most rapid and sensitive method, and presents the fewest variations due to the viral strain used as an antigen **(Roney and Freund, 1988)**. Considerable inter- and intra-laboratory variability can occur with certain commercial kits (**Kreider et al.,1991**).

Although the correlation between results obtained using SN and ELISA is high, ELISA remains less sensitive, and does not detect low neutralizing titres which are sufficient to block vaccine administration (residual maternal antibodies).

Enzyme-linked immunosorbent assays which use are combinant VP2 protein as the sole antigen may be better correlated with protection **(Jackwood et al., 1999)**.

Authors

Zeinab M. S. Amin Girh[1], Nagwa S. Rabie[1] and Mona S. Zaki[2]

[1]Department of Poultry Diseases, National Research Centre, Dokki, Giza, Egypt.

[2]Hydrobiology Department, National Research Centre, Dokki, Giza Egypt

drmonazaki@yahoo.com

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