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An overview of Infectious Bursal Disease in chickens

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Abstract

Gumboro disease, also known as infectious bursal disease (IBD), is a highly contagious immunosuppressive illness of young chickens brought on by the IBD virus (IBDV) that causes significant financial losses for the global poultry industry. Chickens and turkeys are naturally infected with IBDV by the faecal-oral route and also via breathing. Virus targets immature B lymphocytes specifically. All immune system components are activated when chickens get infected with IBDV. However, the degree of activation varies according to the age, immunological condition, genetic background, and virulence of the infecting strains of chickens. The diagnosis is made by molecularly identifying the viral genome and clinically assessing the cloacal bursa. Breeder flock vaccinations are used to create maternal immunity in early chicks, which helps achieve control.

Introduction

The double-stranded RNA virus known as IBDV is a member of the family Birnaviridae and genus Avibirnavirus. There are two segments to the IBDV genome: segment A and segment B. The 110 kDa polyprotein that is encoded by larger open reading frame 1 (ORF1) of segment A auto-catalytically splices into the viral proteins VP2 (48 kDa), VP3 (33–35 kDa), and VP4 (24 kDa). IBDV is known to infect chicken, turkey, duck, guinea fowl, and ostriches. It has two identified serotypes, serotypes 1 and 2, and only serotype 1 has been shown to be pathogenic in chicken. The virus is constantly changing in the field in terms of its virulence and antigenicity. The VP2 is the primary host-protective capsid protein of the virus that contains immunogenic components and elicits neutralizing antibodies. Based on notable variations in the amino acids found in the hypervariable region of the capsid protein VP2 (hVP2) among the various populations, IBDV has recently been divided into seven genogroups, with genogroup 1 being dispersed worldwide. (**Dey *et al.*, 2019**).

Host:

Chickens and turkeys are the natural hosts of IBDV, which is host specific.

Mode of transmission:

IBDV targets the gut mucosa's macrophages and lymphoid cells for infection and replication whereas B lymphocytes in the Bursa of Fabricius (BF) are the primary targets for

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widespread replication. Classical and variant IBDV attack immature B cells, but vvIBDV targets both immature and adult B lymphocytes. Infected macrophages carry IBDV to the BF, where it replicates intracytoplasmically in IgM+ B cells. The activation of macrophages in the BF leads to increased production of interferon-γ (IFN-γ). Pro-inflammatory cytokines including IL-6 and NO are released during this process. Cytokines can exacerbate bursal lesions. IFN-γ generated during IBDV infection may cause apoptosis of both infected and healthy B-cells. Infected hens with vvIBDV may spread the virus to additional lymphoid organs, including bone marrow, thymus, spleen, Peyer's patches, caecal tonsils, and Harderian glands.

Incubation period and target age:

The incubation period for IBD is 2 to 4 days, with the most severe clinical manifestations observed in chickens afflicted between 3 and 6 weeks of age. Clinical symptoms are infrequent in chickens under 2 weeks and over 6 weeks old.

Clinical signs: (Orakpoghenor *et al*., 2020)

1. Acute clinical occurrences of classical IBDV in vulnerable chicken flocks (3-6-week-old broilers and replacement pullet flocks) are marked by fast commencement, significant morbidity, spiked mortality curves, and a quick recovery time of 5-7 days after clinical symptoms.
2. Susceptible chickens exposed to vvIBDV and classical virulent strains may experience depression, lack of movement, watery diarrhoea and ruffled feathers within 2-3 days.
3. Vent pecking, inflammation of the cloaca and urate staining on pericloacal feathers have also been reported. Fecal IBDV can be detected upto 4 weeks using RT-PCR.

Role of maternal antibodies in protection against IBD:

Maternal antibodies have the ability to modify the immune response, and more virulent vaccine strains have the ability to suppress greater antibody levels. For up to three weeks following hatching, most young chicks get shielded against vvIBD viral exposure by high levels of maternal antibodies. Broilers with high maternal antibodies (MAB) levels after exposure to vvIBDV do not exhibit clinical symptoms or death.

Necropsy findings:

1. Pectoral and leg muscles show congestion and hemorrhages.
2. Presence of enlarged cloacal bursa (Fig 1, source: *msdvetmanual*).
3. Atrophied bursa due to some strains of IBD (Fig 2, source: *msdvetmanual*).
4. Presence of atrophied bursa in recovered chickens due to destruction of bursa.

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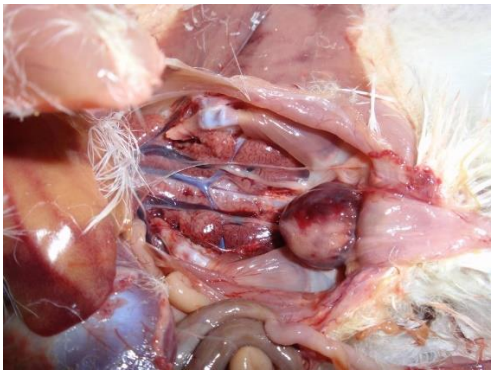

Fig 1: Enlarged cloacal bursa

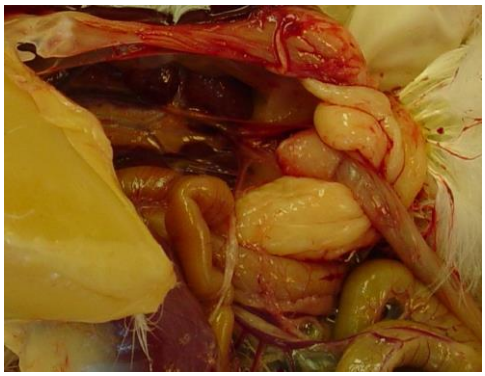

Fig 2: Atrophic cloacal bursa

Treatment: There is no specific treatment for the disease. Control and Prevention by vaccination regimen is suggestive:

Layers (ICAR)		
Age	**Name of Vaccine**	**Route**
14-16th day	IBD	I/O or D/W
24-26th day	IBD (Booster)	D/W
Broilers (ICAR)		
7-9th day	IBD	I/O or D/W
16-18th day	IBD (Booster)	D/W

Depending on level of maternal antibodies

1. At 1-2 weeks of age, and again at 3–4 weeks of age, for chickens with low levels of maternal antibodies.
2. High concentration of maternal antibodies in chickens: - at the ages of 3–4 weeks and 8–12 weeks for the second immunization

Differential diagnosis

The differential diagnosis for inflammatory bowel disease (IBD) includes conditions such as avian coccidiosis, certain visceral forms of Newcastle disease, stunting syndrome, mycotoxicoses, chicken infectious anemia, and nephropathogenic types of infectious bronchitis. IBD may be diagnosed in all acute cases because bursal lesions are present. When bursa atrophy occurs in subclinical instances, it might be mistaken for other conditions such infectious anemia or Marek's disease. Making the distinction between these conditions will be possible through a histological analysis of the bursa (**Getachew and Fesseha, 2020**).

Conclusion

At the moment, live attenuated or inactivated IBDV vaccinations are used to manage the disease; however, live vaccines have the potential to return to virulence, and standard vaccines could not provide complete protection against the vvIBDV strain. Typically, pre-laying birds

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receive inactivated or killed vaccinations to boost antibody production for a minimum of two weeks. Nevertheless, the efficacy of traditional IBD vaccinations against virulent IBDV (vvIBDV) and its antigenic variations has diminished in chickens. Therefore, in addition to safety, convenience of manufacture, and stability, next generation vaccines are generated with the benefit of resisting interference with maternally derived antibodies (MDA).

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