5-Infectious Bursal Disease (I.B.D)

(Gumboro)

***Definition:** it is **acute**, highly **contagious**, viral disease of **young** chickens, it is characterized by **destruction of lymphocytes in the bursa of fabricius**(BF) and to lessor extend in other **lymphoid organs**.

*Etiology:

It is caused by **Birna**.
*First recognized **1962** referred to as **"avian nephrosis"** because of extreme kidney damage in birds that succumbed to infection.

*The virus **resistan**t to many **disinfectants and environmental** factors and remain infectious for at least **4 months** in poultry house environment.

*The economic importance of the disease:

It is manifested in many ways:-
1-**Mortality** may reach **20% or more** in chick **3 weeks** of age and older.
2-Severe prolong **immunosuppression** at early age.
3-**Sequelae** associated with immunosuppression **induced by virus** include **(gangrenous dermatitis, inclusion body hepatitis, anemia syndrome, E.coli).**
4-**Vaccination failures**.

*Transmission of the disease:

*Chicken infected with IBD virus, **shed virus in their feces in feed, water**, poultry house, litter become contaminated.
* Because of the **resistant nature of the IBD virus**, it is transmitted among the farms by **people, equipment and vehicles.**

*Clinical Signs:

IBD follows **one of two courses depending on the age** at which chickens are infected:-

1-Subclinical Form:-

1-It is occur in chick **less** than **3 weeks** of age, chick show **no** clinical signs of disease but experience permanent and **severe immunosuppression.**

2-The **immunosuppression** occur due to damage to **bursa of fabricius** and this form is **more economically important** from the disease.

3-Broiler infected by this form typically have **poor body weights** and **feed conversions**, **high mortality**, excessive reaction to respiratory vaccines and high rate of **condemination at processing**.

2-The clinical Form:-

Usually occur in chickens from **3-6 weeks** of age, the clinical disease has a **sudden onset** and **mortality rate in the flock increase rapidly**.

 # Clinical signs include:

*Dehydration.
*Trembling.
*Ruffled feathers.
***Vent pecking**.
***Depression**.

*Gross lesion (P.M):-

1-**The bursa of fabricius appears to be the primary target organ of the virus.**
2-On **3** days post-infection, the bursa of fabricius is **swollen (inflamed)** appears **edematous and hyperemic and gelatinous yellowish** transudate covering the serosal surface.
3-On **4** days, the size begins to decrease, and usually reach its **normal weight**.
4-On **5** days, the bursa **diminished** in size rapidly **(atrophy),** and the organ may become **gray** (while the normal white color turns to cream color).

5-On**8** days, it is **one-third** its original weight, or even less.

6-**Petechial hemorrhages** and area of necrosis may be present in **more severe cases.**
7-**Necrosis** and **depletion of lymphocytes** also occur in the **secondary lymphoid organs** including **the spleen** (small gray foci on the surface),**Harderian glands and cecaltonsi**l,**bone marrow** these organs are affected but less severity than the bursa of fabricius and recovery may occur after infection.

8-**Hemorrhage** may be present in the **thigh and pectoral muscles** and in the mucosa at the **juncture of the proventriculus and gizzard**.

9-The **Kidneys** may appear swollen in the birds that **die or are in advanced stages** of the disease, and in such lesions probably result from **severe dehydration**, not from viral.

*Microscopic lesions:-

***On **1**day post infection (PI), there was **degeneration and necrosis of lymphocytes** in medullary of bursal follicles. Lymphocytes **replaced by heterophils.**
*On **3 or 4** days PI, **all lymphoid follicles** were affected (few lymphocytes are present), the **increase** in bursal weight caused by **severe edema**, **hyperemia**, and marked accumulation of **heterophils**.

*As the inflammatory **reaction declined**, **cystic cavity** developed in medullary areas of follicles, necrosis and **plasma cells** occurred, there was a **fibroplasia** in **interfollicular connective tissue.**

*Diagnosis:-

1-Flock history, clinical signs and lesions, confirm the diagnosis of clinical IBD be made at necropsy by examination the BF during the early stages of disease for **characteristic gross lesions**.
2-Histopathology of BF.
3-Serology e.g ELISA, AGP.
4-Viral isolation.

*Prevention and Control:-

An effective IBD prevention control programs must involve:-
1-effective **breeder** vaccination programs.
2-effective biosecurity programs.
3-effective **broiler** vaccination programs.

1-Effective **breeder** vaccination programs,Immunization of breeders are important part of IBD control program, because **Maternal Abs** if present in adequate levels protects the chicks against **subclinical IBD.**
*Example of breeder vaccination program:-
→ 2 live vaccine (12-15 day), (30-33 days).
↘ 2 inactivated vaccine 85 days, 120 days.
*Routinely monitor breeder IBD Abs titers to ensure vaccines are administrated properly and the chicken respond appropriately.

2-Biosecurity is very important to reduce the field virus exposure.

3-Effective broiler vaccination programs,good vaccination of broiler to prevent **clinical IBD.**

***Three category of vaccination based on their pathogenicity has been described:-**
1-Mild
2-Intermediate.
3- Virulent.

***Intermediate** type of IBD **vaccines** are most commonly used, because can stimulate the broiler to **produce Abs** earlier than mild type vaccines **without significant damage** to BF as mayoccur with the virulent type vaccines.
*The **timing of broiler vaccination depends** on the **levels of Maternal Abs** present in chicks, high levels of maternal Abs at the time of vaccination will **neutralize** the vaccine virus.

*Approximately **10-12** days are required after vaccination for chicken to develop minimal protection titers.
*In Virulent IBD **viruses** are able to overcome higher maternal titers from milder vaccines, therefore the vaccination of broiler **cannot** protection the flock against infection.

*__Variant strain of IBD virus__:
Control of IBD has been further complecated by variant strain of IBD virus, these variant strain induce **damage** in BF in chicken even with **high Abs** titer are present, and **donot** cause clinical disease but induce serum immunosuppression.
*The BF of affected chicken undergoes rapid **atrophy** without the inflammatory changes that observed early in the infection with classical IBD viruses.

*These variants are not from a different serotype but are **Antigenically different** enough to cause problems, in this case consideration should be given to vaccination breeders with **inactivated vaccines** containing **standard and variant strain of IBD viruses**