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Infectious Bursal Disease of Chickens/ I.B.D./ Gumboro disease/ Avian Nephrosis

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This disease was first recognized in 1962 in an outbreak in ***Gumboro district*** in Delaware (U.S.A.) *i.e.* this disease is also known as "Gumboro disease". The most prominent lesion of this disease is located in the cloacal bursa (bursa of fabricius), hence this disease is known as Infectious Bursal Disease. It is of considerable economic importance and is of scientific interest because of the nature of the virus and its affinity for replicating in dividing pre-B lymphocytes in the bursa of Fabricius, leading to acquired B lymphocyte deficiency in affected birds. Infectious bursal disease occurs worldwide

Two serotypes of the virus show minimal cross protection; only serotype 1 is pathogenic. Serotype 1 has three antigenic subgroups all of which vary markedly in their virulence

1. Classical or standard viruses: - produce 10-50% morality
2. Variant viruses: - produce no mortality
3. Very virulent viruses: - produce 50-100 %morality

Causative agent: - Avibirnavirus

Transmission: - Infectious bursal disease virus is excreted in the feces for 2 to 14 days; it is highly contagious and transmission occurs directly through contact and oral uptake. Indirect transmission via contaminated feed, water, dust, litter, and clothing or mechanical spread through insects may occur. Vertical transmission probably occurs via the egg.

Clinical Features: - in case new introduction of IBD virus into a flock, morbidity approaches 100% and mortality may be up to 90%.

- ✓ Disease is most severe in chicks 3 to 6 weeks of age, when the target organ, the bursa of Fabricius, reaches its maximal stage of development.
- ✓ Chicks less than 3 weeks of age are not susceptible (may have subclinical infection) because of their limited numbers of pre-B lymphocyte (they do not have fully developed burs of Fabricius) or from the presence of protective antibodies.
- ✓ Birds older than 6 weeks rarely develop signs of disease, although they produce antibodies to the virus.

After an incubation period of 2 to 3 days, chicks show distress, depression, ruffled feathers, anorexia, diarrhea, trembling, and dehydration; usually 20 to 30% die. The clinical disease lasts for 3 to 4 days, after which surviving birds recover rapidly.

Pathogenesis, Pathology, and Immunity

Following oral infection, the virus replicates in gut-associated macrophages and lymphocyte in the ceca and small intestine (by 4-5 hours), from which it enters the portal circulation, leading to primary viremia. Within 11 hours of infection, viral is present in lymphocyte of the clocal bursa (but not in lymphoid cells of other tissues) with the production and release of large amounts of virus from the bursa resulting secondary viremia and in localization in other tissues, including other lymphoid tissues.

The most striking feature of the pathogenesis and pathology of infectious bursal disease is the selective replication of virus in the bursa of Fabricius, which ***early in infection becomes enlarged up to five times its normal size and becomes edematous, hyperemic, and cream colored, with prominent longitudinal striation***s. Lymphoid follicles of the bursa become totally necrotic as a consequence of both necrosis and apoptosis, and in surviving birds they are devoid of lymphoid cells. Very virulent virus strains also produce depletion of cells in the thymus, spleen, and bone marrow. Hemorrhages occur beneath the serosa and there are necrotic foci throughout the bursal parenchyma. ***At the time of death (late stage) the bursa may be atrophied and gray and the kidneys are usually enlarged, with accumulation of urates due to dehydration and possibly with immune complexes in the glomeruli.***

Laboratory Diagnosis

1. **Sample -** Bursa (Hypertrophied bursa), kidney, thymus, spleen
2. **Direct identification of virus: -** by electron microscopy of bursal specimens

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3. **Virus isolation**
    - **Virus isolation in experimental birds:** 2-4 weeks old birds are inoculated intra-bursally and intra-ocularly with virus sample. The birds scarified after 2 to 3 days and bursa should be collected which contain virus.
    - **Virus isolation in cell culture:** chicken embryo fibroblast cell culture.
    - **viral isolation in embryonated eggs:** By CAM route
4. **Direct detection of Viral antigen: -**
    - ✓ The impression smears of bursal tissue give positive FA test
    - ✓ Hypertrophied bursa of infected birds is very rich in virus and will give positive gel diffusion tests
5. **Direct identification of viral nucleic acid: -** PCR
6. **Detection and quantitation of antiviral antibodies:** SNT, CFT, FAT, Gel diffusion test

Prevention and Control

- Both modified live and inactivated vaccine are available. Breeding stock is vaccinated by adding vaccine virus to drinking water in the hope that passively transferred maternal antibody will prevent infection of the newly hatched chicks at the time of their maximum susceptibility.
- An increasingly common practice is to follow oral live-virus vaccination of breeding stock, after they have reached the age of about 18 weeks, with an injection of inactivated vaccine in oil adjuvant just before they begin laying. Vaccination is repeated a year later.
- This results in a well-maintained high level of neutralizing antibody throughout the laying life of the birds. Maternal antibody provides effective protection for chicks for between 4 and 7 weeks after hatching.
- In situations where chicks have low or inconsistent levels of maternal antibodies, vaccination is carried out with an attenuated virus vaccine, starting at 1 to 2 weeks of age.

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