INFECTIOUS BURSAL DISEASE

(Commonly known as: "gumboro disease" and "infectious bursitis",)

Infectious bursal disease (IBD) is an acute, highly contagious infection of **young** chickens (less than 10 week age), caused by a Birnavirus.

In the beginning the name "Gumboro disease" was given to this condition, because it was first recognized in the Gumboro district of Delaware, a state of USA. In 1962, IBD was described as a specific new disease by **Cosgrove** in USA, and was referred to as "**avian nephrosis**" because of the extreme kidney damage found in birds that succumbed to infection. In 1970, **Hitchner** proposed the term "**infectious bursal disease**" on the basis of specific pathognomonic lesions of the bursa.

Severe acute disease of 3 to 6 week old birds is associated with high mortality, and signs including prostration, diarrhoea, and sudden death. Post-mortem examinations of acute IBD cases reveal a combination of muscular and proventricular haemorrhages, nephritis and bursal inflammation, with bursal oedema or haemorrhages in the first 4 days, followed by bursal atrophy later in the course of the disease.

IBDV causes severe lymphoid depletion in the bursa of Fabricius. Lymphoid cells, especially B cells, are the primary target cell. Significant lymphoid depletion and depression of the humoral antibody responses may lead to vaccination failures, secondary infections like airsacculitis, *E. coli* septicaemia, gangrenous dermatitis, inclusion body hepatitis-anaemia syndrome.

The disease is of great economic importance for two reasons: 1) Due to the heavy mortality in chickens 3 weeks of age, and older, and 2) Due to a severe prolonged immunosuppression of chickens infected at an early age.

AETIOLOGY

Infectious bursal disease (IBD) is caused by **Birnavirus**, more specifically the type-1 avibirnavirus strains, that is a non-enveloped, double stranded RNA virus.

Birna viruses causing IBD have two main serotypes. **Type-1 avibirnavirus** strains that is classic and highly pathogenic (vv IBD) serotype having tropism for B lymphocytes of the bursa and the other **Type-2 avibirnavirus** which infect turkeys and ducks but apathogenic to chickens.

SPREAD

IBD is highly contagious and virus is persistent in poultry houses, even when thorough cleaning and disinfection procedures are followed virus persists. Direct contact of young birds with infected flocks in multi-age units results in persistent "rolling" infection which is difficult to control. Contaminated water, feed, and droppings in poultry houses are important source of infection.

Mechanical vectors such as wild birds, human workers, and vermin (mealworms, litter mites, mosquito etc) also have role in spead. There is no evidence that the virus is transmitted through the egg, or that a carrier state exists in recovered birds.

PATHOGENESIS

The most common route of infection is oral, but conjuctival and respiratory routes may also be important. 4-5 hrs after oral infection, virus can be detected in macrophages and lymphoid cells and the virus first reaches the LIVER (kupffer cells). It then enters bloodstream and viraemia occurs when the virus infects other organs including the spleen, the harderian gland, thymus and importantly the Bursa.

The bursal infection is followed by a second massive viraemia. The virus affects lymphoid tissue, causing destruction of lymphoid cells within the bursa of Fabricius, spleen, and caecal tonsils. **B-Lymphocytes and their precursors are the main target cells**, although virus may be found in macrophages. T-Lymphocytes are relatively unaffected. The consequences of immunosuppression are lowered resistance to disease and suboptimal (inadequate) responses to vaccines given during this time.

In some birds the kidneys may appear swollen and may contain urate deposits and cell debris; this is probably due to blockage of ureters by a severely swollen bursa. The cause of muscle haemorrhage is unknown.

The period of greatest susceptibility is between **3 and 6 weeks of age**. Susceptible chickens younger than 3 weeks do not exhibit signs, but have subclinical infections that lead to severe immunosuppression.

Microscopic lesions in the bursa resemble an Arthus reaction, characterized by necrosis, haemorrhage, and large numbers of polymorphonuclear leukocytes. This reaction (**type III hypersensitivity**) is a localized immunological injury caused by antigen-antibody-complement complexes which induce chemotactic factors.The chemotactic factors, in turn, cause haemorrhage and leukocyte infiltration. Increased clotting times in affected chickens have led to the suggestion that such coagulopathies may also contribute to the haemorrhagic lesions observed with this disease.

SIGNS

A subclinical disease is common in 0-3 week-old birds. IBD virus (IBDV) causes lymphoid depletion of the bursa, and, only lesions associated may be bursal atrophy and lesions associated with secondary infections.

The acute form is seen in chicks between 3-6 weeks of age after an incubation period of 2-3 days. Earliest sing is 'Self vent picking', other signs include depression, white watery diarrhoea, soiled vents, anorexia, ruffled feathers, reluctance to move, closed eyes, and death.

The course of the disease in the individual chick is short, leading to death or recovery. On the flock basis mortality reaches a peak 3-5 days after infection. Morbidity ranges from 10%-100% and mortality 0%-20%, sometimes reaching 50%. Strains of very virulent IBDV (VVIBDV) cause 90%-100% mortality.

LESIONS

GROSS LESIONS

The carcasses are dehydrated. Severe dehydration and muscular hemorrhages are evident in dead birds.

- Paint-brush haemorrhages in the thigh and pectoral muscles, and sometimes on the mucosa of the proventriculus. Haemorrhages of leg muscles are typical of IBD.
- Enlarged and inflamed **Bursa fabricus**, which is oedematous and surrounded by gelatinous yellow exudate on serosal surface with colour changing from white to cream. There are prominent longitudinal striations on bursa. Haemorrhages may be seen on the internal and serosal surfaces, and a caseous core is formed within the lumen from sloughed epithelial tissue. Recovered birds show bursal atrophy.
- Sever catarrh or increased mucus in the intestine. The liver may be swollen and show peripheral infarcts. In some cases spleenomegaly occurs. The swelling and white appearance of the kidneys, and dilatation of the tubules with urates and cell debris are seen in some outbreaks, but do not appear to be a consistent finding.

MICROSCOPIC LESIONS

Microscopic changes occur mainly in the lymphoid structures, i.e., bursa, spleen, thymus, Harderian gland, and caecal tonsil.

Changes in the bursa comprise the initial inflammatory response with hyperaemia, oedema, and infiltration of heterophils, accompanied by B lymphoid cell necrosis. Proliferation of interfollicular connective tissue and bursal epithelium gives it a glandular appearance. With the decline in the acute inflammatory response, the cortico-medullary epithelium proliferates and cystic cavities develop in the medullary areas of the follicles.

In the spleen there is some necrosis of lymphoid cells. The thymus and caecal tonsils show some cellular reaction in the lymphoid tissues in the early stages, but the damage is less extensive. There may be plasma cell depletion in the Harderian gland. Microscopic lesions of the kidney are non-specific. The liver may show slight perivascular infiltration of monocytes.

DIAGNOSIS

- The history, clinical evaluation of cloacal bursa and gross lesions are adequate for the diagnosis of acute disease.
- **Serological Tests** for identification of IBDV using AGID, Immunoflourescence and ELISA. Antigen-capture enzyme-linked immunosorbent assay (AC-ELISA) using polyclonal antibodies have a higher sensitivity and used for rapid diagnosis.
- Diagnosis can be confirmed by using macerated bursa as antigen in a gel diffusion test, or in an ELISA test, against a known positive antiserum; by microscopic examination of tissues for typical lesions, or the presence of antigen following immunoperoxidase staining. The detection of antigen by immunofluorescence in frozen bursal sections, or smears, may also be used.
- Reverse Transcriptase-Polymerase Chain reaction (RT-PCR) is useful for confirmative detection of virus, as it is a dsRNA virus.
- Virus isolation is rarely used for diagnosis, as it is time consuming. Bursa is the most commonly used tissue for isolation and identification of the causative agent. Most strains can be grown on the chorio-allantoic membrane of 10-11 day-old embryonated eggs.

DIFFERENTIAL DIAGNOSIS:

Differential diagnosis of acute IBD should take into account other diseases that can induce sudden death in young chickens, with either haemorrhages or nephritis or bursal lesions. This includes infectious diseases such as (i) Newcastle disease (ND), (ii) Chicken Anaemia, and infections by (iii) Infectious Bronchitis viruses with nephropathogenic tendencies.

In case of subclinical IBD, differential diagnosis may be necessary and this should include **Coccidiosis**, **Ranikhet disease**, **haemorrhagic syndrome of muscles** and other haemorrhages, **Avitaminosis-A**, fatty liver and kidney syndrome, **water deprivation** with swollen kidneys, and excess renal urates.