INFECTIOUS BURSAL DISEASE
(IBD; Gumboro Disease)

DEFINITION

Infectious bursal disease (IBD) is an acute, contagious, viral disease of young chickens characterized by diarrhea, vent picking, trembling, incoordination, inflammation followed by atrophy of the bursa of Fabricius, and by variable degrees of immunosuppression.

OCCURRENCE

IBD occurs primarily in chickens. Clinical signs and mortality are generally more severe in birds 3-6 weeks old. However, IBD may occur in chickens as long as they have a functional bursa of Fabricius (1-16-weeks of age). Birds infected at less than 3 weeks of age do not have clinical signs. However, destruction of the bursa results in immunosuppression. The younger the bird at the time of infection, the more severe the immunosuppression, resulting in a high degree of susceptibility to subsequent pathogens. Once a premise has been contaminated with IBD virus, the disease tends to recur, usually as a subclinical infection.

In turkeys, subclinical infection with IBD virus occurs without immunosuppression. However, there is no known disease associated with IBD viral infection. Most of the IBD viruses from turkeys are serologically distinct from those isolated from chickens. Ducks can also be subclinically infected with no resultant immunosuppression.

IBD now occurs in all of the major poultry-producing countries of the world.

HISTORICAL INFORMATION

In 1962, “avian nephrosis”, a condition now believed to be IBD was reported to be occurring on farms near Gumboro, Delaware. Initially the disease was confused with a variant form of infectious bronchitis accompanied by nephrosis, but the disease is now well delineated and well characterized. The immunosuppressive effects of IBD were first reported by Allan in 1972. Variant strains of serotype 1 IBD were found in the Delmarva region in the 1980’s. Very virulent strains of IBD have been reported in The Netherlands, Africa, Asia, and South America. In the United States the disease is a persistent problem in the broiler industry despite vaccination.

ETIOLOGY

1. IBD is caused by a virus belonging to the family Birnaviridae. The viral genome has two double-stranded RNA segments. Other similar viruses occur in fish and mollusks. The virus may be propagated in chicken embryos or chicken embryo cell cultures. Two serotypes exist, with only serotype 1 being pathogenic. Within serotype 1, there are six unrelated or partially related strains.

2. The virus is very resistant to most disinfectants and environmental factors. It persists for months in contaminated houses and for weeks in water, feed, and droppings. It can be transmitted by fomites. It has some susceptibility to formalin and iodide disinfectants. Invert soaps with 0.05% sodium hydroxide may kill IBD virus.

3. The virus is lymphocidal (immunoglobulin-bearing lymphocytes) and severely damages the bursa of Fabricius. The thymus, spleen, and cecal tonsils are also damaged but less severely.

4. It has been demonstrated that IBD can severely damage the humoral responsiveness of susceptible chicks when they are infected at less than 3 weeks of age. Those chicks then do not respond properly when vaccinated against other diseases. There is evidence that inclusion body hepatitis and gangrenous dermatitis occur frequently in such flocks. Some live vaccines may have a similar potential for damage as field infections.
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5. The passive transfer of maternal antibodies to baby chicks is very important for the prevention of early infections with the virus. Breeder flocks must receive vaccines or field exposure to the virus followed by booster vaccinations to stimulate high levels of maternal antibody. Progeny from well-immunized breeder flocks will resist infection for 2-4 weeks. Passive immunity will interfere with vaccinations and it is necessary to vaccinate chickens after maternal immunity has fallen to a point that the vaccine will overcome the lower levels of maternal antibody. If chicks are from nonimmune flocks, vaccinations should be administered at hatch.

EPIZOOTIOLOGY

1. The virus spreads rapidly from infected chicks and from contaminated premises or fomites to susceptible chicks. The disease is highly contagious.

2. Transmission of virus through the egg does not appear to occur and there is no evidence for a carrier state.

3. The lesser meal worm (Alphitobius diaperinus) harbors the virus for weeks after an outbreak and may transmit it to susceptible birds. The worm lives in poultry litter.

4. The incubation period is very short with clinical signs evident 2-3 days post exposure.

5. Subclinical infection (before 3 weeks of age) is economically important due to suppression of humoral immunity and subsequent secondary infections.

CLINICAL SIGNS

1. Clinical disease is observed only after 3 weeks of age. There is a sudden onset, particularly with the first outbreak. There may be tremor or unsteadiness. There is depression, anorexia, ruffled feathers, and a droopy appearance that resembles coccidiosis.

2. Diarrhea and dehydration are usually present. Occasionally there is voiding of blood and straining during defecation. Vent picking is common and may be self-inflicted.

3. Morbidity is very high. Mortality is usually low although it can be substantial (approaching 30%) if husbandry is poor or if strains are particularly virulent. Mortality in a flock has usually peaked and receded within a week of onset. IBD tends to be more severe in leghorn strains than in broiler stock.

LESIONS

1. Initially the bursa is enlarged to about twice normal size, severely edematous [Fig. 1; Infectious bursal disease; AAAP], and reddened. It may contain hemorrhages [Fig. 2; Infectious bursal disease; Cornell U]. The swelling recedes about the 5th day and the bursa atrophies rapidly until 8-10 post infection. There is increased mucus in the intestine. Bursal lesions are evident in both clinically and subclinically infected birds.

2. In field outbreaks, hemorrhages are common in thigh and pectoral muscles and, perhaps, at the junction of the proventriculus and gizzard.

3. The parenchymatous organs, especially the kidneys, may be swollen. The ureters may contain urates.

4. Necrotic lesions/atrophy may also be found in other lymphoid organs such as the thymus and the spleen, particularly with highly virulent IBD strains.

5. Microscopically, in the bursa there is lymphoid follicle depletion and destruction followed by atrophy. Similar changes occur in the spleen, thymus, and cecal tonsils but they recover more rapidly and completely than does the bursa.
6. Some variant strains of the virus cause few clinical signs and minimal gross acute changes in the bursa. However, these variant strains may induce rapid bursal atrophy and severe immunosuppression.

DIAGNOSIS

1. In an acute outbreak in susceptible chicks, the short course and bursal lesions are very suggestive of IBD. Signs and lesions can be less apparent in subsequent outbreaks and in chicks with parental antibody.

2. Paired serologic testing with rising titers using the ELISA, agar-gel precipitin, or virus neutralization will usually confirm the diagnosis. However, the virus neutralization test is the only serological assay that will identify the infecting serotype or strain (subtype).

3. If susceptible chicken embryos and known-positive antiserum are available in a laboratory, the virus can be isolated from the bursa or spleen and then identified by neutralization. The virus neutralization assay, PCR and antigen-capture enzyme immunoassay with monoclonal antibodies can be used to differentiate serotype 1 subtypes.

4. Microscopic bursal lesions are said to be specific for IBD in the early stages of the disease. The direct fluorescent antibody technique and electron microscopy also can be applied to virus-containing tissues.

5. Coccidiosis, hemorrhagic syndrome, and adenoviral infection must be differentiated from IBD infection.

CONTROL

1. Vaccination of breeders to confer immunity to progeny is an effective method of reducing the disease in young chicks. Killed oil-emulsion vaccines are effective in producing high levels of antibody in breeders, after priming with a live vaccine applied prior to the onset of lay.

2. Chicks can be vaccinated against the disease but timing the vaccination in maternally immune chicks can be difficult. When maternal antibodies wane use of “hot” vaccines in nonimmune chicks may result in bursal atrophy. Vaccination with milder vaccines will not be effective in birds with high levels of maternal antibody. Therefore, knowledge of passive antibody levels and correct timing are necessary for successful vaccination.

3. An in ovo immune complex vaccine is available that results in decreased vaccine pathogenicity without loss of immunogenicity.

4. Sanitation programs are rarely successful due to the highly resistant nature of the virus.

TREATMENT

Treatment is of no value. However, good husbandry and adequate temperature may reduce the severity of the disease.