Infectious Bursal Disease (Gumboro) in Commercial Broilers [1]

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Infectious bursal disease (IBD) is an acute and highly contagious viral infection of immature chickens. IBD is characterized by destruction of lymphocytes in the bursa of Fabricius (BF) and to a lesser extent in other lymphoid organs. The disease is a major problem in concentrated poultry production areas throughout the world. However, it is often not recognized due to a subclinical form. Affected chickens have reduced antibody response to vaccinations, strong post vaccinal reactions, and increased susceptibility to concurrent or secondary infections.

Characteristics of the IBD Virus

IBD is caused by a birnavirus. The virus is resistant to many disinfectants and environmental factors, and remains infectious for at least four months in the poultry house environment. Because of the resistant nature of the IBD virus, once a poultry house becomes contaminated, the disease tends to recur in subsequent flocks.

Pathogenesis of IBD

To better understand how the IBD virus adversely affects the chicken's immune system, relevant factors of this system's early development will be described. During embryonic development, and through approximately 10 weeks of age, immune system cells (lymphocytes) travel to the BF to become programmed to become antibody-producing cells. If the IBD virus damages the BF in young chickens, the BF will not be capable of programming sufficient numbers of lymphocytes. Thus, the chickens will experience reduced immune system capabilities (immunosuppression).

The earlier the damage to the BF occurs, the few lymphocytes with antibody-producing capability will be programmed. Therefore, any IBD virus control program should attempt to protect the BF as long as possible. In practical terms, if the BF can be protected against disease until at least 3 weeks of age, an adequate number of lymphocytes should be programmed, and the immunosuppressive effects of an IBD outbreak should be ininal.

Transmission of IBD Virus

Chickens infected with the IBD virus shed the virus in their feces. Feed, water, and poultry house litter become contaminated. Other chickens in the house become infected by ingesting the virus. The lesser mealworm (Alphitobus diaperinus) has been shown to carry the virus. Because of the resistant nature of the IBD virus, it is easily transmitted mechanically among the farms by people, equipment and vehicles.

Subclinical and Clinical IBD

Infectious bursal disease follows one of two courses, depending on the age at which chickens are infected. The subclinical form of the disease occurs in chickens less than

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3 weeks of age. Chickens present no clinical signs of disease, but experience permanent and severe inirnunosuppression. The reason young chickens exhibit no clinical signs of disease is not known. However, immunosuppression occurs due to damage to the BF. The majority of field infections are subclinical, and this form is the more economically important form of the disease.

Broiler integrations commonly have farms described as problem farms. Broilers grown on these farms typically have poor body weights and feed conversions, high mortality, excessive reactions to respiratory vaccines, and high rates of condemnation at processing. In many cases, investigations have shown that these farms are heavily contaminated with the IBD virus. The poor performance of the broilers is due to factors relating to immunosuppression caused by subclinical IBD.

The clinical form of IBD usually occurs in chickens from 3 to 6 weeks of age. The clinical disease has a sudden onset, and the mortality rate in the flock increases rapidly. Clinical signs of disease include dehydration, trembling, ruffled feathers, vent pecking, and depression. Affected chickens experience a transient immunosuppression. On necropsy, the principle lesions are found in the BF.

Gross Lesions

Initially, the BF is swollen (inflamed); appears edematous and hyperemic; and has a gelatinous yellowish transudate covering the serosal surface. Hemorrhage and areas of necrosis may be present in more severe cases. Five days after infection, the BF diminishes in size rapidly (atrophies).

Necrosis and depletion of lymphocytes also occur in the secondary lymphoid organs, including the spleen, glands of Harder, and cecal tonsils. These organs are typically affected less severely than the BF and may recover following infection.

Hemorrhage may be present in the thigh and pectoral muscles, because the IBD virus interferes with the normal blood clotting mechanism. The kidneys may appear swollen in birds that die or that are in the advanced stages of the disease. Such lesions probably result form severe dehydration, not direct viral damage.

Microscopic Lesions

Microscopically, lymphocyte necrosis is present in the BF within 36 hours after infection. By 48 hours, few lymphocytes are present. Edema, hyperemia, and inflammatory cell infiltration are evident, which account for the enlarged BF during the initial days following IBD virus infection. By 8 to 12 days after infections, the BF is shrunken to less than one-fourth of its original size. The lymphoid follicles are cystic and depleted of lymphocytes. The epithelium lining the BF is irregular and infolded. Fibroplasia is present in the interfollicular connective tissue.

In severe cases of IBD, all the follicles are affected simultaneously. In less severe cases, only scattered follicles are affected, and the lesions spread to other follicles. Typically, the follicles in the tips of bursal folds are affected first. Factors that determine the severity of an infection include virulence of the IBD virus, concentration of IBD virus exposure, level of immunity against IBD virus, and management factors.

Diagnosis of IBD

Diagnosis of IBD involves consideration of the flocks' history, and of the clinical signs and lesions. Obviously, chickens less than 3 weeks of age present no clinical signs of disease, while chickens greater than 3 weeks of age present clinical signs as described. The severity of the clinical signs will depend upon the factors described. Confirmation of a diagnosis of clinical IBD can be made at necropsy by examining the BF during the early stages of disease for characteristic gross lesions.

During later stages of disease it is difficult to confirm a diagnosis of IBD by examining only shrunken, atrophied BF, as other diseases (for example, Marek's disease, mycotoxicosis) produce similar changes. In birds less than 3 weeks of age or in young chickens with maternal antibodies, IBD virus infections are usually subclinical. Thus, typical clinical signs are not present, and diagnosis should be supported by histopathologic study of suspect BF, serologic studies, or by virus isolation.

Prevention and Control of IBD

An effective IBD prevention and control program must involve an effective breeder vaccination program, an effective biosecurity program, and an effective broiler vaccination program. Immunization of breeders is an important part of the IBD control program. Antibodies produced by the hen are passed through the egg to the broiler chick. These maternal antibodies, if present in adequate levels, protect the chicks against subclinical IBD. An example of a comprehensive breeder vaccination program where subclinical IBD is a problem might have a vaccine schedule such as this: at 12 to 15 days of age—IBD live; at 30 to 33 days of age—IBD

live; at 85 days of age—IBD live or inactivated; and at 120 days of age—IBD inactivated.

Revaccinate at 38 to 42 weeks of age with an inactivated IBD vaccine if breeder titers are low or of poor uniformity. Routinely monitor breeder IBD antibody titers to ensure vaccines are administered properly and that the chickens respond appropriately.

Effective control of IBD in commercial broilers requires that field virus exposure be reduced by proper clean-up and disinfection between flocks, and that traffic (people, equipment and vehicles) onto the farm be controlled. The development and enforcement of a comprehensive biosecurity program is the most important factor in limiting losses due to IBD.

Phenolic and formaldehyde compounds have been shown to be effective for disinfection of contaminated premises. Efforts at biosecurity (cleaning, disinfecting, traffic control) must be continually practices, as improvement is gradual and often only seen after 3 or 4 flocks.

A third factor to consider in the IBD prevention and control program is vaccination of the broilers to prevent clinical IBD. Three categories of vaccines, based on their pathogenicity, have been described: 1) mild, 2) intermediate, and 3) virulent. The intermediate type IBD vaccines are most commonly used. These vaccines can stimulate the broiler to produce antibodies earlier than the mild-type vaccines, without significant damage to the BF as may occur with the virulent type vaccines.

The timing of broiler vaccination depends on the level of maternal antibody present in the chicks. High levels of maternal antibody at the time of vaccination will neutralize the vaccine virus. Thus, only a limited active immune response results, and chickens will be susceptible to disease as maternal titers decrease. If low levels of maternal IBD titers are present in the chicks, vaccination may not be effective on farms contaminated with virulent field virus.

Approximately 10 to 12 days are required after vaccination for chickens to develop minimal protective titers. During this "lag time," chickens are susceptible to IBD. In addition, virulent IBD viruses are able to break through higher maternal titers than milder vaccine viruses. Thus, if IBD field virus contamination on a broiler farm is high, nor broiler vaccination can stimulate protection in the flock before damage occurs.

If the maternal antibody titer is not uniform in the broiler flock, multiple costly vaccinations will be required. For example, some producers may vaccinate broilers at one day of age and again at fourteen days of age. This multiple IBD vaccination would be recommended when maternal titers are poorly uniform, which results from poor vaccine administration in breeders or when mixing broilers from different breeder flocks. In a recent study, even a group of breeders that had fairly uniform IBD titers had chicks with titers that were variable, with many chicks have little or no maternal antibody protection.

Although the 1 day of age vaccination would be of little direct benefit to broilers with high maternal titer levels, multiple vaccinations would provide some protection to chicks with lower levels of maternal antibody and would help reduce replication of IBD field virus and subsequent shed in the poultry house environment.

The important factors to consider in the control of IBD are the prevention of broiler losses through an effective IBD breeder vaccination program (maternal titers) and decreasing exposure through a comprehensive biosecurity program. Relying on broiler vaccination has met with only limited success when not coordinated with effective breeder vaccination and biosecurity programs.

Variant Strains of IBD

Control of IBD has been further complicated by the recognition of variant strains of the IBD virus. Variant viruses induce damage in the BF in chickens, even when high and uniform antibody titers are present. Variant strains do not cause obvious clinical disease, but induce severe immunosuppression. The BF of affected chickens undergo rapid atrophy (lymphocyte depletion) without the inflammatory changes observed early in the infection with the classical IBD viruses. These variants are not from a different serotype, but are antigenically different enough to cause problems.

Often IBD is a serious problem in an integration, and losses occur despite persistent efforts at reducing field virus exposure through a biosecurity program, maintenance of adequate and uniform maternal titers, and an effective broiler vaccination program. In this case, consideration should be given to vaccinating breeders with inactivated vaccines containing standard and variant strains of the IBD virus.