INFECTIOUS BURSAL DISEASE

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INTRODUCTION

A disease entity affecting the cloacal bursa was first reported by Cosgrove in 1962. He referred to the condition as "avian nephrosis" because of severe kidney damage in birds that succumbed to the disease. The disease was also referred to as "Gumboro disease" because it was first observed on farms near Gumboro, Delaware. Winterfield and Hitchner (1962) and Winterfield et al. (1962) provided the information which determined that the nephrosis observed with infectious bursal disease was similar to that caused by some strains of infectious bronchitis virus. Hitchner (1970) proposed the name for the disease that we now recognize as infectious bursal disease (IBD). The virus is a unique type of virus and similar viruses are not found in mammals, as yet. Similar viruses do exist in fish, bivalve mollusks and the fruit fly. The virus has been shown to have world-wide distribution and is of great economic significance because of two manifestations of infection, i.e. 1) immunosuppression and 2) clinical disease. The virus also naturally infects turkeys and ducks, however, no disease has been consistently seen in these species. There is evidence that the turkey has mild suppression of the cell mediated immune response.

CLINICAL FEATURES

Flock History

Age Incidence: Infections with IBD virus can occur at any time, however, the critical ages are 1 day to 2 weeks of age for severe immunosuppression and 3 to 6 weeks for the clinical disease. Clinical disease, however, can occur in susceptible light breeds up to 18 weeks of age.

Genetic Influence: There does appear to be a difference in the severity of the clinical disease between light and heavy breeds with light breeds being the most susceptible. There have been no reported differences in susceptibility between commercial strains.

Seasonal Incidence: There is no reported difference in a seasonal incidence of the disease.

Flock Distribution: Initially, this disease appeared to be confined to localized areas as far as the occurrence of the clinical disease. This observation was true prior to the discovery of non-clinical immunosuppressive infections. Serologic surveys, however, did indicate that nearly all flocks had antibodies to the virus and these surveys were done prior to the availability of vaccines. In retrospect it is apparent that there was considerable variation in the virulence of strains and the occurrence of the disease in localized areas reflected differences in virulence and the prevalence of maternal antibodies in hatched chicks.

Morbidity and Mortality: The virus is highly contagious and spreads rapidly through a flock. The incubation period is 2 to 3 days and the entire flock will exhibit signs in severe clinical cases. Mortality rates may be as high as 20-30% but is quite variable. The course of the disease is usually 5 to 7 days.

Influence of Complicating Disease: As indicated previously, the first descriptions of this disease were confused with infectious bronchitis which also caused a nephrosis. The principal concern with IBD infections

is the immunosuppressive effect that it exerts on other infections. The disease conditions that are exacerbated by prior IBD infection are gangrenous dermatitis, inclusion body hepatitis and hemorrhagic anemia. With the control of early IBD infections by maternal antibody, these conditions are rarely seen now. If they occur it is a good indication that the IBD vaccination program is inadequate, or the possible presence of a different serotype.

Clinical Signs

Appearance of Affected Chickens: The onset of the disease is rapid and chickens exhibit severe prostration, incoordination, whitish or watery diarrhea, soiled vent feathers and vent picking. The majority of the flock will exhibit these signs and mortality will continue from the first day of signs until 4 to 5 days

Slide 1 - A typically depressed chicken with ruffled feathers and evidence of watery diarrhea. In houses the litter will be very slippery from the copious watery diarrhea covering the floor. Birds will not drink or eat during the acute phase of the disease. Because of the 5 to 7 day course of the disease, market birds will be delayed in their weight gains by 4 or 5 days.

PATHOLOGIC FEATURES

Gross Lesions: The gross lesions are limited to the cloacal bursa and musculature of the legs, thighs and breast. There may be kidney lesions characterized by urate deposits in the tubules. These lesions are, however, secondary and a result of dehydration. Blood from affected birds has an extended clotting time.

Slide 2 - Cloacal bursa that is swollen and edematous. Bursas are yellow to greenish rather than the normal white to gray color. This type of bursa is most often encountered in birds that are sick and have been sacrificed for postmortem examination.

Slide 3 - Some bursas exhibit extensive hemorrhage and this type of lesion is most often seen in birds that have succumbed to the disease.

Slide 4 - Hemorrhages of the leg, thigh and breast muscles are very common, however, they may be absent in some birds. Similar type hemorrhages may be seen in inclusion body hepatitis. The lesions in the cloacal bursa would differentiate the two diseases.

Slide 5 - Small atrophied cloacal bursa in a bird that has recovered from infectious bursal disease. Because of the destruction of the lymphoid follicles of the bursa, the size will be greatly diminished and the follicles will not regenerate.

Slide 6 - Two small atrophied cloacal bursas in 6-week-old broilers in comparison to a normal cloacal bursa from a similar aged bird. The cloacal bursa of a normal chicken at this age will be 15-20 cm (1/2 to 3/4 in.) in diameter. Atrophied bursas will be 5-7 cm in diameter.

Slide 7 - A cheesy "core" may sometimes be found in the lumen of the cloacal bursa. This represents caseous material deposited in the lumen as a result of the extensive necrosis and inflammation of the bursal follicles during the acute phases of the disease.

Microscopic Lesions: Microscopic lesions are most evident in the cloacal bursa but changes may also be seen in the spleen, cecal tonsil, thymus and gland of Harder. The cloacal bursa undergoes extensive necrosis of the lymphocytes making up the follicles. Changes can be seen as early as 1 day postinfection with all lymphoid follicles being affected by 3 or 4 days. Degeneration and necrosis of lymphocytes is followed by edema, hyperemia and accumulation of heterophils. As the inflammation declines, cystic cavities develop in the medullary areas of the follicles and there is fibroplasia of the interfollicular connective tissues. Ultimately there is chronic atrophy of the follicles and some follicles remain cystic. The bursal epithelial layer becomes more prominent and glandular in appearance with columnar epithelial cells containing globules of mucin (Cheville, 1976).

On the third day postinfection, the spleen will exhibit lymphoid necrosis of germinal follicles and the periarteriolar lymphoid sheath. The spleen recovers rather rapidly with no sustained damage to germinal follicles. The damage to the thymus and cecal tonsil is similar and rapid repair and recovery occurs by 5 to 6 days postinfection.

Early infections by IBD affects the plasma cell population of the gland of Harder. After an infection of 1-day-old chicks the populations of plasma cells are 5- to 10-fold less than uninfected birds (Dohms et al. 1981).

Slide 8 - Normal cloacal bursal follicles showing the medullary and cortical regions.

Slide 9 - Necrosis and inflammation of a bursal follicle 3 days post-infection. Note pyknotic nuclei of necrotic lymphocytes and fibrinous exudate in the medullary area as well as collapsing of the follicle.

Slide 10 - Cystic cavities in the necrotic bursal follicles as the inflammatory reaction subsides in the follicular areas of the bursa.

Slide 11 - Bursa from a bird recovered from infectious bursal disease showing the chronic atrophy with fibroplasia of the interfollicular tissue and absence of follicles. The epithelial layer is prominent and glandular in appearance.

Slide 12 - Atrophied bursa with a cystic follicle. Other follicles are showing the changes of chronic atrophy.

ETIOLOGY

Isolation of the Virus: Several methods may be used to isolate IBD virus. The embryonating egg from susceptible flocks or lymphoid cell cultures derived from the bursa are the most sensitive methods for cultivating the virus. Chicken embryo fibroblast cultures are sensitive to some strains of the virus or they are sensitive to virus serially passed in embryos or bursal cells. The chorioallantoic membrane of 9- to 11-day-old embryos is the preferred route of inoculation and deaths of some embryos occurs in 3 to 5 days. The IBD virus produces cytopathic effects in cell culture and plaques are formed under agar after 3 or 4 days incubation. Rapid diagnosis of IBD may be made by the staining of impression smears of the cloacal bursa with fluorescein labelled anti-IBD virus antibody. Samples for staining should be collected during the acute phase of the disease.

There is a difference between the sites of replication of virulent and avirulent strains of IBD virus in the cloacal bursa. Virulent strains replicate in the lymphocytes of the medullary and cortical areas of the

follicles while avirulent strains only replicate in the interfollicular tissues of the bursa. This would explain why the virulent virus is immunosuppressive and avirulent vaccine strains are not. It should be remembered, however, that all vaccine strains are not avirulent and are capable of causing immunosuppression in susceptible birds that are not protected by maternal antibody.

Slide 13 - Flourescent antibody (FA) stained lymphocytes in an acetone fixed impression smear of the cloacal bursa 3 days postinfection with IBD virus. The staining of such smears with fluorescein tagged antibody may be used to rapidly and definitively diagnose IBD. If smears are made of the bursa during the recovery stages of the disease there may be an absence of intact infected cells and FA staining would be negative. Virus, however, can be isolated for at least 10 to 14 days postinfection.

Slide 14 - Immunofluorescent staining of a section of cloacal bursa infected with a virulent strain of IBD virus 2 days postinfection. The virus is replicating in cells throughout the follicle.

Slide 15 - Immunofluorescent staining of a section of cloacal bursa infected with an avirulent vaccine strain of IBD virus 2 days postinfection. The virus is replicating in cells of the interfollicular tissue.

Slide 16 - Electronmicrograph of IBD virus exhibiting viral particles with an approximate diameter of 68 nm. They are non-enveloped and have icosahedral symmetry. Many particles are empty and represent incomplete non-infective virions. The virus has 2 segments of double stranded RNA and has been placed in a family called the Birnaviridae.

Slide 17 - Plaques produced by 3 different strains (C, T and V) of IBD virus. Note that the C & T strains have a mixture of large and small size plaques. The V strain is a cloned strain of IBD virus and has only the large size plaque.

Slide 18 - Cell culture cells stained by FA and showing that the virus replicates in the cytoplasm and the nucleus is unstained.

Serology: It is very important that the immune status of breeder flocks is known. The primary method for the control of the immunosuppressive effects of IBD is through the transfer of maternal immunity. Therefore, it is imperative that the breeder flocks have a high uniform level of antibody so that chicks will be protected for the first 2 to 3 weeks of life. Several serologic procedures are available to detect antibodies in breeders. The agar gel precipitation test is rapid but it is only qualitative and not quantitative. It is no longer used extensively and has been replaced with the constant virus-diluting serum virus neutralization test or the enzyme linked immunosorbent assay (ELISA) procedure. Both of the tests are quantitative and are preferable to the agar gel precipitation test. The ELISA procedure is available commercially and is very sensitive while giving results in hours rather than days. It is certainly the test for the future.

At least 2 and possibly 3 serotypes of the IBD virus exist. The significance of antigenic variation of this virus has not been apparent. The virus is an excellent antigen and induces exceptionally high antibody titers in chickens.

Slide 19 - An agar gel precipitation test with antigen prepared from infected cloacal bursas placed in the center well. Serum is added to surrounding wells and of the 6 sera represented in this slide, 5 are positive and 1 is negative.

Slide 20 - A microtiter cell culture virus neutralization test. Cell control wells are represented in the 12th well of each row at the far right. Virus controls are in well 11. Antibody is diluted 2-fold from left to right. The eight sera in this slide have titers of 64, 32, 128, 128, 16, 32, 16 and 16, respectively as they protect the cells from destruction by 100 infectious doses of virus at these dilutions. The plates are fixed in ethanol after 72 hours of incubation and stained with 1% aqueous crystal violet solution to visualize the cell monolayers.

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