Recent trends in control of Infectious Bursal Disease in Poultry

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Importance

Infectious bursal disease (IBD), an immunosuppressive disease of young chickens, has been responsible for major economic losses in the poultry industry worldwide including India. The disease affects lymphoid organs, leading to destruction of bursa of Fabricius an important lymphoid organ of poultry leading to immunosuppression. Young chicks are highly susceptible to the virus between 3 and 6 weeks of age. The economic impact of Infectious Bursal Disease (IBD) in India extends beyond the 30% flock mortality, which is frequently recorded in broilers on pullet replacements. Modern trends for Prevention and control of IBD in broiler flocks depends on biosecurity measures which limit introduction of virus into flocks combined with high maternal antibody protection and subsequent administration of attenuated vaccines.

Background

Infectious bursal disease (IBD) is an important viral disease of poultry throughout the world. The disease is also known as Gumboro because it was first recognized in Gumboro district of USA. IBD is an acute contagious viral disease of immature chickens and is characterized by destruction of lymphocytes in the bursa of fabricius 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 subclinical form. Affected chickens have reduced antibody response to vaccination and increased susceptibility to concurrent or secondary infections.

Cause

Infectious bursal disease is caused by a birnavirus. The virus is resistant to many disinfectants and environmental factors, and remains infective for at least four months in the poultry shed and surroundings. Because of the resistant nature of the IBD virus, once a poultry house becomes contaminated the disease tends to recur in subsequent flocks. During embryonic development and through approximately 10 weeks of age immune system cells (Lymphocytes) travel to the bursa to become programmed as antibody producing cells. If the IBD virus damages the bursa in young chicken, the bursa will not be capable of programming sufficient numbers of lymphocytes. Thus, chickens will experience reduced immune system capabilities (immune suppression). Chickens infected with IBD virus shed the virus in their feces thereby contaminating feed water and poultry house litter. Other chicken in the house becomes infected by ingesting the virus. It is also easily transmitted mechanically among the farms by people, equipment and vehicles.

Symptoms and lesions

Chickens present no clinical signs of disease but experience permanent and severe immune suppression. The reason young chickens exhibit no clinical sign of disease is not known. However, immune suppression occurs due to damage to the bursa. The majority of field infections are subclinical, and this form is the more economically important form of disease. Broilers grown on these farms typically have poor body weight, feed conversions, high mortality excessive reactions to respiratory vaccines and high rate of condemnation at processing. In many cases investigations have shown that these farms are heavily contaminated with IBD virus. The clinical form of IBD usually occurs in chickens from 3 to 6 weeks of age. The clinical disease has a sudden onset and mortality rate in the flock increases rapidly. Clinical signs of disease include dehydration, trembling, ruffled feather, vent picking and depression. Affected chickens experience a transient immune suppression. On necropsy the principal lesions are found in the bursa which is swollen; 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 bursa diminishes in size rapidly (atrophhies). 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 advanced stages of the disease along with deposition of uric acid salt on kidney.

Diagnosis

Diagnosis of IBD involves consideration of the flock 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 clinical signs will depend upon the factors described. Confirmation of a diagnosis of clinical IBD can be made at necropsy by examining the bursa during the early stages of disease for characteristic gross lesions. During later stage of disease, it is difficult to confirm a diagnosis of IBD by examining only shrunken, atrophied bursa, as other diseases (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 BURSA, serologic study, or by virus isolation.

Prevention and control

Poultry producers world-wide have developed specific strategies to prevent and control mortality and immunosuppression due to infectious bursal disease. The use of attenuated vaccine from the early 1970s onwards has ameliorated the effect of clinical IBD. With the emergence of variant strains in the United States during the mid-1980s, greater reliance was placed on maternal antibody protection initiated by priming the immune system with a live attenuated vaccine followed by administration of oil emulsion vaccines to boost immunity. The appearance of the highly pathogenic form of IBD (vvIBD) has dictated new approaches to vaccination involving the use of less-attenuated vaccine strains which can stimulate immunity in young chicks in the presence of significant level of maternal antibody. Recent studied have shown that vvIBDV is similar to the type 1 classic strain but the virus possesses an additional antigenic site donated by a monoclonal antibodyMAb21.The intermediate-plus (or "hot") vaccines also possess this antigen. The economic impact of vvIBD in India extends beyond the 30% flock mortality, which is frequently recorded in broilers on pullet replacements. Losses include death and downgrading due to secondary bacterial and viral infections following immunosuppression. In addition, disruption of the supply-demand equilibrium in the market place results in an escalation in cost of eggs and broiler meat to consumer. Ultimately, reduced egg production and elevated mortality are reflected in a lowered nutritional quality of diets for Indian population. Prevention and control of IBD in broiler flocks depends on measure which limit introduction of virus into flocks combined with high maternal antibody protection and administration of attenuated vaccines.

Biosecurity

The avibirnavirus responsible for vvIBD can resist a temperature of 56 degree Celsius for 5 hours, it unaffected by pH value of 2, exposure to 0.5% phenol for 1 hour or by 0.5% formalin for 6 hours. Persistence of birnavirus in units housing infected flocks confirms the ability of the IBD virus to remain viable and to infect successive cycles of broilers or replacement pullets. Although thorough decontamination of housing will not ensure that a successive flock will be free of IBD, removal of litter and disinfection of equipments and concrete floors are recommended, especially if severe losses have occurred. Decontamination of earth floored building is extremely difficult, but removal of top 2 cm of soil may be attempted. Feather, dander (poultry dust) and biological material should be removed from ceilings, walls and equipments using a pressure sprayer followed by application of an effective iodophor or quaternary compound. Rodents and Alphatobius beetles have been shown to be reservoirs of IBD virus, and appropriate rodenticides and insecticides should be used at the end of each cycle. Residual feed should not be transferred to other farms in areas where IBD is prevalent.

The necessarily of maintaining complete separation of broiler, breeder and commercial pullet flocks of different ages is to prevent cross transmission of IBD among and within farms. Field studies in India have confirmed that multi aged flocks, or units in close proximity, are severely and consistently affected by vvIBD in contrast to units which are well spaced and where adequate biosecurity is maintained. Persistent infection with vvIBD in biologically contagious farms results in recurrent losses in flocks aged from 20-40 days. Appropriate measures to separate types and ages of flocks and acceptable biosecurity procedures are necessary components of IBD control. These measures require capital investment in fencing, showers and moveable equipment for each farm.

Vaccination

Selecting a vaccination programme for broiler and commercial layers involves the following considerations:

1. Size and scope of operation-Operation of single or multiple-age facilities.
2. Standard of biosecurity- Density of poultry population in the area.
3. Pathogenicity of IBD virus to which flocks may be exposed.
4. Availability of live attenuated (intermediate plus or "hot") and inactivated vaccines.
5. Source of chicks, whether from company operated parent flocks or from purchased commercial eggs or chicks.

The significant component of control by vaccination requires solid immunization of parent stock to produce chicks with high and uniform levels of maternal antibody. Breeding flock operators can achieve an acceptable antibody level in parents by priming the immune system with at least 2 successive live attenuated vaccines followed by administration of one or two inactivated oil emulsion vaccines. In many situations growers purchase day old with highly variable levels of maternal immunity carried over from grandparent flocks either maintained locally or imported. These flocks may be housed in units which have been

improperly disinfected or due to deficiencies in biosecurity, early challenges with IBD virus of moderate to high Pathogenicity may occur. Early exposure results in severe damage to immune system. These parent flocks may show gangrenous dermatitis, necrotic enteritis, or coccidiosis during the rearing period. Immunosuppressed flocks show a suboptimal response to live attenuated priming vaccines against respiratory diseases such as Newcastle disease (NCD) and Infectious bronchitis (IB). The degree of maternal immunity against IBD and the intensity of challenge will determine the subsequent vaccine response against IBD and other diseases. It is possible that a severely immunosuppressed parent flock will produce chicks with highly variable maternal antibody levels for the duration of the laying period. The problem facing the parent flock operator is to select a vaccination programme which primes the immune system without causing any appreciable burasal damage which may compromise subsequent immune response. If parent stock pullets are delivered with a high, uniform maternal antibody level, it is advisable to administer a mild strain IBD vaccine at 18 to 20 days of age. This assumes placing flocks in thoroughly decontaminated housing, using an all-in all-out placement programme, implementing high standards of biosecurity and the absence of highly pathogenic (vv IBD) field challenge. If moderate risk factors are present, it is advisable to administer a mild strain vaccine by injection at day old or alternatively, substitute an intermediate strain vaccine capable of stimulating immunity in the presence of low levels of maternal antibody.

Under extreme conditions where severe risk factors are present, survival is the critical objective and some measures of bursal damage can be tolerated. Accordingly, the administration of an intermediate plus strain of IBD vaccine may be used as an interim measure until decontamination procedures, single flock placement, and improved biosecurity are affected. A programme suitable in Asian countries would comprise administration of intermediate strain IBD virus vaccine at 12-14 and 21-24 days. A third vaccination at 7 weeks will provide additional protection. Although IBDV is relatively stable, vaccine should be in strict accordance with the manufacturer's recommendations to ensure viability of virus. Immunity of pullets should be boosted using an inactivated oil-emulsion vaccine during the 18-to-20-week period, depending on the time of transfer. Due to the natural decline in antibody level through the production, a mid-cycle boost of hens at 40 to 45 weeks of age is suggested. Although the cost of oil emulsion vaccine may appear high when considered on a per bird basis, it must be remembered that each hen has the potential to produce more than 120 chicks representing a live mass of 250 Kg depending on slaughter age.

Broiler chicks derived from parent flocks with high uniform titer will generally be protected against conventional (standard) type 1 IBD virus through 25 days of age. It is therefore unnecessary to administer live attenuated vaccine if broiler flocks are reared on all-in all-out basis, with acceptable levels of biosecurity and low probability of field challenge. This ideal situation is seldom attained in Asian countries especially with the emergence of vvIBD.

The level and uniformity of maternal antibody is the most significant determinant of the age at which live attenuated IBD vaccine should be administered to broiler and pullet replacement flocks. Maternal antibody declines at a rate corresponding to a half-life of 3 to 5 days. Generally, broiler flocks are susceptible to IBD at 2 to 3 weeks of age. Maternal antibody will protect against a challenge with104E EID 50 conventional strain 1, IBD virus at a level of 1,000 ELISA units, corresponding to a virus neutralizing titer of 1: 500.With a normal decline in maternal antibody level, chicks can be vaccinated successfully with a mild strain vaccine between 14 to 20 days. The actual time of vaccination is subject to a number of environmental, managemental and vaccine- related factor. Intermediate (moderately attenuated) strain IBD vaccine can stimulate immunity at relatively high level of maternal antibody and can be administered from day old onwards. Highly attenuated, mild IBD vaccine administered during the first 7 days are totally ineffective in broiler flocks with moderate to high levels of maternal antibody.

It is evident that chicks derived from parent flocks with a variable level of immunity or, alternatively, placing chicks from different parent flocks in a single house or unit will result in a mosaic of acquired maternal antibody protection. If conventional IBDV is present in a house or introduced into the flock at an early age, variable manifestation of clinical disease and immunosuppression will result. Under these conditions it may be necessary to administer a mild IBD vaccine by injection at day old. This will result in higher protection compared to administration via the oral or intraocular routes. Maternal antibody levels greater than 3,000 ELISA units will inhibit immunization by transmucosal (water, eye drop, or intranasal) introduction of vaccine route.

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