Infectious Bursal Disease (IBD)
Gumboro disease

Dr./ Wafaa Abd El-ghany
Assistant Professor of poultry dis.,
Introduction

• Infectious bursal disease (IBD) is an acute and highly contagious viral infection of immature chickens characterized by white diarrhea, vent picking, trembling, incoordination and involvement of bursa of Fabricus (BF) with variable degree of immunosuppression.
Economic importance

• Increased mortalities.
• Poor body weight due to poor feed conversion rate.
• Severe immun-suppression, so reduced antibody response to vaccinations, strong post-vaccinal reactions, and increased susceptibility to concurrent or secondary infections.
• Increase condemnation rate at processing (muscle haemorrhages).
• Increase the cost of prevention and control.
The causative agent

• IBD is caused by a birna virus.
• The virus has 2 segments of double strand RNA.
• Two serotypes are present (serotype 1 & 2).
• Only serotype 1 is pathogenic for chickens.
• The virus is resistant to many disinfectants and environmental factors, and remains infectious for months in the poultry house environment, and for weeks in water, feed and droppings.
• Because of the resistant nature of the IBD virus, once a poultry house becomes contaminated, the disease tends to recur in subsequent flocks.
Pathogenesis

- 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 minimal.
Pathogenesis

• After IBD viral infection:
  1. The virus reaches to the lymphocyte cells of the caecum (4 hrs post infection).
  2. Lymphocyte cells in the dudenoum (5 hrs post infection).
  3. Liver (5 hrs post infection).
  4. Then the virus enter the blood stream inducing 1st viraemmia.
  5. The virus distribute into another tissues including bursa of fabricus.
  6. The virus enters the blood stream again producing 2nd massive viraemia.
Susceptibility

• Chickens is considered as the only natural host (species) where the infection occur.
• All breads of chickens could be infected.
• The highly susceptible age to the infection is between 3-6 weeks old.
• Birds younger than 3 weeks old could be infected, but not show clinical signs (sub-clinical infection, and these birds show permanent immunosuppression (economic looses).
• Natural infection of turkeys and ducks have been recorded.
Mode of infection and transmission

• Chickens infected with the IBDV 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 meal worm (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.
• No vertical transmission and no carriers after recovery.
Clinical signs

• Infectious bursal disease follows one of two courses, depending on the age at which chickens are infected.

• **The sub-clinical form** of the disease occurs in chickens less than 3 weeks of age.

• Chickens present no clinical signs of disease, but experience permanent and severe immunosuppression.

• 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 sub-clinical, and this form is the more economically important form of the disease.
Clinical signs

• Broiler integrations commonly have farms described as problem farms.
• Broilers grown on these farms typically have:
  1. Poor body weights and feed conversions.
  2. High mortality.
  3. Excessive reactions to respiratory vaccines.
  4. 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 sub-clinical IBD.
Clinical signs

- 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 with incubation period 2-3 days.
- Clinical signs of disease include:
  1. Vent pecking.
  2. Trembling (tremors) and unsteadiness (incardination).
  3. Ruffled feathers, depression, anorexia and droopy appearance.
  4. Profuse whitish or watery diarrhea and severe prostration.
  5. Dehydration and death.
Morbidity and mortality rate

- In fully susceptible flock, the clinical signs appear suddenly with high morbidity rate usually approaching 100%.

- Mortality rate ranges from 20-30% usually peaks (maximum) on the 3rd day post infection, recedes (gradually decrease) in a period of 5-7 days post infection and no deaths are observed after 8-9 days post infection (spiking death or mortality curve).
Post-mortem lesions

1. Dehydration and darkening discoloration of the pectoral muscles and carcass.
2. Hemorrhage may be present in the thigh and pectoral muscles, because the IBD virus interferes with the normal blood clotting mechanism.
3. The kidneys may appear swollen and pale (nephrosis) and ureter distended with ureats in birds that die or that are in the advanced stages of the disease.
4. Such lesions probably result form severe dehydration, not direct viral damage.
5. Occasionally, haemorrhages could be seen in between the proventriculär glands and at the junction between the proventriculus and gizzard.
6. The liver is enlarged, friable with streaks of haemorrhges.
Post-mortem lesions

5. Initially at the 3rd day post infection, BF is severely enlarged (swollen); appears edematous and hyperemic; and has a gelatinous yellowish transudate covering the serosal surface.

6. In the 6th day post infection, BF is enlarged, the inside follicles contain haemorrhages (haemorrhagic bursitis) (sac filled with blood) and it surrounded with bloody sac.

7. In the 9th day post infection, the BF diminishes in size rapidly (atrophy) and contain specific calcified material.

8. 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.
3주령 닭알육체의 관리:

철근한 갈색 부종이 일어났으며, 절액이 표면에 전채를 덮고 있다.
Diagnosis

• Diagnosis of IBD involves consideration of the flocks' history (3-6 weeks old chickens, Sudden onset, short course, high morbidity, spiking mortality curve and rapid recovery (5-7) days from a characteristic clinical signs if the disease).

• 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.
Diagnosis

• 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.
Isolation and identification of IBDV

- The sample could be taken from BF and spleen of infected birds.
- IBD virus (IBDV) could be inoculated on CAM or in YS or AS of ECE that inducing embryo mortality 3-5 days post inoculation. Gross lesions of the embryo are oedematous distension of the abdominal region, cutaneous congestion and petichial haemorrhages particularly along the feather tract and on toe joints.
- IBDV could be grow on cell cultures of chicken origin as DEF and CEK, also of mammalian origin as BGM-70, Vero and MA-104 inducing cytopathic effect.
- The virus detection in infected tissues by IF or EM and PCR.
- Detection of antibodies using AGPT, VN and ELISA test.
Differential diagnosis

- Coccidiosis.
- Adeno virus infection (inclusion body hepatitis).
- Nephrogenic strain of IB.
- Mycotoxins (Ochratoxin, citrinin and oosporin poisoning).
- Water deprivation.
- Vit. A deficiency.

Differentiation through detection of the most characteristic lesion of IBDV in the BF and or isolation and identification of the causative agent.
Prevention

• **Living embryo or cell culture attenuated vaccine:**
  A) **Mild vaccine:**
  • Not cause immuno-suppression as it not cause bursal damage, but gives weak antibody response.
  B) **Intermediate vaccine:**
  • Causes slightly immuno-suppression (slight bursal damage), and gives good immune response.
  C) **Intermediate plus (hot vaccine):**
  • Gives high level of antibodies.
  • Causes immuno-suppression due to bursal damage.
  • Used in case of improper immunized flocks.
  • Used in epidemic areas and high maternal immunity.
  • **Inactivated oil-adjuvant vaccine:** for breeders and layers only at 16-18 weeks old.
Prevention

• **In endemic areas:**
  ✓ Monitor the titer of maternal antibodies.
  ✓ In case of presence of high level of maternal antibodies use intermediate plus live vaccine as other vaccines can be neutralized by maternal antibodies.
  ✓ The living vaccines used in chickens only at age 10-14 days of age using eye drop or drinking water method and revaccinate at 3-4 weeks of age.
Prevention

• 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 sub-clinical IBD.
Prevention

• An example of a comprehensive breeder vaccination program where sub-clinical 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.
Prevention

• 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.
Prevention

• 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 practiced, as improvement is gradual and often only seen after 3 or 4 flocks.
Prevention

• 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.
Prevention

- 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.
Prevention

• 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.
Prevention

- 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 having 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.
Prevention

• 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.
Control

• Adequate warmthness of the birds.
• Fluid therapy and electrolytes for dehydration.
• Vitamin K to reduce haemorrhages.
• Vitamin A and E for replacing of damaged tissues (Vit. A) and enhancement of the immune system (Vit. E).
• Diuretics as potassium citrate for kidney wash (nephrosis).
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.
Variant strains of IBD

• 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.
THANK YOU