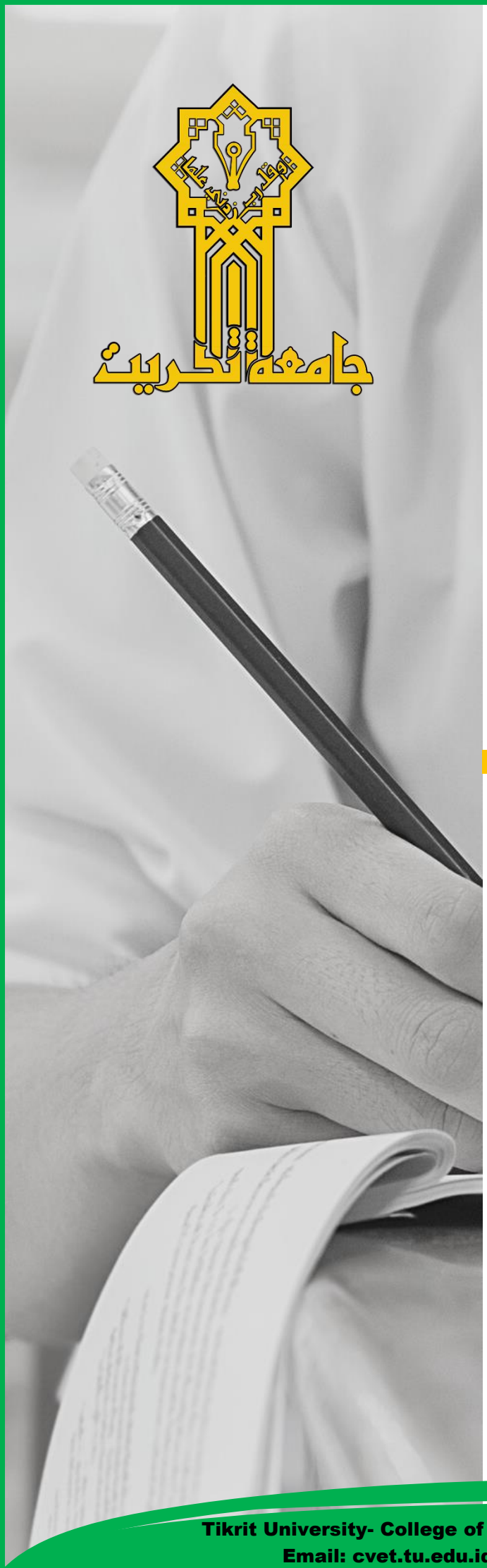

Tikrit University
College of Veterinary Medicine

Infectious Bursal Disease

Subject name: Poultry Diseases

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Infectious Bursal Disease

Infectious bursal disease (IBD) is an acute, highly contagious viral infection of young chickens that has lymphoid tissue as its primary target with a special predilection for the bursa of Fabricius (cloacal bursa). was referred to as "avian nephrosis" because of the extreme kidney damage found in birds that succumbed to infection. Since the first outbreaks occurred in the area of Gumboro, Delaware, "Gumboro disease" was a synonym for this disease and is still frequently used.

Economic Significance

1- Mortality.
2-Decreased egg production.

Etiology:-

Infectious bursal disease virus is a member of the ***Birnaviridae*** family: the genera include *Avibirnavirus* whose type species is IBDV which infects birds.

Transmission:-

1-Environment of a poultry house.

2-No evidence suggests that IBDV is transmitted through the egg.

3-Mealworm adults and larvae that were fed the virus earlier.

4-Mosquitoes.

5-Dog fed chickens that had died of acute IBD.

Morbidity, and Mortality

1-Morbidity rate usually approaching 100%.

2-Mortality may be nil but can be as high as 20–30%, exceptionally higher with vvIBDV, usually beginning on day 3 postinfection and peaking and receding in a period of 5–7 days.

Clinical Signs:-

1-Tendency for some birds to pick at their own vents.

2-Soiled vent feathers, whitish or watery diarrhea, anorexia, depression, ruffled feathers, trembling, severe prostration, and finally, death.

3-Affected birds became dehydrated.

Gross lesions:-

1-Darkened discoloration of pectoral muscles.

2-Hemorrhages are present in the thigh and pectoral muscles.

3-There is increased mucus in the intestine.

4-renal changes may be prominent in birds that die or are in advanced stages of the disease.

5- Bursa enlarge at day 2 and start to atrophy at day 8 of infection.

6-By day 2 or 3 post-infection, the bursa has a gelatinous yellowish transudate covering the serosal surface.

7-Longitudinal striations on the surface become prominent.

8-The infected bursa often shows necrotic foci and at times petechial or ecchymotic hemorrhages on the mucosal surface.

9-The spleen may be slightly enlarged and very often has small gray foci uniformly dispersed on the surface.

10-Occasionally, hemorrhages observed in the mucosa at the juncture of the proventriculus and gizzard.

11-decrease in thymic weight index and more severe lesions in the cecal tonsils, thymus, spleen, and bone marrow, but bursal lesions were similar.

Microscopic changes :-

1-Degeneration and necrosis of lymphocytes in the medullary area of bursal follicles.

2-Hemorrhages.

3-Severe edema, hyperemia, and marked accumulation of heterophils.

4-Cystic cavities developed in medullary areas of follicles.

5- Necrosis and phagocytosis of heterophils and plasma cells occurred.

6-Proliferation of the bursal epithelial layer produced a glandular structure of columnar. epithelial cells containing globules of mucin.

7-Depletion of B lymphocytes was maximum during the first week, and combined with a transient massive influx of T cells and macrophages until three days post-infection.

8-The spleen had hyperplasia of reticuloendothelial cells around the adenoid sheath arteries in early stages of infection.

9-Lymphoid necrosis (day 3) of germinal follicles and the periarteriolar lymphoid sheath.

10-Histologic lesions of the kidney are non-specific because of severe dehydration of affected chickens; include large casts of homogeneous material infiltrated with heterophils.

Diagnosis:-

1-Clinical signs.

2-The rapid onset, high morbidity, spiking mortality curve, and rapid recovery (5–7 days).

3-Necropsy.

4-Changes in size and color of the bursa during the course of infection (i.e., enlargement due to inflammatory changes followed by atrophy).

5-Histopathology.

6-Virus isolation.

Differential Diagnosis:-

1-Coccidiosis.

2-Nephrosis-causing conditions.

3-Water deprivation.

4-Nephropathogenic strains of infectious bronchitis virus.

5-Hemorrhagic syndrome.

6-Marek’s disease.

Treatment:

No therapeutic or supportive treatment has been found to change the course of IBDV infection.

Prevention and Control

1-The virus resistant to many physical and chemical agents.

2-The sanitary precautions that are applied to prevent the spread of most poultry infections must be rigorously used in the case of IBD.

3-The possible involvement of other vectors (e.g., the lesser mealworm, mosquitos, dogs and rats) they could certainly pose extra problems for the control of this infection.

Immunization:

1-Maternal antibody will normally protect chicks for 1–3 weeks.

2-Boosting the immunity in breeder flocks with oil-adjuvanted vaccines, passive immunity may be extended to 4 or 5 weeks.

3-According to virulence, vaccines are classed as mild, mild intermediate, intermediate, intermediate plus, or "hot." Vaccines that contain Delaware variants, either in combination with "classic" strains or alone.

4-Highly virulent (hot), intermediate, and avirulent strains break through maternal VN antibody titers of 1:500, 1:250, and less than 1:100, respectively.

5-Killed-virus vaccines in oil adjuvant are used to boost and prolong immunity in breeder flocks, Killed-virus vaccines are usually not practical, Oil adjuvant vaccines are mosteffective in chickens that have been "primed" with live virus either in the form of vaccine orfield exposure to the virus.

6-Recent concept for the vaccination of chickens for IBD and other agents is in ovo vaccination at 18 days of incubation.