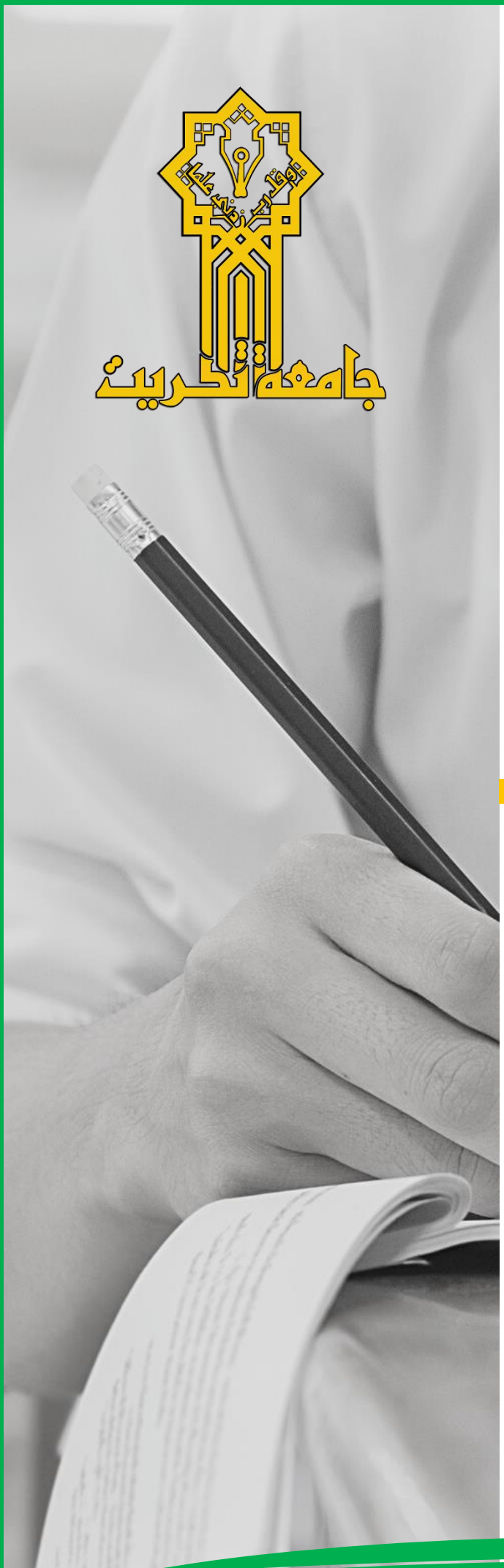

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Chicken Infectious Anemia

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Chicken Infectious Anemia

Summary

Chicken infectious anemia (CIA) is caused by CIA virus, which is classified as the only recognized species of the Gyrovirus genus of the Anelloviridae. Chicken infectious anemia virus (CIAV) has a single-stranded, circular DNA genome of approximately 2.3kb coding for three viral proteins (VP). VP1 codes for the structural capsid protein, VP2 has multiple functions, and VP3 causes apoptosis. CIAV virions consist of non-enveloped, icosahedral particles with an average diameter of 25–26.5nm.

Infection of newly-hatched chicks lacking maternal antibodies can result in severe thymus atrophy, replacement of hematopoietic cells by adipose tissue, anemia, and gangrenous dermatitis. Infection after maternal antibodies disappear causes a subclinical infection with immunosuppression.

Susceptibility to Chemical and Physical Agents

The CIAV is extremely resistant to most treatments. Treatment of virus in liver suspensions with 50% phenol for five minutes inactivates CIAV but treatment with 5% phenol for two hours at 37°C is ineffective. The virus is resistant to treatment with 50% ethyl ether for 18 hours and chloroform for 15 minutes. Treatment of liver suspensions with 0.1N NaOH for two hours at 37°C or 24 hours at 15 °C inactivates CIAV incompletely. Treatment with 1% glutaraldehyde for 10 minutes at room temperature, 0.4% β-propiolactone for 24 hours at 4°C, or 5% formaldehyde for 24 hours at room temperature inactivates the virus completely. Commercial disinfectants based on invert soap, amphoteric soap, or orthodichlorobenzene are not effective against CIAV.

Public Health Significance: There are no public health significance.

Replication

Virions probably enter the cell by conventional adsorption and penetration. Low levels of the 2.1kb polycistronic viral RNA transcript can be demonstrated at 8 hours postinfection Viral DNA replication occurs via a double-stranded replicative form (RF), probably by the rolling-

circle mechanism The double- stranded RF may lead to the presence of latent episomal DNA and be responsible for the presence of viral DNA in gonadal tissues VP3 can be detected at 6 hours postinfection (PI) in a few cells. VP2 is present at 12 hours postinfection (PI), while the capsid protein (VP1) was not detectable until 30 hours PI

Transmission

CIAV spreads both horizontally and vertically

Incubation Period:

Clinical signs generally develop after 10 to 14 days, and mortality begins at 12 to 14 days after inoculation

Clinical Signs:

1-The only specific sign of CIAV infection is anemia, with a peak at 14 to 16 days PI.

2-. Anemia is characterized by hematocrit values ranging from 6–27%. Affected birds are depressed and become pale.

3-Weight gain is depressed between 10 and 20 days

4-If mortality occurs, it generally does not exceed 30%.

5-Surviving chicks completely recover from depression and anemia by 20–28 days PI

6-mortality may be associated with secondary bacterial or viral infections. Secondary infections, causing more severe clinical signs

7-Hematocrit values begin to drop below 27% at eight to 10 days PI

Gross (P.M.) lesions:

1-Thymic atrophy, sometimes resulting in an almost complete absence of thymic lobes

2-The thymic remnants may have a dark reddish color.

3-Bone marrow atrophy, becomes fatty and yellowish or pink. In some instances, its color appears dark red

4-Bursal atrophy, In many cases, the outer bursal wall appears translucent

5- Hemorrhages in the proventricular mucosa and subcutaneous and muscular hemorrhages

6-swollen and mottled livers.

7-Hemorrhagic-Aplastic Anemia, associated with inclusion body hepatitis (IBH) (301) and with the IBH/hydropericardium syndrome or infectious bursal disease (IBD) hemorrhages may be present even more frequently in the mucosa of the distal part of the proventriculus

8- Intracutaneous or subcutaneous hemorrhages of the wings are often complicated by severe edema and subsequent dermatitis, which may become gangrenous due to bacterial infection

10- Subcutaneous hemorrhage of shanks and feet may result in formation of ulcers

11- Endothelial lesions and impaired liver functions, partly caused by viral infection and enhanced by secondary bacterial infection, are likely to be more important in the pathogenesis of hemorrhagic diathesis

Histologic changes:

1- panmyelophthisis and generalized lymphoid atrophy. In the bone marrow, atrophy and aplasia involve all compartments and hematopoietic lineages

2- Necrosis of residual small cell foci may occasionally be seen

3- Hematopoietic cells are replaced by adipose tissue or proliferating stroma cells.

4- Severe lymphoid depletion is seen in the thymus, starting with the cortical lymphocytes, but the non-lymphoid leukocytes and stroma cells are not affected.

5- The thymus cortex and medulla become equally atrophic, with hydropic degeneration of residual cells and occasional necrotic foci

6- In chicks that recover, repopulation of the thymus with lymphocytes becomes distinct at 20–24 days, and the morphology returns to normal by 32–36 days PI

7- Lesions in the cloacal bursa may be present. These lesions consist of mild to severe atrophy of the lymphoid follicles with occasional small necrotic foci, infolded epi thelium, hydropic epithelial degeneration, and proliferation of reticular cells.

8- In the spleen, depletion of T cells with hyperplasia of reticular cells is seen in the lymphoid follicles as well as in the Schweigger-Seidl sheaths. Necrotic foci in follicles or sheaths have been observed rarely.

9- In the liver, kidneys, lungs, proventriculus, duodenum, and cecal tonsils, lymphoid foci are depleted of cells, making them smaller and less dense than those in unaffected birds. Liver cells are sometimes swollen, and hepatic sinusoids may be dilated.

10- Small eosinophilic nuclear inclusions have been detected in altered, enlarged cells of affected tissues, predominantly in the thymus and bone marrow, where they are most frequent at five to seven days after experimental infection

Pathogenesis of the Infectious Process

When chicks were inoculated at 7 days but not at 1 day of age; inoculation at 14 days of age failed to induce lesions . thymus and bone marrow lesions when 10-week-old broiler breeders were infected

Immunity

Active

Antibody responses are the major arm of protective immunity to CIAV, but neutralizing antibodies cannot be detected until three weeks PI of susceptible one-day- old chicks. Titers are low (1:80) and show little increase (1:320) until four weeks. Chickens inoculated intramuscularly at two to six weeks of age have a faster response with neutralizing antibody detectable as early as four to seven days and with maximum titers (1:1280–1:5120) at 12 to 14 days PI.

High titers of neutralizing antibody persist in all birds of a flock for at least 52 weeks. The prevalence of antibodies detected by indirect immunofluorescence assays, however, may decrease with increasing age and is frequently less than 100% in a flock. Antibodies detected by a commercial ELISA kit will remain present until 60 to 80 weeks of age.

Passive

Maternal antibodies provide complete protection of young chicks against CIAV-induced anemia. This protection can be abrogated if chicks are immunosuppressed by other factors, including viral infections, especially infections such as IBDV that affect humoral immune responses. Maternally derived immunity, including protection against experimental challenge, persists for about three weeks.

Diagnosis:

1- Clinical signs and gross and histologic lesions.

2-virus isolation

3- RT-PCR.

4-ELISA

Differential Diagnosis

Anemia, IBD, Dermatitis

Treatment

1-Antibiotics: 2-Anti-inflammatory:3-Supplements:

Vaccination

Vaccination strategies are based on the prevention of vertical and horizontal transmission of virus to very young chicks by immunization of breeder flocks and have been successful in reducing the incidence of anemia in young chicks.

Commercial live vaccines for pullets are available in several countries. Vaccination should be performed at about 9 to 15 weeks of age, but never later than three to four weeks before the first collection of hatching eggs to avoid the hazard of vaccine virus spread through the egg. Vaccines can be applied in the drinking water or by injection. However, approximately one-third of the complexes surveyed did routinely vaccinate all breeder pullets between 10 and 12 weeks of age. Males are not usually vaccinated