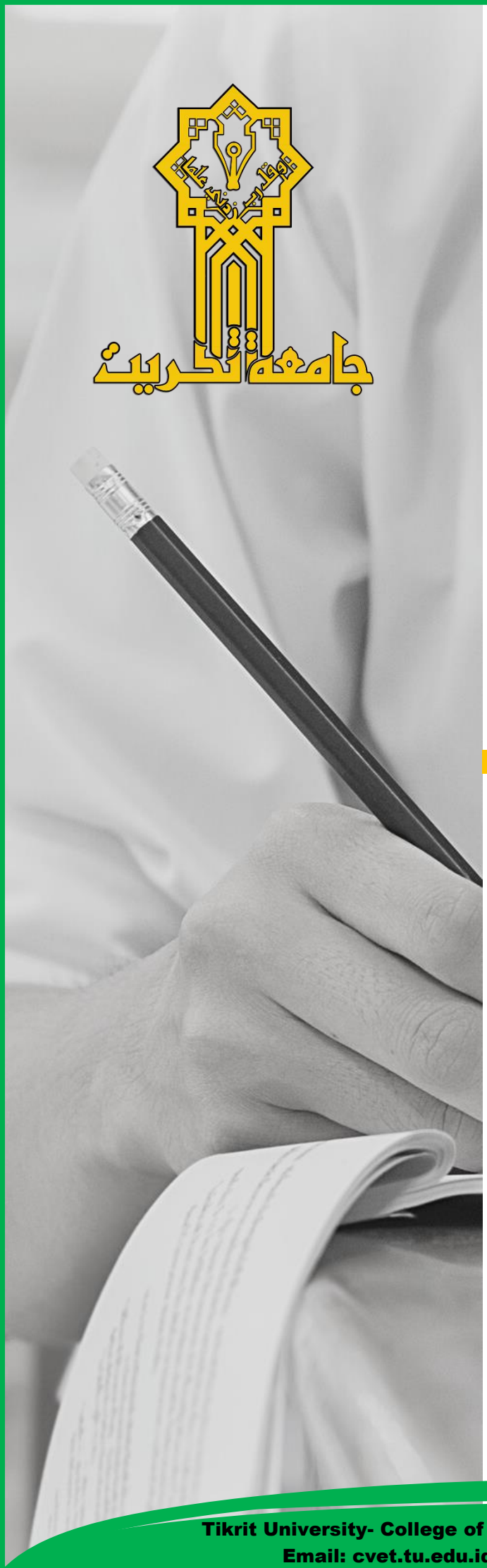

Tikrit University
College of Veterinary Medicine

Adenovirus Infections Hemorrhagic Enteritis and Related Infection

Subject name: Poultry Diseases

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Lecturer name: Ismael I. Hasan

Academic

Email:ismailhasan@tu.edu.iq

Lecturers link

Adenovirus Infections
Hemorrhagic Enteritis and Related Infection

Summary

Hemorrhagic enteritis (HE) of turkeys and related infections in other gallinaceous fowl are caused by a group of related Siadenoviruses. Hemorrhagic enteritis (HE) is an acute viral disease of turkeys four weeks of age and older, characterized by depression, bloody droppings, and death. Clinical disease usually persists in affected flocks for 7–10 days. Due to the immunosuppressive nature of the virus, secondary bacterial infections may extend losses for an additional 2–3 weeks.

Public Health Significance:

The viruses responsible for HE are not known to cause illness or seroconversion in humans

Replication

Macrophages and B lymphocytes are believed to be the primary target cells. The spleen appears to be the major site of viral replication; however, infected cells were also detected in a variety of other tissues including intestine, cloacal bursa of Fabricius, cecal tonsils, thymus, liver, kidney, peripheral blood leukocytes, and lung. The replication strategy at the cellular level is presumed to be similar to that of other adenoviruses. receptor-mediated endocytosis, most likely via cell surface integrins. DNA transcription utilizing host RNA polymerase II takes place in the nucleus. Genome replication also occurs in the nucleus and involves virus-encoded DNA-dependent DNA polymerase and the formation of a pan-handle intermediate with base pairing occurring at inverted terminal repeats. Virions are assembled in the nucleus and released upon cell disintegration.

Susceptibility to Chemical and Physical Agents

TAdV-3 remains stable under a variety of harsh conditions: heating at 65 °C (149°F) for 1 hour, liquid at 37 °C (99°F) for 4 weeks or 4 °C (40°F) for 6 months, freezing at −20°C (−4°F) for 4 years, and at low pH. Infectivity can be destroyed by heating at 70°C (158°F) for 1 hour and drying at 37 °C (99°F) or 25 °C (77°F) for 1 week. Chlorine- and iodine-based disinfectants are known to be effective against TAdV-3 and quaternary ammonium compounds should also be useful due to their efficacy against non-enveloped viruses

Pathogenesis

It is not uncommon for TAdV-3 isolates to be referred to as avirulent or virulent based on the severity of lesions they produce, that is, splenomegaly in the former and splenomegaly, gastrointestinal hemorrhage, and death in the latter. ORF1, E3, and fib genes were suggested to be key factors affecting the virulence of the TAdV-3 strains. In the field, mortality due to virulent HE is reported to vary from more than 60% to less than 0.1%. Mortality rates in pheasants naturally infected with MSDV are reported to range between 5% and 20%.

Transmission:

Vertical & Horizontal

Incubation Period:

In turkeys, clinical signs and mortality begin 5–6 days after oral or cloacal infection and 3–4 days after intravenous inoculation with infectious splenic homogenate.

Clinical Signs:

1- HE is characterized by a rapid progression of clinical signs over a 24-hour period.

2- These include depression, bloody droppings, and death.\

3- Fecal material containing dark blood is often present around the vent. It also may be expressed from the vent if pressure is applied to the abdomen.

4- Signs of disease tend to subside 6–10 days after the appearance of bloody droppings.

5- morbidity approaches 100%.

6- Turkeys exhibiting clinical signs usually die within 24 hours or recover completely.

7- Mortality ranges from less than 1% to greater than 60% and averages 10–15%

8- If birds survive the acute phase of TAdV-3 infection, immunosuppression still makes them vulnerable to secondary infections

9- Leading to a second peak of mortality.

Gross (P.M.) lesions:

1- dead turkeys routinely appear pale due to blood loss, but are typically in good flesh and have feed in their crops.

2- The small intestine is commonly distended, grossly discolored, and filled with bloody contents.

3-The intestinal mucosa is congested and, in some cases, covered with a yellow fibrinonecrotic membrane.

4- Lesions are usually more pronounced in the duodenum, but can extend distally.

5- Spleens are characteristically enlarged, friable, and mottled in appearance; however, those ofdead birds tend to be smaller due to contraction of the spleen in response to blood loss.
6- Lungs may be congested, but other organs are generally pale.
7- Hepatomegaly and petechiae in various tissues also have been reported.8-Lesion formation with virulent strains appears to be dose-dependent

Histologic changes (Microscopic Lesions):

1- Splenic lesions present at death include hyperplasia of white pulp and lymphoid necrosis.
2- Basophilic Cowdry type B intranuclear inclusions (INI) can be found within mononuclearcells, that is, macrophages and lymphocytes.
3- Proliferation of white pulp surrounding splenic ellipsoids is evident as early as 3DPI.4-large, irregular, confluent islands of white pulp which are grossly visible 4–5DPI.
5- Hematoxylin and eosin (H&E) staining reveals numerous INI in these splenic zones between 3and 5DPI but also in other organs.
6- By 4–5DPI, the white pulp begins to undergo necrosis and by 6–7DPI it has completelyinvoluted with only occasional plasma cells appearing in the red pulp.
7- In addition to splenic changes, lymphoid depletion is also noted 3–9DPI in both the corticaland medullary areas of the thymus and bursa of Fabricius.
8- Typical intestinal lesions include mucosal congestion, hemorrhage in the villus tips, andepithelial necrosis.
9- Hemorrhage and necrosis are thought to be the result of endothelial disruption rather than destruction because blood vessels in the lamina propria appear intact and diapedesis of red cells is observed.
10- Increased numbers of mononuclear cells with INI are observed in the lamina propria in addition to mast cells, plasma cells, and heterophils.
11- These changes are most pronounced in the duodenum just posterior to the pancreatic ducts, but similar, less severe lesions also may occur in the proventriculus, gizzard, distal small intestine, ceca, cecal tonsils, and bursa of Fabricius.

Diagnosis:

1-Clinical signs and gross and histologic lesions.

2-virus isolation

3- PCR.

4- Serology

Differential Diagnosis

1- Bacteremia (spleen).

2- Acute viral (highly pathogenic avian influenza, HPAI; Newcastle disease,

ND).3-Parasitic (coccidiosis).

4- Toxic (heavy metals, sulfa drugs).

5- Postmortem autolysis of the intestinal tract.

6- Respiratory diseases including AI, ND, Syngamus

trachea.7-Carbon monoxide.

8-Neoplastic diseases such as Marek's disease, lymphoid leukosis, or reticuloendotheliosis.

Treatment

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

Prevention and Control (Intervention Strategies):

1- Management Procedures

2- Vaccination:

- Two forms of vaccine are currently in use for turkeys. The first is a crude homogenate prepared from spleens obtained from 6-week-old SPF turkeys inoculated with a splenic homogenate containing the Domermuth strain (Virginia Avirulent 1). The second is a commercially available cell culture product which contains an adapted/attenuated virus originally derived from the Domermuth strain.

-In some countries only inactivated (i) TAdV-3- vaccines are commercially available.

-The application of an iTAdV-3-vaccine once or twice may not sufficiently protect against the infection with circulating strains, possibly due to a poor immune response