



Tikrit University College of Veterinary Medicine

# Avian Adenovirus

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# **8-Avian Adenovirus Infections (AADV)**

Family:Adenoviridae

Genus:Aviadenovirus Group 1 HHS (Serotype 4).

→ IBH (Serotype 11).

Genus:Siadenovirus Group  $2 \longrightarrow$  Hemorrhagic enteritis(turkey).

Genus: Atadenovirus Group 3  $\longrightarrow$  EDS76(Egg drop syndrome virus).

# **1-Hydropericardium Hepatitis Syndrome(H.H.S)** (Angara disease)

\* **Definition:**:it is acute infectious disease of chickens characterized by **high morbidity and mortality rate, excess pericardial fluid**, multifocal hepatic necrosis and present **basophilic intranuclear inclusion bodies** in hepatocytes.

\***Etiology:** Pathogenic Group I Adenovirus Serotype 4.

\*The economic important difficulties to determine because the condition occur with other disease such as Velogenic ND, Mycoplasmosis, MD, Salmonellosis, IBD, CAV.

## \*Incubation period:- is short (24-48 hr.).

#### \*<u>Synonyms:</u>

1-Angara disease.2-Inclusion body hepatitis-hydropericardium.

# \*<u>Host Susceptibility:</u>

**Immature** chickens are the natural host and most commonly in **3-5 weeks** of age of broiler.

### \*Transmission:

- 1-Vertical.
- 2-Horizontal transmission by carrier.
- 3-Commercial transmission.

Viral replication in the **intestine tract** and the contamination occur by **feces** in clothes, foot wear, equipment, vehicles.

4-**Contaminated vaccine** prepare in embryo derived from infected flock may also source of infections.

5-There is evidence that needle or plate use in vaccination and bleeding of viremic birds if not sterile can transmit infection.

#### \*Morbidity and Mortality rate:-

Duration of infection usually ranged from **9-14 days** with morbidity rate (**10-30%**) and **mortality 30-70%** depending on the preparing and titer of virus and route of administration, the **daily mortality 3-5%**.

#### \*<u>Clinical Signs</u>:

Flocks with HHS show no specific clinical signs.

- 1-sudden death.
- 2-Ruffled feather.

3-yellow mucoid drooping are characteristic.

4- Severe **anemia** (because bone marrow affected) in affected birds.

### \*<u>Gross lesion</u>:

1-The most characteristic lesion is presence of up to 10ml of clear transudate in the pericardial sac.

2-Liver and kidney are usually enlarge, pale, friable, hepatic necrosis.

3-**Petechial hemorrhage** may be present on the pericardium and under the capsule of liver.

4-Congestion and pulmonary edema.

## \*<u>Microscopic lesions</u>:-

1-The lesion in heart consist of **myocardial edema**, degeneration and necrosis with mild mononuclear cells infiltration.

2-Multifocal **coagulative necrosis** with mononuclear cells infiltration and **basophilic intranuclear inclusion bodies in hepatocyte** in liver.

3-May be extensive areas of necrosis in renal epithelium.

## \*<u>Diagnosis</u>:

1- Case history.

2-clinical signs.

3-Gross lesion.

4-laboratory test.

5-Can be done by histological demonstration of **basophilic intranuclear bodies in hepatocytes**, it consider highly suggestive of HHS.

6-To confirm the diagnosis by isolated by infected embryonic chick liver in egg of chicken.

### \*<u>Treatment</u>:-

1-There is no specific treatment of HHS.

2-Using Iodophore(0.07-1)% of 2.5% in drinking water of affected flock to reduce mortality and severity of disease.

## \*Prevention andControl:-

1-Management procedure.

#### 2-Vaccination (killed vaccine).

Referens:

1-Saif, Y. M. (2009). Diseases of poultry. Twelfth edition. Iowa. Blackwell.2009. 251-290.

# 9-Inclusion Body Hepatitis (IBH) (Aplastic anemia)

\***Definition:** acute infection disease of young and mature chicken characterized by sudden onset, short course, pathogenic liver change and anemia.

\*Etiology: pathogenic adenovirus serotype 11 Group I.

\***Transmission**: like HHS.

#### \*Clinical Signs:

- 1-Depression (Crouching position).
- 2-Ruffled feathers.
- 3-Pale of comb and wattles.

#### \*Gross lesion:

- 1-Liver swelling, yellow, mottled, with petechial hemorrhage.
- 2-Kidney and **bone marrow** are pale and mottled spleen.

## \*<u>Microscopic lesions</u>:-

The important lesion **Basophilic intranuclear inclusion body in hepatocyte**.

# **10-Egg drop syndrome (EDS76).**

\***Definition:** it is infection disease of laying hens, characterized by producing thin shelled or shell-less eggs.

## \* **Etiology:** - Pathogenic adenovirus Group 3.

\*First isolate 1976, and introduced into chickens through contaminated vaccine.

\* Incidence and prevalence: -wide spread in many countries.

\*Transmission of the disease: same to HHS.

# \*<u>Clinical Signs</u> (Symptoms<u>)</u>:-

\*The first was loss of color in pigmented eggs, followed by production of thinshelled, soft-shelled or shell-less eggs.

\*The thin-shelled eggs were often rough with **sandpaper-like texture** or granular roughening of the shell at one end of the egg.

\*The fall in egg production was **very rapid** or extended over **several weeks**. \*EDS outbreak usually lasted **4-10 weeks**, and egg production was reduced by **upto(40%)**.

\*Watery albumin has described.

## \*Gross lesion (P.M):-

\*In naturally occurring outbreaks of EDS, inactive ovaries and atrophied

oviducts, uterine edema, flaccid ovules, and eggs in various stages of formations in the abdominal cavity.

\*Mild splenomegaly.

## \*<u>Microscopic lesions</u>:-

\*The major pathologic changes occur into **oviduct and shell glands.** \*Virus replication in the nuclei of epithelial cells, and **intranuclear inclusion bodies** from **7 days post infection**.

\*Many affected cells were sloughed into the lumen and there was rapid and severe inflammatory response with macrophage, plasma cell, lymphocytes, variable number of heterophil.

\*Inclusion bodies were **not seen after the 3rd day** of abdominal egg production, but viral antigens persist for up to 1 week.

## \* Diagnosis:-

1-case history.
2-Clinical signs.
3-P.M lesion.
4-Isolation and Identification.
5-HI, ELISA.

### \*Prevention andControl:-

1-Management procedures.

2-Vaccination.**Oil-adjuvant inactivated vaccine** is widely use and gives good protection against EDS, the birds are vaccinated between **14-16 weeks of age**.

### \*Treatment:-

1-No Successful treatment.

2-Vitamins, increase calcium or protein in the ration.