

# **DUCK VIRUS ENTERITIS**

## **(DVE)**

### *(Duck Plague)*

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# **DISEASES OF WATERFOWL**

1. DUCK VIRAL HEPATITIS.
2. DUCK VIRUS ENTERITIS.
3. HEMORRHAGIC NEPHRITIS ENTERITIS OF GEESE.
4. GOOSE PARVOVIRUS INFECTION.
5. GOOSE HERPESVIRUS.
6. AVIAN PNEUMOVIRUS.
7. CIRCOVIRUS-LIKE INFECTION OF GEESE.
8. WEST NILE VIRUS.
9. MUSCOVY DUCK REOVIRUS.
10. AVIAN INFLUENZA VIRUS.
11. AVIAN PARAMYXOVIRUSES.
12. ADENOVIRUSES

# Definition

- It is an acute, contagious herpes virus infection of ducks, geese, and swans, characterized by vascular damage, tissue hemorrhages, digestive mucosal eruptions, lesions of lymphoid organs, and degenerative changes in parenchymatous organs.
- Economic losses in domestic and wild waterfowl due to mortality, condemnations, and decreased egg production.

# Cause

- The causative agent of DVE is DNA Alpha herpes virus, non hemagglutinating and nonhemadsorbing, sensitive to ether and chloroform.
- Virus infectivity was destroyed after heating for 10 minutes at 56°C.
- DEV strains are differed in virulence, but all appear to be immunologically identical and antigenically related. The virus is immunologically distinct from other avian viruses.
- The virus replicates in nuclei of the host cells inducing intranuclear inclusion bodies,

# Cause

- DVE can be propagated in duck embryo fibroblast tissue culture incubated at 39.5-41.5°C, or on the chorioallantoic membrane (CAM) of 9-14-day old embryonated duck eggs .
- Virus can also be grown in duck embryo liver or kidney cells.
- Duck enteritis virus can be adapted to grow in embryonating chicken eggs.
- A cytopathic effect occurs in cell cultures.

# Susceptibility

- Concentrated large numbers of susceptible domestic ducks and geese enhance the probability of disease detection.
- Higher incidence of DVE was noted in the spring.
- Natural infections have occurred in ducks ranging from 7 days of age to mature breeder.
- Naturally occurring outbreaks have occurred in a variety of domestic ducks, including white Pekin, Muscovy ducks, domestic geese and swans.
- No differences in mortality rates were found in mallard and white Pekin ducks.
- Recovered birds are immune to re-infection by DEV.

# Transmission

- Duck virus enteritis transmitted by direct contact between infected and susceptible birds or indirectly by contact with a contaminated environment.
- Water appears to be the natural means of transmission.
- DVE is self-limiting.
- Potential transmission by blood sucking arthropods may be possible during viremia.
- A carrier state has been suspected in wild ducks and recovered birds shed the virus periodically.
- DEV latency and re-activation have been blamed for precipitating outbreaks in domestic and migrating waterfowl populations.

# Signs

- In domestic ducks, the incubation period ranges from 3-7 days and death usually follows within 1-5 days.

## **Breeder ducks:**

- Signs are sudden, high; persistent flock mortality is the first signs.
- Mature ducks die in good flesh.
- Prolapsed penis may be evident in dead mature males.
- In laying flocks, a marked drop in egg production may be noted during the period of highest mortality.
- Photophobia associated with half-closed and pasted eyelids.

# Signs

## **Breeder ducks:**

- Inappetence, extreme thirst, droopiness, ataxia, ruffled feathers.
- Nasal discharge.
- Soiled vents with watery diarrhea.
- Affected ducks are unable to stand; maintain drooping posture (outstretched wings and down extended head), weakness and depression.
- Sick ducks forced to move may have tremors of head, neck, and body.
- Adult breeder ducks tend to experience greater mortality than young ducks.

# Signs

- **Young ducklings:**
- Birds in 2-7 weeks of age show:
- Dehydration.
- Loss of weight.
- Blue beaks.
- Conjunctivitis and lacrimation.
- Nasal exudates.
- Blood-stained vent.
- Total mortality in domestic ducks may range from 5-100%.
- All sick birds usually die.

# Lesions

- Lesions of DVE are those of vascular damage, eruptions at specific locations on gastrointestinal tract mucosal surface, lesions of lymphoid organs, and degenerative sequelae in parenchymatous organs. These collective lesions, when present, are diagnostic of DVE.

## 1. Vascular damage:

- Petechial, echymotic, or larger extravasations of blood may be found on or in the myocardium and other visceral organs and their mesentery and serous membranes.
- On the epicardium, especially within coronary grooves, closely packed petechiae give the surface a red “paintbrush” appearance; this lesion is more observed in mature breeder ducks than in young market ducklings.
- Endocardial mural and valvular hemorrhages may also be observed.

# Lesions

## **1. Vascular damage:**

- Surfaces of liver, pancreas, intestine, lungs, and kidney may be covered with petechiae.
- In mature laying females, hemorrhages may be observed in deformed, discolored ovarian follicles, and massive hemorrhage from the ovary may fill the abdominal cavity.
- Lumina of intestines and gizzard are often filled with blood.
- The esophageal proventricular sphincter appears as a hemorrhagic ring.
- Specific digestive mucosal lesions are found in the oral cavity, esophagus, ceca, rectum, and cloaca.

# Lesions

## **2. Eruption lesions:**

- Each of previous lesions undergoes progressive alterations during the course of the disease.
- Initially, macular surface hemorrhages appear (1-10 mm in length), later covered by elevated yellow white crusty plaques, and finally the lesion becomes organized into a green superficial scab.
- In the esophagus, macules occur parallel to longitudinal folds, numerous small lesions may merge to form larger ones, forming a patchy diphtheritic membrane.
- In young ducklings, the esophagus sloughing of the entire mucosa is more common, and the lumen becomes lined with a thick yellow-white membrane.

# Lesions

## 2. Eruption lesions:

- Oral erosions can be found at openings of sublingual salivary gland ducts in chronically infected waterfowl.
- Meckel's diverticulum may be hemorrhagic and contain a fibrinous core.
- In ceca, macular lesions are singular, separated, and well defined between mucosal folds, while the serosal surface presents a barred, congested appearance.
- Rectal lesions are usually few in number with greatest concentration at the posterior portion of the rectum, close to the cloaca.
- Cloacal macular lesions are densely packed; initially the mucosa appears reddened and later, individual plaque-like elevations become green and form a continuous scale-like band lining the lumen of the organ.

# Lesions

## 3. Lesions in lymphoid organs:

- All lymphoid organs are affected.
- The spleen is normal or smaller in size, dark, and mottled.
- Thymus has multiple petechiae and yellow focal areas on the surface and cut section and is surrounded by clear yellow fluid that infiltrates and discolors subcutaneous tissues of the adjacent cervical region from the thoracic inlet to the upper third of the neck.
- The bursa is intensely reddened during early infection and become surrounded by clear yellow fluid that discolors adjacent tissue of the pelvic cavity. The lumen of the bursa has pinpoint yellow areas in an intensely reddened surface. Later, the bursal wall becomes thin and dark, and its lumen is filled with white coagulated exudates.

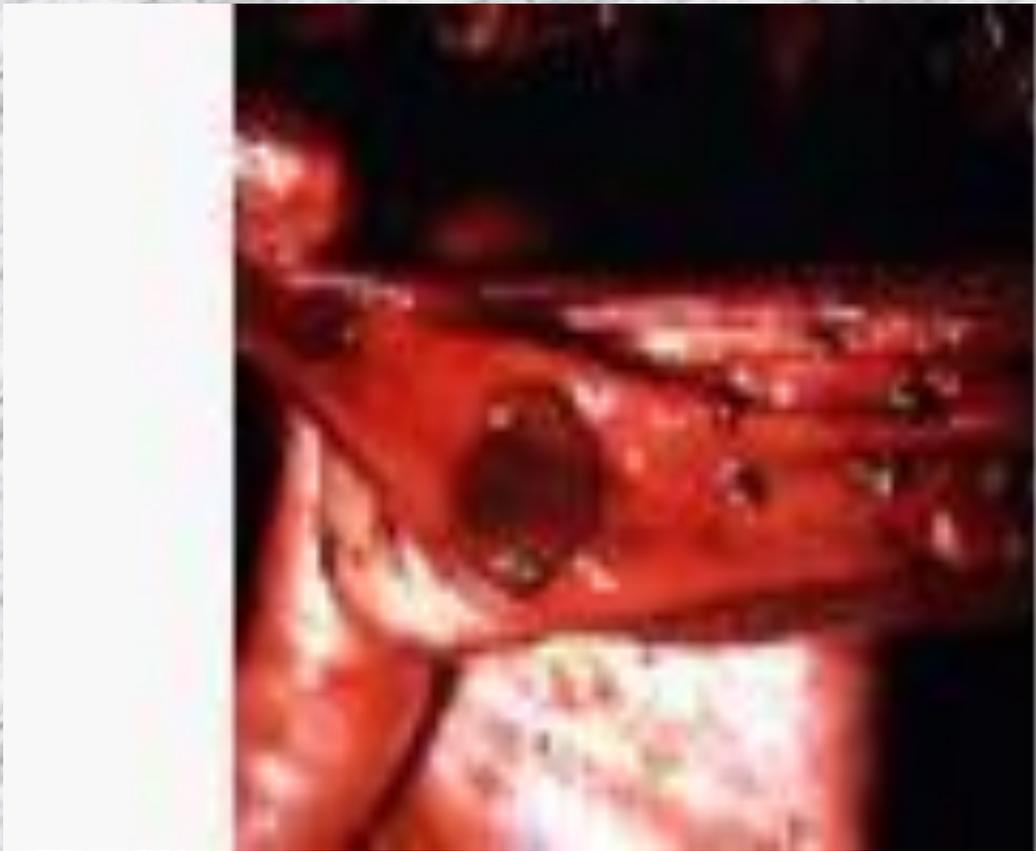
# Lesions

## **3. Lesions in lymphoid organs:**

- Intestinal annular bands appear as reddened rings visible from external and internal surfaces. Later, the entire band becomes dark brown and tends to separate at its margins from the mucosal surface.
- The multifocal necrosis of gut-associated lymphoid tissue causes ulceration covered by fibrinous pseudomembranes.
- AT the early infection, the entire liver surface is a pale copper color with irregularly distributed pinpoint hemorrhages and white foci, giving it a heterogeneous, speckled appearance, while in late stages liver appear dark bronze or bile-stained without hemorrhages; more distinct large white foci on darker background.
- In ducklings, tissue hemorrhages are less pronounced and lymphoid lesions are more prominent. In mature domestic ducks with naturally regressed bursa and thymus, tissue hemorrhages and reproductive tract lesions predominate.

# Lesions

- In geese, intestinal lymphoid disks are analogous to annular bands in ducks.
- Lesions of the intestinal lymphoid disks resembled “button- like ulcers” can be observed in an outbreak.
- In swans, diphtheritic esophagitis is a consistent lesion.
- Recently, natural infection with a low pathogenic DEV in commercial 2-6 week old white pekin ducklings caused atypical gross lesions, including diphtheritic membranes under the tongue and in nasal and infraorbital sinuses. Esophageal mucosa had a few necrotic plaques, and cloacal mucosa was covered with necrotic greenish diphtheritic membranes. No lesions were seen in the intestines including annular bands. Thymus and bursa were atrophied and hemorrhagic, and the bursal lumen was filled with cheesy exudates. The bursal atrophy could last for at least 39 days postinfection; however, the thymus recovered after 10 days of infection.



# Diagnosis

- Signs, post mortem and histopathologic lesions are greatly suggestive.
- Isolation and identification of virus confirms the diagnosis even in the absence of typical lesions.
- Samples should be collected from liver, spleen, bursa, kidneys, and cloaca.
- Primary virus isolation should be made by inoculation of susceptible 1-day-old Muscovy, white Pekin ducklings, or the CAM of 9-14 day-old embryonated duck eggs or duck embryo fibroblasts, liver, or kidney cells.
- Immunofluorescence tests, ELISA, or PCR, can be used to detect viral antigens in cell or tissue

# Differential diagnosis

- Differential diagnosis should be carried out with other diseases producing hemorrhagic and necrotic lesions in ducks as duck virus hepatitis, fowl cholera, necrotic enteritis, coccidiosis, Vit. E deficiency and specific intoxications.

# Prevention

- Adaptation of good hygienic measures is important to avoid direct and indirect contact with possibly contaminated material.
- Prevent contact between domestic and wild water fowl.
- Good hygiene and sanitation of drinking water sources.
- **VACCINATION:**
- Active immunity has been demonstrated following the use of a modified live-virus vaccine and inactivated tissue culture vaccine.
- It is assumed that both humoral and cell-mediated immunity are involved in protection and also for controlling disease outbreaks (interference or blockage phenomena).

# Prevention

- **VACCINATION:**
- An inactivated tissue culture-grown virus vaccine has been shown to provide protection against the virulent strain. This vaccine could be used in domestic and captive waterfowl without the risk of introducing a live virus.
- A chicken embryo-adapted DEV strain, avirulent for domestic ducks, has been used to prevent and control (emergency) DVE outbreaks on commercial duck farms and captive waterfowl.
- The vaccine can be used in the face of an outbreak, as it provides immediately protection after vaccination by interference phenomenon
- Attenuated live virus vaccine is administered by S/C or I/M routes (0.5 ml/ bird) in domestic ducklings more than 2 weeks of age.
- The breeding flocks are vaccinated and revaccinated annually.

THANK YOU