Duck viral enteritis (DVE)
**Other names:**
Duck plague, DVE

**Definition:**
This is an acute viral disease of Ducks, Geese and Swans characterized by weakness, thirst, diarrhea, short course, high mortality, and present lesions of the vascular, digestive, and lymphoid systems.
Etiology:
The causative agent is *Herpes virus*. This virus have ability for grows well on chorioallantoic membrane at 9-14 days-old embryonating duck eggs or on duck embryo fibroblasts. It's can be produced intranuclear inclusion bodies in hepatocytes of infected ducks.

Incubation period:
The incubation period extended from 5-7 days.

Susceptibility:
1. Wild and domestic ducks are affected.
2. All age groups and many varieties are susceptible; however, mostly adult ducks are affected.
**Transmission:**

1. The virus can be transmitted when health birds contact with infected birds or contaminated water.

2. Recovered birds remained source for viral infection.

3. It's suspected that viremic birds may be transmitted the infection through eating arthropods.

4. Vertical transmission has been reported experimentally.
Clinical signs:

A. In case of duckling we can seen:
   1. Ducks are usually infected at age 3 weeks old.
   2. Diarrhea.
   3. Dehydration.
   5. Cyanotic bill and death usually occurs in 1-5 days after infection
   6. Photophobia.

B. In case of adult ducks we can showed:
   1. Sudden death.
   2. High mortality.
   3. Decrease in egg production.
   4. Sick birds can be seen undergo from loss appetite, weakness, ataxia and photophobia.
   5. Adhered eyelids, nasal discharge, extreme thirst, and watery diarrhea.
   6. Unable to stand, drooping wings, and hang their heads down.
P.M. lesions:

1. Hemorrhages often occur on the mucosa of the gastrointestinal tract.

2. Edema may be present in the cervical region.

3. There is severe enteritis. There may be elevated, crusty plaques in the esophagus, ceca, rectum, cloaca or bursa of Fabricius.

4. Hemorrhage and necrosis in the annular bands discs of lymphoid tissue along the intestines is present.

5. The spleen is usually of normal or reduced size but the liver may be discolored and contain petechial hemorrhages.
**Microscopically:**
Present the *intranuclear inclusion bodies* in degenerating hepatocytes.

**Diagnosis:**
1. Typical clinical signs and lesions (especially demonstration of intranuclear inclusion and the virus in tissues using fluorescent antibody technique) are diagnostic.
2. The viral agent can be isolated in 9-14 days-old embryonated duck eggs incubated by the chorioallantoic route.
3. Acute and convalescent sera can be used to demonstrate an increasing antibody titer to duck viral enteritis.
Differential diagnosis:
1. Duck enteritis.
2. Pasteurellosis (fowl cholera).
3. Newcastle disease, highly pathogenic avian influenza, coccidiosis, and other causes of enteritis.

Treatment:
There is no effective treatment.

Prevention:
1. The commercial ducks should be isolated from free-living water fowl which are reservoirs of infection.
2. The quarantine and sanitary practices should be followed to prevent the introduction of this disease.
3. Live attenuated chickens embryo derived vaccine has been used for prevent outbreaks.
Nervous signs
Entritis in the infected duck
Intestinal Ulcers
Duck Viral Hepatitis (DVH).

**Definition:**
Its viral disease which responsible for economic loss in Ducks and characterized by short course, high mortality and by punctuate or ecchymotic hemorrhages in the liver.
**Etiology:**

This disease caused by an *Enterovirus* in the family *Picorna viridae* which responsible for *DVH type 1* and *3*. Its chloroform resistant and does not hemagglutinate. *DVH type 2* has been identified as an *Astrovirus*. DVH viruses stimulate a high degree of immunity in ducklings that survive infection and in inoculated adult ducks.
**Incubation period:**
The virus has a short incubation of around 24 hr in experimental birds.

**Transmission:**
1. Wild birds have been suspected of acting as mechanical carriers of virus over short distances.
2. The virus is excreted by recovered ducklings for up to 8 weeks after onset of infection.
3. Susceptible ducklings can be infected by contact with infected ducklings.
4. Rodents serve as mechanical factor for affected farms.
Clinical signs:
1. The sudden onset, rapid spread, short course, and focal, hemorrhagic hepatitis in young ducklings
2. Morbidity may be reached to 50–100%.

P.M. lesions:
1. The carcass may be in opisthotonous position.
2. Enlargement the liver with punctuate or ecchymotic hemorrhages.
3. The kidneys may be swollen and the spleen enlarged.
4. Airsacculitis and peritonitis may be observed.

Microscopically:
Microscopically there may be areas of hepatic necrosis, bile duct proliferation, and some degree of inflammatory response.
**Diagnosis:**

1. DVH type 1 can be isolated in embryonating chick or duck embryos or 1-day old susceptible ducklings and identified by serum neutralization test.
2. DVH type 2 can be identified through electron microscopy on liver or blood.
3. DVH type 3 cannot be isolated in chicken embryos and is difficult to reproduce in ducklings.
4. A direct fluorescent test on duckling liver has been reported.
**Differential diagnosis:**

The disease must be differentiated from duck viral enteritis, Newcastle disease, Avian influenza through susceptibility to chloroform and haemagglutination of erythrocytes.

**Treatment:**

Treatment is of no value.

**Prevention:**

1. Hyperimmune serum from flocks surviving duck viral hepatitis can be administered to ducklings.
2. An injection of 0.5 ml filtered serum is recommended using the intramuscular route.
3. Breeders can be immunized with a live attenuated chicken-embryo origin vaccine.
4. Rodents should be eradicated.
Stargazing
Duckling septicemia

**Definition:**
The disease caused economic losses in all areas where ducklings are reared commercially and characterized by cause morbidity and mortality during the first two weeks of the brooding period.

**Etiology:**
The disease caused by *Riemerella anatipestifer* is the principal pathogen responsible for duckling septicemia. Concurrent infections include E. coli, septicemia, salmonellosis and duck viral hepatitis.
**Transmission:**
1. Direct contact of susceptible ducklings with a contaminated environment.
2. Foot pad lesions from defective wire floors predispose to percutaneous infection.

**Clinical Signs:**
1. Affected ducklings show depression and ataxia.
2. Ocular, nasal discharge and respiratory rales.
**P.M. lesions:**
1. Septicemic changes characterized by perihepatitis, pericarditis and fibrinous airsacculitis.
2. Hepatomegaly and splenomegaly are observed.
3. In some cases, fibrinous meningitis occur, especially in ducklings which display nervous signs.

**Diagnosis:**
Diagnosis is based on isolation and identification of *Rimerella anatipestifer* from heart blood, liver or brain tissue on either blood agar or trypticase soy agar.

**Treatment:**
1. Supportive therapy and administration of water soluble tetracycline may be attempted.
2. Enrofloxacin can be administered in drinking water at a level of 250 – 300 gm. / 1000 liters drinking water for the first 5 days.
**Prevention:**

1. Managemental interventions including effective sanitation between placements, avoiding overcrowding and chilling should be implemented.

2. Multivalent or homologous bacterins have been prepared for administration to ducklings at 1 to 2 weeks of age.

3. Live attenuated vaccine against serotypes 1, 2 and 5 has been developed, which is administered to ducklings by the aerosol route or in drinking water at day-old.
Figure 1: Gross lesions of (a) fibrinous pericarditis; (b) congestion of meninges in *R. anatipestifer* affected ducklings