DUCK VIRUS ENTERITIS
(DVE; Duck Plague)

DEFINITION

Duck virus enteritis (DVE) is an acute viral disease of ducks, geese, and swans characterized by weakness, thirst, diarrhea, short course, high mortality, and by lesions of the vascular, digestive, and lymphoid systems.

OCCURRENCE

1. Wild and domestic ducks, geese, and swans (order Anseriformes) are affected. All age groups and many varieties are susceptible; however, mostly adults are affected. The blue-winged teal is the most susceptible and the pintail duck is the least susceptible.

2. In the United States the disease has occurred in New York, Pennsylvania, Maryland, California, and South Dakota. The disease has been reported in the Netherlands, France, China, Belgium, and India. Because wild waterfowl are migratory, it seems likely that the disease may occur in other countries that have migratory waterfowl.

HISTORICAL INFORMATION

1. The disease was first observed in the Netherlands in 1923. Initially it was mistaken for avian influenza but in 1942 it was clearly differentiated from avian influenza and was termed duck plague. Subsequently the disease was identified in many other countries.

2. In 1967 DVE appeared in white Pekin ducks raised commercially on Long Island, New York. It also was identified in wild waterfowl. An effort to eradicate the disease in the domesticated white Pekin duck appeared to be successful.

3. Multiple outbreaks of DVE have been recognized in California and it is classified as a reportable disease in this state. In the spring of 1973 the disease appeared in congregated wild waterfowl in South Dakota and resulted in the death of approximately 48,000 waterfowl, mostly ducks.

4. DVE is now considered to be enzootic in North America. Prior to the 1973 outbreak, DVE was considered an exotic disease by the USDA. It is being watched with interest because its ability to kill congregated, susceptible waterfowl is recognized.

ETIOLOGY

1. The etiologic agent is a herpesvirus. Although strains vary in pathogenicity, all appear to be identical immunologically.

2. The virus is nonhemagglutinating. This differs from the viruses of Newcastle and avian influenza, which do hemagglutinate and which must be differentiated in diagnostic work.

3. The virus grows best on the chorioallantoic membrane of 9-14-day-old embryonating duck eggs or on duck embryo fibroblasts. Initially it does not grow in chicken eggs although it can be adapted to them. The virus also can be isolated in ducklings, with Muscovy ducklings being the most sensitive.

4. The virus produces intranuclear inclusion bodies in a variety of cells of infected waterfowl.
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EPIZOOTIOLOGY

1. The virus can be transmitted when susceptible birds contact infected birds or an environment (particularly water) contaminated by them. Natural infection is limited to ducks, geese, and swans.

2. A carrier state for as long as 1 year has been demonstrated in wild ducks. Perhaps carrier birds under stress shed virus intermittently, thus exposing susceptible birds.

3. Because viremia occurs in affected birds, arthropods feeding on those birds may transmit the disease. However, this method of transmission is unproven.

4. Vertical transmission has been reported experimentally.

CLINICAL SIGNS

1. In young commercial ducklings, signs appear 3-7 days after exposure. Ducklings have diarrhea, a blood-stained vent, dehydration, and a cyanotic bill. Death usually occurs in 1-5 days.

2. In domestic breeder ducks there is a sudden, high, persistent mortality and a marked drop in egg production (25-40%). Sick birds show inappetence, weakness, ataxia, photophobia, adhered eyelids, nasal discharge, extreme thirst, prolapsed penis, and watery diarrhea. They soon become exhausted and unable to stand. They then maintain a position with drooping outstretched wings and with the head down. Tremors may be apparent. Morbidity and mortality are usually high but vary from 5 to 100%. Most birds that develop clinical signs die. Wild waterfowl are said to have similar signs. They often conceal themselves and die in vegetation near the water.

LESIONS

1. Hemorrhages are present at many sites and there may be free blood in body cavities, gizzard, or intestine. Hemorrhages often occur on the liver, in the mucosa of the gastrointestinal tract (including the esophageal-proventricular junction), throughout the heart, and in the pericardium and ovary. There may be edema in the cervical region.

2. There is severe enteritis. There may be elevated, crusty plaques in the esophagus, ceca, rectum, cloaca, or bursa of Fabricius. In young ducklings the esophageal mucosa may slough.

3. There is hemorrhage and/or necrosis in the annular bands or discs of lymphoid tissue along the intestine. The spleen is usually of normal or reduced size.

4. Initially the liver may be discolored and contain petechial hemorrhages. Later it may be bile-stained and contain scattered small, white foci as well as many hemorrhages.

5. Microscopically there may be intranuclear inclusion bodies in degenerating hepatocytes, epithelial cells of the digestive tract, and in reticuloendothelial cells.

DIAGNOSIS

1. Typical signs and lesions, along with epizootic losses, are highly suggestive of duck plague. The diagnosis can be strengthened if intranuclear inclusion bodies can be demonstrated or if the virus can be demonstrated in the tissues through fluorescent antibody tests.

2. The virus should be isolated and identified for confirmation. The virus will grow initially in embryonating duck eggs but not in chick embryos. Using known antisera to DVE, the virus can be identified by a neutralization test.
3. Retrospectively, it is possible to identify an outbreak of DVE if acute and convalescent sera are used to
demonstrate an increasing antibody titer to duck plague virus.
4. DVE must be differentiated from duck viral hepatitis, pasteurellosis, Newcastle disease, avian influenza,
coccidiosis, and other causes of enteritis.

CONTROL

1. Owners should prevent cohabitation or contact of their waterfowl with wild waterfowl. All appropriate
quarantine and sanitary practices should be followed to prevent disease.

2. All suspected outbreaks should be reported immediately to state authorities. They, with federal authorities,
will decide how an outbreak is to be handled. In commercially raised waterfowl, outbreaks were once
controlled by combining slaughter with indemnification and by the application of quarantine measures.
Presently, slaughter with indemnification has been discontinued and vaccination has been authorized on
certain premises.

3. A vaccine is available for prevention but approval by animal health authorities is required before it can be
used. It has not been authorized for general use.

4. A monitoring system for detection of DVE has been established in the United States. Suspected outbreaks
should be processed through official state or federal diagnostic laboratories.

TREATMENT

There is no effective treatment.