Marek’s Disease

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Marek's disease (MD) is a tumoral and fatal disease in chickens

The disease is a common lymphoproliferative disease of chickens, characterized by mononuclear cellular infiltrates in peripheral nerves and various other organs and tissues including iris and skin

Marek's disease also caused significant economic losses with deaths and productivity loss in Turkey

It is an immunosuppressive disease

Etiology

- *Herpesviridae (family)*
  - *Alpa-herpesvirinae (subfamily)*
    - *Mardivirus (genus)*
      - ***Gallid alpha-herpesvirus 2***
- DNA
- There are **three serotypes** of Marek's disease virus.
- Serotypes are detected by
  - monoclonal antibodies

Serotypes

Serotype-1 (*Gallid herpesvirus*-2, MDV-1): The group includes virulent and oncogenic viruses. The virulence of the viruses in this group was determined as moderate, virulent and very virulent.

Serotype-2 (*Gallid herpesvirus*-3, MDV-2 : includes non-oncogenic MDV. Serotype-1 which isolated in 1978 is included in serotype-2.

Serotype-3 (*Meleagrid herpesvirus*-1, Herpesvirus of turkey-1 (HVT)): Contains virus isolated from turkeys. Turkey herpes virus (HVT) does not cause disease in chickens

Epidemiology

- **Marek's disease has only been seen in chickens and game birds**. Experimentally, infection has been established in turkeys, pheasants and quails.
- Marek’s disease virus (MDV) is transmitted among chickens by direct and indirect contact.
- Transmission occurs primarily via the respiratory route following contact with contaminated environments.
- The virus is consistently present in the feather follicles of infected birds and is continuously shed into the environment.
- Infection mainly occurs through inhalation of dust particles containing infectious virus.
- **Vertical transmission does not occur**
- The disease has been reported in almost all countries worldwide

Epidemiology

- The incubation period varies according to the virulence of the virus, the dose, the transmission route, the genetic characteristics of the chicks and whether they are female or male.
- It is difficult to determine the incubation period in infection that occurs under natural conditions.
- Outbreaks can sometimes be seen in 3-4 week old chicks.
- The most severe cases in Marek's disease appear after 8-9 week

Clinical Symptoms

- Marek's disease is a progressive disease
- The symptoms of the disease are different. Symptoms are mainly neurolymphomatosis, acute signs of Marek's disease, ocular, lymphomatosis, and skin lesions.
- After the peripheral nerves are affected, a particular or complete paralysis is seen in the leg and wing.
- In the infected flock, paralysis may have occurred in only a few chickens. Paralysis may not be seen in other infected chickens.
- When the wing nerves are affected, the wing falls.
- When the neck nerves are affected, the head is kept down and sometimes torticollis is seen.

Clinical Symptoms

- When the vagus is affected, the crop expands and hangs, and difficult breathing is observed.
- Because locomotory disturbances are easily recognized, incoordination or stilted gait may be the first observed sign
- **A particularly characteristic clinical presentation is a bird with one leg streched forward and the other back as a result of unilateral parasis or paralysis of the leg**

Fowl paralysis. Spastic paralysis of limbs associated with peripheral nerve involvement in Marek’s disease

Transient paralysis. Flaccid paralysis of neck of young chicken 9 days after inoculation with Marek’s disease virus

Clinical Symptoms

- In infection with some highly virulent MDVs, lesions develop in the eye, resulting in blindness.
- In the acute form of Marek's disease, most of the chickens are weakness and depressed.
- Nonspecific symptoms such as weight loss, paleness, anorexia and diarrhea occur
- Usually, chickens that have clinical symptoms die.
- Not all infected chickens in the flock develop clinical symptoms.
- Morbidity was 60% or higher in some commercial flocks
- Appropriate and effective vaccines are applied to almost all laying hens. Therefore, morbidity and mortality in Marek's disease is below 5%.

Necropsy Findings

- Thickening (2-3 folds) and discoloration are seen in the affected peripheral nerves. Nerve thickening is unilateral.
- Lymphoid tumors are formed in different organs. These organs are lung, genitalia, ovary, spleen, liver, pancreas, proventriculus, muscle, and skin.
- Enlargement of the viscera may be several times bigger the normal organ size.
- Grayish discoloration is usually seen.
- Diffuse enlargement and nodular tumors are seen in the liver
- Some follicles in the ovary are normal and some are tumorous
- Mature follicles continue their function
- The proventriculus thickens and hardens

Necropsy Findings

- The affected heart is pale
- Macroscopic changes in the eye include discoloration of the iris (gray eye) and smooth removal of the pupil and progressively smaller hole.
- **Bursa Fabricius is atrophic**
- Changes in the skin can be seen after the cut hairs are plucked
- Gray-white interactions occur in the muscles

Diagnosis

Clinical symptoms and necropsy findings

Laboratory examinations

a) Virus isolation

b) Antigen detection

c) Antibody detection

d) Histopathological examinations

e) Molecular analysis

Protection and Control

- Layers and breeders should be vaccinated.
- The important thing in vaccination against Marek's disease is to vaccinate before the virus of the disease enters the body. For this reason, Marek's disease vaccines are applied as early as possible after chicks hatch.

Fowl Pox Infection

Etiology

- *Poxviridae (family)*
  - *Chordopoxvirinae (subfamily)*
    - ***Avipoxvirus***
- DNA
- The inclusion bodies (**Bollinger bodies**) formed by the virus in the cytoplasm of the cells and the virus particles (**Borrel particles**) contained in them can be easily seen with light microscopes (1500x magnification)
- The virus is resistant to environmental conditions, drying and some disinfectants
- They can survive on scabs, skin rashes, feathers and dust and remain infective for a long time (months, sometimes years)

Epidemiology

- Infection can occur in almost any season

  Mortality may exceed 50%

- The virus can easily enter through the lesions on the skin and mucous membranes and initiate the infection
- Fights, hard foods, scratches, and the resulting microscopic and macroscopic lesions play an important role in the entry of the virus into the body and the spread of the infection
- Transmission is usually ocur by direct contact from body portants or by dusts, droplets, hairs, etc. It occurs as aerogen with the viruses in it

Clinical Symptoms

After the virus enters the body, the first symptoms begin to appear within 4-15 days, depending on the sensitivity, age, breed, immune status, environmental conditions, stress factors, pathotype of the virus, virulence and other conditions.

The incubation period differs between bird species

1. **Skin form (cutaneous form, flower form):** Develops in the form of small lesions and blisters on the face, comb, beard, mouth and eyes of chickens, around the hairless parts of the body. **In mild forms** of the disease, significant clinical signs cannot be observed. However, reductions in egg production and regressions in growth can be noticed. As the lesions enlarge and their number increases, weakening and death occur (**in severe forms**).In mild forms of infection, scabs may shed within 1-3 weeks, and open wounds may heal if there is not much contamination

2. **Diphtheric form**: Occurs in mucous membranes. Yellow, necrotic pseudomembranes develop in the mouth, tongue, pharynx, esophagus, larynx, crop, trachea. These also grow over time and can merge with each other. When these membranes are lifted by holding them with forceps, bleeding surfaces occur in their places. Since these lesions prevent feeding and breathing, they cause weakening, wheezing and difficulty breathing, asphyxia and death in animals.

Necropsy Findings

Cutaneous (Dry) Form of Fowl Pox: Lesions are commonly observed on **featherless areas**, including the **comb, wattles, periocular region, beak, ear lobes, legs, and wing web**.

- These areas show **prominent crusted lesions**.
- Lesions progress through characteristic **stages**:
  - **Papules**: small, reddish raised nodules
  - **Vesicles**: fluid-filled blisters
  - **Pustules**: vesicles filled with pus
  - **Scabs (crusts)**: dry, dark brown crusted lesions

Diphtheritic (Wet) Form of Fowl Pox: Characterized by the presence of **thick yellow-white plaques and pseudomembranes** in the **oral cavity, larynx, and trachea**.

- These lesions are **firmly attached to the mucosa**, and when removed, they leave **ulcerated and bleeding surfaces**.

Diagnosis

1. **Clinical and necropsy findings**: **Lesions developing on the skin and mucous membranes in fowl pox disease may be sufficient to diagnose the disease**. However, some bacterial (CRD, Infectious coryza, chronic cholera, Infectious sinusitis, etc.), viral (ILT, IB, ND, etc.), fungal (moniliasis, aspergillosis, etc.) and other disorders (skin sores, A avitaminosis, etc.) they can be complicated
2. **Laboratory examinations**

a. Microscopy

b. Virus isolation

c. Serological tests

d. Biotechnological methods

Treatment

- Since fowl pox disease is a viral infection, it cannot be treated with antibiotics. However, broad-spectrum antibiotics can be used to prevent bacterial contamination and secondary infections
- Glycerin iode is applied on the lesions on the face and mouth of the animals and the application is continued until the wounds heal.
- The crusts of the wounds formed in the mouth are removed by holding them with a forceps and glycerin iode is applied to the wound surface. Antibiotic pomade and solutions can also be used for the same purpose

Protection and Control

Biosecurity

Vaccination

Avian Encephalomyelitis (Epidemic Tremor)

Etiology

- *Picornaviridae (family)*
  - *Tremovirus (genus)*
    - ***Avian encephalomyelitis virus*** (AEV),

Epidemiology

- Chicken, turkey and duck susceptible to disease
- Avian encephalomyelitis virus (AEV) has two main pathological types:

1. **Wild-type strains:** These strains show intestinal (enterotropic) tropism.Transmission occurs mainly via the fecal–oral route.
2. **Embryo-adapted strains:** These strains are highly neurotropic. They are obtained through continuous passage of an enterotropic strain in SPF chickens under laboratory conditions. Cause severe neurological signs.

- AEV can be transmitted by both vertical and horizontal routes.
- Contaminated feed, water, litter, equipment, and personnel play an important role in horizontal transmission.

Clinical Signs

- Depression, ataxia and tremor in young animals
- Decrease in egg production in layers, decrease in hatchability in breeders
- Single and/or double sided opacity in the lens
- Nervous symptoms appear 1 week after hatching
- Although the disease is usually seen at 5-6 weeks of age, it has also been reported in older animals
- Mortality up to 15% and morbidity 60% in infected poultry
- Clinical signs are more severe in turkey
- 5-10% decrease in egg production reaches normal level after 2-3 weeks
- Approximately 5% decrease in hatching yiel
- Weekly mortality in animals increases by 0.2-1% in egg production

Necropsy Findings

- Clinically, affected poultry usually show no prominent signs, except for lens opacity.
- At necropsy, no pathognomonic macroscopic lesions are observed.
- Histopathological examination reveals widespread inflammation of the nervous system, with neuronal degeneration and inflammatory cell infiltration.

Diagnosis

Clinical and necropsy: It should be differentiated with ND, MD, and nutritional encephalomalacia. Clinical signs are not sufficient to diagnose the disease.

Laboratory diagnosis:

- Histopathological examinations
- Virus isolation; 5-6 days old egg yolk inoculation
- VN, ELISA, FAT
- Molecular analysis

Control

Vertical transmission

Biosecurity

Vaccination

- Live vaccine
- Inactivated vaccine