Marek's Disease in Chickens: Description and Prevention

Overview of marek's disease in chickens and tips to prevent the diseases via vaccination.

Susceptibility of the agent

Being an encapsulated virus, this agent is not very resistant to common chemicals. However, cellular debris from the feather follicles provides the virus with additional protection, conferring the ability to tolerate ambient conditions and some disinfectants. Viral particles protected with dander can remain infective for a very long time (months or years). Efforts to disinfect the premises should include removal of organic material followed by washing with a detergent to remove the cellular protection surrounding the virus; such practices will greatly increase the efficacy of common disinfectants.

Occurrence

Marek’s disease is common around the world and is a very common infection in small backyard flocks that have not been vaccinated (or have been improperly vaccinated) against the disease. The disease only affects chickens (serotype 1) and to some extent turkeys (serotype 3). The disease is uncommon in birds younger than 6 weeks old and common in birds from older than 6 weeks.

Etiology: cell-associated herpesvirus

Features of the agent: double-stranded DNA, encapsulated. Three serotypes have been described: serotype 1 (Gallid Herpesvirus 2), serotype 2 (Gallid Herpesvirus 3; avirulent), and serotype 3 (Meleagrid or HVT, Herpesvirus 1). Based on the pathogenicity of the agent, four pathogroups are generally recognized: mild, virulent, very virulent, and very, very virulent.
Pathogenesis

The virus develops in the feather follicles and it is shed in combination with feathers and dander. Viral particles are inhaled, and phagocytes likely transfer the viral particles to lymphoid tissue. Affected lymphocytes are detected as early as 5 days post-infection in the nerves of susceptible birds (maternal antibody negative and genetically susceptible chicken lines). The virus then affects the thymus and bursa of Fabricius producing massive apoptosis in their lymphoid tissue, generating variable degrees of immunosuppression. After a few days, a stage of latency begins. At about 14 days post-infection, viral replication begins in the feather follicle epithelium, where complete viral particles are shed. In the field, transient paralysis is normally seen after 8 weeks of age. However, in unvaccinated birds field cases can be sporadically seen after 3 weeks of age. Protective immunity starts with the engagement of NK cells which will lyse virus-infected and tumor cells (without the need for a previous antigen presentation). Further development of cellular immunity (Cytotoxic T cells) plays a role in controlling the extent of infected cells. Antibodies are produced approx. two weeks after the infection, however, since much of the infection occurs inside the cells the efficacy of such an immune response is limited.

Clinical manifestations

They will be variable depending on the tissue in which neoplastic infiltration is taking place. The most typical manifestation in PA involves infiltration of the sciatic nerves, which produces an asymmetric and progressive paralysis of one (more common) or both legs which normally progress to a complete spastic paralysis of one or more extremities. Brachial, cervical, and/or vagus nerves can also be affected, in which case paralysis of the wings, torticollis, and/or pendulous crop are the main signs, respectively. Infiltration of the iris is also seen on a regular basis. In this case, the pupil of the affected eye may appear smaller and irregular, which can affect the vision of the animal. Weight loss, anorexia can be observed especially in the chronic cases of the disease. Under commercial settings, birds can appear weakened and dehydrated due to increasing difficulty to reach feed and water. Death often is the result of starvation.

Necropsy findings

Inflammation in nerves with loss of typical striations is commonly seen in the sciatic nerves. The nerves appear much thicker than normal (2 or 3 times thicker) and may have a yellowish appearance due to the accumulation of edema on their surface (picture 2). Visceral tumors can be observed in some birds (picture 3). These tumors can be seen infiltrating many organs and tissues (liver, gizzard, heart, feather follicles, iris, nerves). As a result of the infiltration hepatomegaly and splenomegaly can occur (picture 4); however, as a practical tool to differentiate from other neoplastic diseases of poultry, in Marek’s disease, the bursa of Fabricius is never affected with tumors.

Diagnosis

A tentative diagnosis can be made according to the history of paralysis and postmortem lesions showing enlarged nerves and tumors not involving bursal tissue. ELISA and PCR assays are available for confirmation of the presumptive diagnostics.
Relevant differential diagnosis
Newcastle, avian encephalomyelitis, lymphoid leukosis, other neoplasia, trauma.

Prevention and treatment
Treatment is not available. Commercial vaccines are available and highly efficacious to prevent the disease. Vaccines should be used as early as possible, ideally at day of age. The vaccine must be injected subcutaneously. It is very important to consider that birds are not immediately protected against the disease. Birds will be protected only once a protective immunity develops which occurs 7-10 days after vaccination.

Virus from serotype 2 (Gallid Herpesvirus 3; avirulent) and serotype 3 (Meleagrid or HVT, Herpesvirus 1) are commonly used in the production of commercial vaccines. Immunity generated by the vaccines protects against early replication of virulent viruses in lymphoid organs. However, it is important to notice that vaccines do not prevent the infection from taking place.

The best option is to get chickens that were vaccinated in the hatchery. Vaccines can be currently found at First State Veterinary Supply, Jeffers, and Valleyvet. The vaccine must be prepared following the instructions printed on the label. The vaccine must be used within 2 hours and then discarded. After this period the vaccine will not be effective. Another disadvantage of the available commercial vaccines is that the vial contains 1,000 doses. Fortunately, the vaccine is not expensive and the vial with a thousand doses should cost about $25-35.

References