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124 – Marek's disease III

Pathology

The lesions found in Marek's disease are mainly lesions in nerves and visceral lymphomas. No macroscopic changes are seen in the brain but spinal ganglia often show gross enlargement.

Nerves may show striation loss or grey/yellow discolouration and sometimes look oedematous. Often the plexi of the sciatic and bronchial nerves are enlarged.

Lymphomas can be large masses (up to four times organ size) or small focal nodular growths.

Skin lesions are an important cause of condemnations in broilers and are nodular in nature centred on feather follicles. In the virulent form of this disease the skin of the shank of the legs may be reddened.

Pathogenesis

There are usually four phases to infection – an early productive-restrictive virus infection that causes primary degenerative changes, a latent infection, a second productive-restrictive infection phase which coincides with permanent immunosuppression and, finally, the proliferative phase.

Immunity

Infection with pathogenic Marek's disease virus or vaccine strains results in the activation of innate and acquired immune responses but may also cause immunosuppressive effects, especially after infection with pathogenic serotype 1 strains. The interactions between immune responses and immunosuppression are important in the pathogenesis of Marek's disease.

When it comes to acquired immunity chickens infected with Marek's disease virus develop antibodies within two weeks but due to the cell-associated nature of Marek's disease, antibodies are of limited importance in Marek's disease immunity.

HVT, attenuated Marek's disease virus and serotype 2 protect against early replication of virulent viruses in lymphoid organs and reduce the level of latent infection but do not prevent superinfection.

Everything associated with Marek's disease immunity is complicated!

Diagnosis

Infection is ubiquitous but disease is not. Infection can be diagnosed by viral isolation, demonstration of viral DNA or antigens in tissues or the detection of antibodies.

Disease diagnosis can be difficult because there are no pathognomonic gross lesions. Diagnosis is based on clinical data, gross pathological findings, histopathology and virological criteria.

Differential diagnosis includes avian leucosis and for nervous signs – myeloid leucosis, myeloblastosis, erythroblastosis, ovarian tumours, other tumours, riboflavin deficiency, tuberculosis, histomoniasis, genetic grey eye, Newcastle disease, avian encephalomyelitis and joint lesions.