1. Marek's Disease

Disease background

The virus was discovered in 1907 by Jozsef Marek, a Hungarian vet and there are non-pathogenic, mildly virulent, virulent and very virulent strains.

Cause

This is a herpes virus which gravitates towards lymphoid tissue (lymphoid tissue is spread throughout a chicken, unlike a mammal where it is confined mostly to lymph nodes) and also causes demyelination of peripheral nerves (leg and wing). Various strains of the virus have been isolated and there are two types, one which lives in the cell and one which is cell-free and lives in the feather follicle. The virus replicates in the host chicken's lymphoid tissue and is shed in feather dander.

Clinical signs

Chickens from 6 weeks of age are affected, symptoms are most frequently seen 12-24 weeks of age with the hormonal stress of point of lay being a classic time for the signs to appear. Older birds can sometimes be affected if stressors (changes in weather, food, handling, environment) are not minimised. Females are more susceptible than males. Affected birds go lame and this can be mistaken for a sprain or injury, often with one leg forward and one back. The lameness worsens despite anti-inflammatory drugs and the bird uses one or both wings order to balance itself.

Mortality is variable and depends on which of the peripheral nerves is affected but leads to progressive spastic paralysis of the legs and wings with the bird remaining quite bright and still eating. Sometimes, if the neck nerves are affected, the neck can twist around. There is an acute form where birds may die suddenly with no symptoms and tumours may be found in the liver, gonads, spleen, kidneys, lungs, gizzard, heart, muscle and skin.

Transmission

The virus can remain viable for at least one year in feather dander and henhouse dust. The virus is ubiquitous in poultry worldwide. Infection in chicks occurs by inhalation and two weeks later, the virus is shed by the infected chick in feather dander and by oral and nasal secretions. The virus is NOT passed on through the egg. Infected and recovered birds continue to shed the virus for life.

Economic impact

Affected chickens must be culled as their welfare is severely compromised by this disease.

Diagnosis

Lack of response to treatment for lameness, clinical signs of paralysis and post mortem lesions.
Treatment (control)
Cull any affected birds. This increases the resistance to the disease in the surviving birds.

Vaccination is feasible, especially if Silkies or Sebrights are kept. These are very susceptible to clinical signs of Marek's and there would be few of these breeds seen at exhibitions if vaccination was not used. The vaccine is administered by injection when chicks are ideally dayold or before 3 weeks old. The vaccine is not effective in older birds.

In other breeds, using vaccine can hide the virus and so the whole stock gets progressively more susceptible (weaker) without any symptoms and if birds are sold without the recipient being told of the vaccination, the birds can pass on the virus to unvaccinated chicks, thereby bringing the disease to a flock which may have been free of it before.

Prevention
Good biosecurity is important, quarantine any new stock for 4 weeks. Rear chicks for 2-3 months away from adult feather dander if adult birds have shown symptoms. Ask vendors if stock has been vaccinated. Be prepared to vaccinate chicks at under 3 weeks.

Genetically resistant breeds include the Fayomi.

2. Other tumours

Disease background
The avian leukosis/sarcoma group of viruses was discovered by Ellerman and Bang in 1908 to cause erythroid leukemia - the first virus shown to cause leukaemic disease. Subsequently, an avian sarcoma virus was the first solid tumour shown by Rous (1911) to be transmitted by a virus. These diseases have become widely used as model systems of viral oncogenesis in biomedicine.

Cause
Other tumours in poultry (not Marek's disease, see above) are caused by retroviruses and these include the avian leukosis/sarcoma viruses. The genetic makeup of lymphoid leukemia is associated with slow cell transformation and tumour development over several months. Other avian oncoviruses cause rapid neoplastic tumour development within a few weeks.

Clinical signs
Any or all of the following: poor appetite, weak and emaciated, diarrhoea, pale wattles, enlarged liver.

Lymphoid leukemia (most common): enlarged liver, spleen, bursa, kidneys, ovary.

Erythroid leukemia (rare): moderately enlarged liver and spleen, leukaemia, liquid cherry red bone marrow.

Myeloid leukemia (sporadic): enlarged liver, spleen, kidneys, ovary, yellowish grey bone marrow.

Osteopetrosis (rare): thickening of long bones in legs and wings.

Transmission
There are two types of mechanism: lymphoid leukemia virus (most common) slowly transforms cells into neoplastic ones and the acutely transforming viruses do the damage faster, all of them using and damaging genes and causing tumours which can include fibrosarcoma, chondroma, endothelioma, haemangioma, nephroblastoma and hepatocarcinoma - almost anywhere in the chicken as lymphoid tissue is spread throughout the bird (unlike mammals which have lymph nodes).

Lymphoid leukemia: incubation period from infection to the developed disease and death is about 4 months. Losses occur from 5-9 months of age in egg-laying and breeding stock. Other leukemia viruses affect adults sporadically.

The virus is ubiquitous in poultry worldwide and is passed down through the egg as well as transmitted by direct or indirect contact. Virus survival outside the body is only hours, so the disease is not very contagious.
Economic impact

Birds affected by lymphoid leukosis die in about four months but pass the disease through the egg before symptoms are apparent. Less virulent strains of the virus do not produce tumours but egg production is severely depressed and hatchability affected.

Diagnosis

There are virological tests available, plus post mortem lesions and tumours are diagnostic.

![Fig 4: The internal organs of a chicken affected by lymphoid leukosis](image)

Treatment (control)

No treatment or vaccines are available. Control is based on high standards of hygiene and selection for resistance to the viruses by culling affected birds.

Prevention

Some breeds are resistant to tumour development. Good husbandry with minimal stress will help prevent the disease transmission.

![Fig 5 Osteopetrosis](image)