

Marek's mortalities

By Lisa Schmidt

The highly contagious Marek's disease virus is a common cause of mortality in backyard chickens – and the clinical signs are diverse and overlap with several other common conditions in poultry flocks. So what's the best way to diagnose it?

Marek's disease (MD) is a major cause of death in backyard chickens (Cadmus et al., 2019; Mete et al., 2016). It is caused by an alpha herpesvirus that produces lymphoproliferative tumours (lymphoma), primarily in chickens and to a lesser degree in quail (Sellers and Ojkic, 2019). Turkeys, pheasants and partridges, while rarely infected, have also been reported as having the disease (Sellers and Ojkic, 2019; Schock et al., 2016).

MD virus is highly contagious and can survive in the environment for up to eight months. It is shed in feather follicle dander, and the infectious dander can travel over long distances. Infection occurs via the inhalation of infected dander. Like other herpesviruses, infection is lifelong and the virus can become latent. Carriers may or may not be clinically ill and can shed the virus intermittently throughout their lives (recrudescence).

Infected live birds, fomites infected with feather dander and other poultry products are sources of infection. Large commercial flocks primarily control MD through strict biosecurity protocols and in-ovo vaccination. Unfortunately, owners of backyard flocks generally do not implement biosecurity measures and vaccination is often not practical, as vaccines are sold in vials of 500–1,000 doses.

TABLE 1. **Clinical signs of Marek's disease virus in poultry**

CLINICAL SIGNS	
Non-specific	<ul style="list-style-type: none"> ➔ Depression ➔ Inappetence ➔ Weight loss ➔ Anaemia ➔ Dehydration ➔ Diarrhoea ➔ Sudden death
Neural form (classical MD)	<ul style="list-style-type: none"> ➔ Leg weakness and lameness that progress to paraplegia and paralysis ➔ Flexing of toes ➔ Torticollis ➔ Stiff gait or hobbling ➔ Drooping of wings ➔ Lateral or sternal recumbency, with legs extending backwards, to the side or in a hurdler's position ➔ Crop stasis ➔ Dyspnoea
Ocular form	<ul style="list-style-type: none"> ➔ Blindness ➔ Patchy or diffuse grey discolouration and distortion of the iris, with indentation of its margin ➔ Pupillary shape change from circular to ovoid, elliptical or pear-shaped, and a reduction in size ➔ Death from starvation or dehydration due to inability to reach food and water
Visceral (acute) form	<ul style="list-style-type: none"> ➔ Nodular tumours in different organ tissues ➔ Non-specific clinical signs
Cutaneous form	<ul style="list-style-type: none"> ➔ Nodular skin lesions ➔ Severe necrotic dermatitis of the comb



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FIGURE 1: Cutaneous MD. Round, nodular, multifocal to coalescing lesions are present in the skin, especially around feather follicles. On non-feathered skin the lesions may appear red. Inset shows a cross-section of skin with nodular dermal lymphoid proliferation.

PHOTO CREDIT: SAWANG KESDANGSAKONWUT, IMAGE TITLE F34335, SKIN: FOLLICULAR INFILTRATION OF MAREK'S DISEASE. ACCESSIBLE AT: [HTTPS://DAVISTHOMPSONFOUNDATION.ORG/IMAGE-DETAIL?IMAGE=F34335](https://davisthompsonfoundation.org/image-detail?image=F34335).



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FIGURE 2: Cutaneous MD. Severe necrotic dermatitis of the comb and wattle in an adult chicken.

PHOTO CREDIT: BRENDA M BRYAN, VETERINARY DIAGNOSTIC LABORATORY, MANITOBA AGRICULTURE, WINNIPEG, MANITOBA, CANADA.

Clinical signs

In large commercial flocks, MD outbreaks peak when the birds are between two and seven months old (Sellers and Ojkic, 2019), and clinical signs are not reported before three weeks of age. By comparison there is no age or breed predisposition reported for small backyard flocks (Mete et al., 2016). Therefore, in outbreaks on lifestyle blocks, clinical signs will commonly be seen in both juvenile and adult birds concurrently and will not be restricted to one age group.

Clinical signs can be non-specific or organ-system specific, with the latter primarily targeting the central nervous system, eyes, viscera and skin (Table 1). In addition, MD can cause immunosuppression with secondary bacterial infections. Clinical signs in some birds may wax and wane before they succumb to the disease.

Gross lesions

While the cutaneous (Figures 1 and 2) and ocular (Figure 3) forms of MD have lesions that can be seen in live birds, lesions in the visceral (Figure 4) and neural (Figures 5 and 6) forms are only seen on postmortem examination and/or microscopically. Visceral tumours can affect any internal organ, with the liver, spleen and ovary being the most common. However, the kidneys, heart and proventriculus can also be infiltrated by neoplastic lymphocytes.

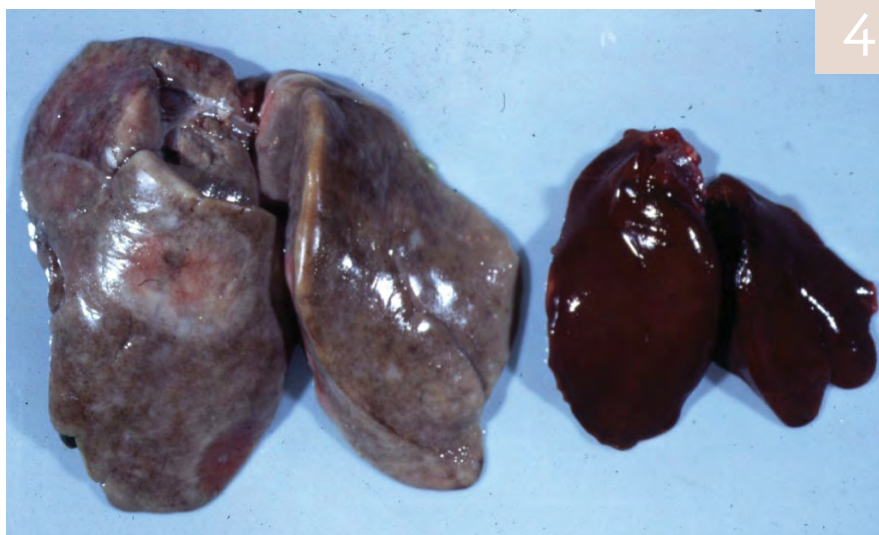




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FIGURE 3: Ocular MD. Unilateral greying of the iris (left) due to lymphoblastoid cell infiltration with an irregular, eccentric pupil that has a decreased size compared to the normal eye (right). Lesions may be unilateral or bilateral and clinically are associated with blindness.

PHOTO CREDIT: HARRINGTON, IMAGE TITLED F28834, IRIDOCYCLITIS MAREK'S DISEASE AVIAN HERPESVIRUS AND NORMAL CHICKEN. ACCESSIBLE AT: [HTTPS://DAVISTHOMPSONFOUNDATION.ORG/IMAGE-DETAIL?IMAGE=F28834](https://davisthompsonfoundation.org/image-detail?image=F28834).



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FIGURE 4: Visceral MD. The liver (left) has generalised organ enlargement due to tumoural lymphoid proliferation, with grey to tan discolouration compared to normal liver (right). Alternatively, tissues can have multifocal tan nodules. Visceral tumours can also occur in the spleen, kidney, heart, ovaries and proventriculus. Clinical signs are non-specific and dependent on the organ affected.

PHOTO CREDIT: LUGINBUHL, IMAGE TITLED F11277, LIVERS NORMAL (RT) VS MD HEN. ACCESSIBLE AT: [HTTPS://DAVISTHOMPSONFOUNDATION.ORG/IMAGE-DETAIL?IMAGE=F11277](https://davisthompsonfoundation.org/image-detail?image=F11277).

Histologic lesions

Microscopically, most cellular infiltrates are composed of CD3+ T cells. MD lesions can further present as proliferative masses of pleomorphic lymphocytes (Type A) or as inflammatory infiltrates composed of small lymphocytes and plasma cells (Type B).

In peripheral nerves, lesions may be present segmentally (Figure 7). Both proliferative and inflammatory patterns (Figure 8) are seen and either can result in demyelination. However, visceral organ tumours (Figures 9 and 10) are mostly made up of pleomorphic lymphocytes and may or may not have inflammatory cells.

Differential diagnoses

Histologically, there are very few diseases that resemble MD, and the biggest differential diagnosis is avian leukosis (a retrovirus). As it rarely occurs in peripheral nerves, the iris or skin and is usually due to the proliferation of lymphocytic B-cells, routine histology or immunohistochemistry can be used to differentiate the two diseases.

Overall, clinical signs are very diverse and overlap several other common conditions in poultry flocks. Diarrhoea, dehydration, lethargy, weakness, recumbency and sudden death could be due to enteric pathogens (eg, coccidiosis

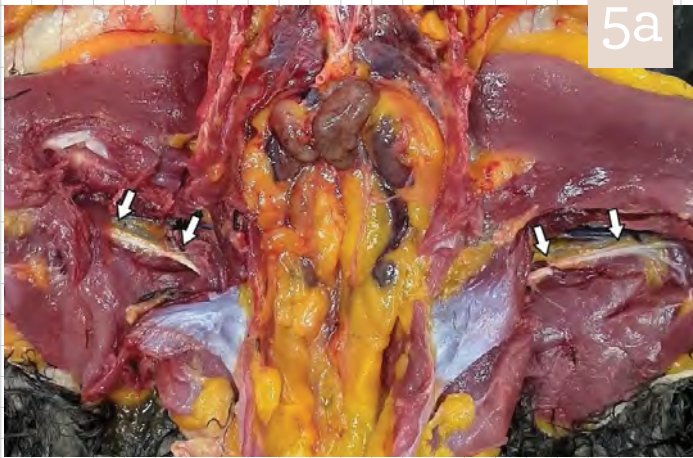


FIGURE 5A: Normal sciatic nerves (arrows) in a partridge. The keel and pectoral muscles are removed.

FIGURE 5B: Neural MD. Mildly enlarged and rounded sciatic nerves (arrowheads) with loss of striations in a chicken. Neural lesions can be subtle and may require histology to detect in many cases. Clinical signs include leg weakness, lameness, stiff gait, paresis, paralysis and recumbency (eg, hurdler's position).

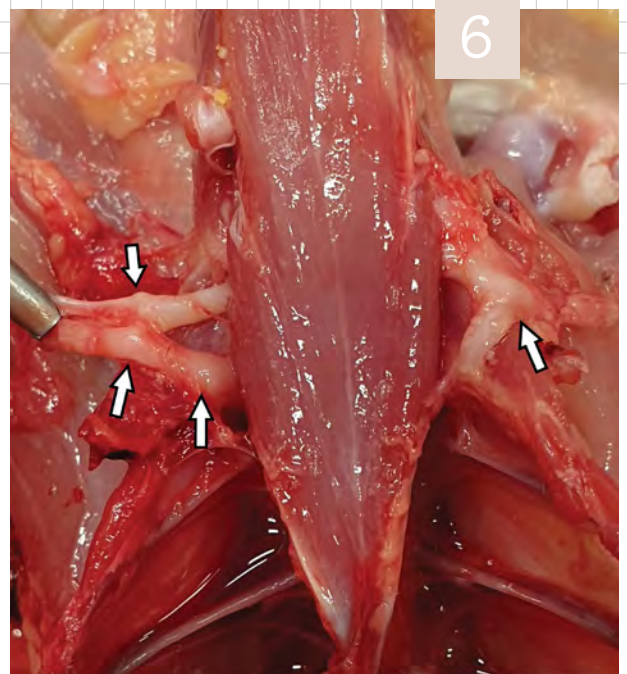


FIGURE 6: Neural MD in a chicken. Enlarged and rounded brachial plexus nerves (arrows) with loss of gross striations. Trachea and lungs have been removed. Associated clinical signs include drooping wings.

and histomoniasis). Weakness, stiff gait, paresis and recumbency could be due to nutritional diseases (eg, calcium/phosphorous imbalances or deficiencies, and riboflavin deficiency). Respiratory disease would need to be differentiated from other causes, including bacterial pneumonia, aspergillosis, lung worm and viral diseases (including some foreign animal disease viruses).

Diagnostic testing options

There are two main options for testing for MD virus:

1. Postmortem examinations with histology looking for characteristic lymphocyte infiltrates in the brain, peripheral nerves, feather follicles and other visceral organs. If these examinations are performed in the field, multiple peripheral nerves should be collected, including both sciatic and brachial plexuses. If neural involvement is not seen on

histology, PCR (polymerase chain reaction) testing or immunohistochemistry would then be needed to differentiate MD from avian leukosis virus.

2. PCR assays for MD virus can be performed on peripheral blood, spleen or tumours.



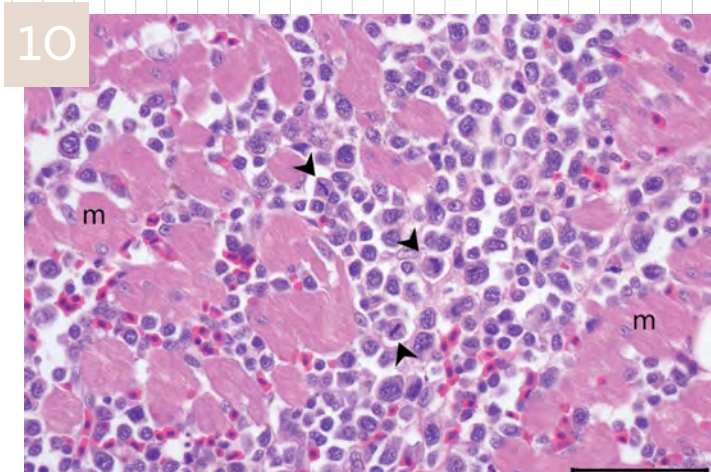
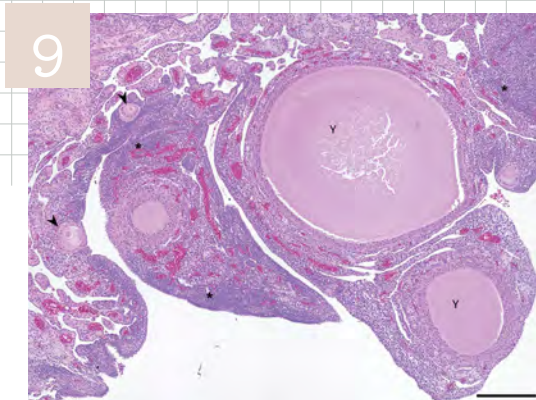
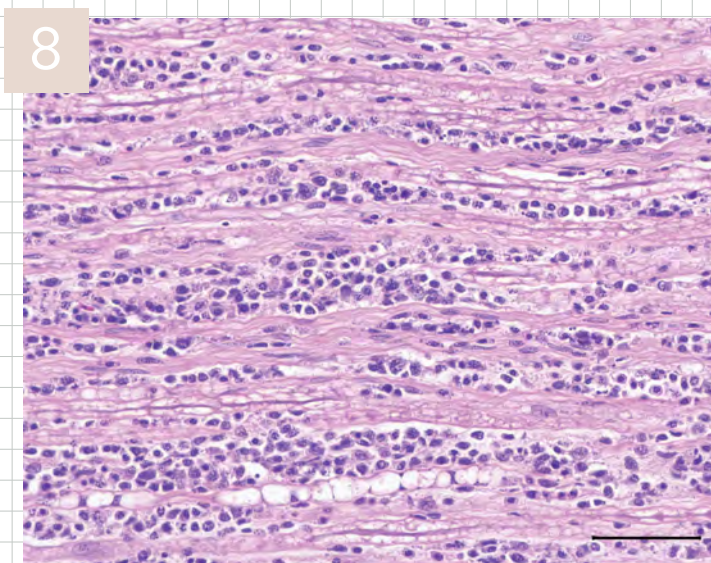
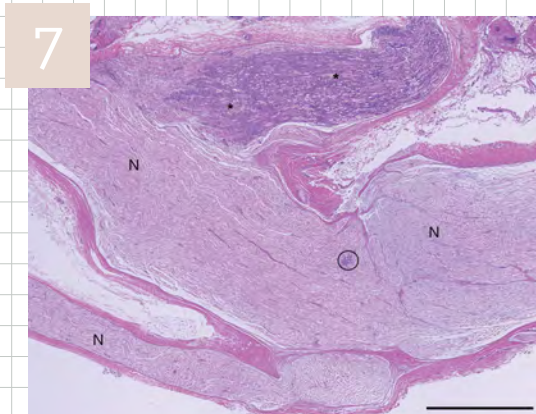


FIGURE 7: Neural MD. Brachial nerve plexus from the nerve shown in figure 6. The main nerve and one branch (N) are relatively unaffected with a small focus of neoplastic cells (circled), and a larger branch is heavily infiltrated by neoplastic lymphocytes (*). This demonstrates the variability of lesions within nerves and the need to submit multiple peripheral nerves from suspected cases. Bar = 1mm.

FIGURE 8: Neural MD in a sciatic nerve. Nerve fibres are separated by mostly an inflammatory cell population composed of lymphocytes and plasma cells. Bar = 50 microns.

FIGURE 9: Visceral MD in an ovary. The cortical stroma is expanded by neoplastic lymphocytes (*) that surround developing follicles. Follicles contain variable amounts of yolk-laden cytoplasm (Y), and a few early follicles have oocyte nuclei (arrowheads). Bar = 200 microns.

FIGURE 10: Visceral MD in a heart. Pleomorphic lymphocytes separate myocardiocytes (m). Mitoses (arrowheads) are regularly present. Neoplastic cells are occasionally admixed with a few small lymphocytes and macrophages.

Summary

Marek's disease virus is a common cause of mortality in backyard chickens. As its clinical signs overlap with other common disease presentations, the work-up for MD should include physical examinations to try to differentiate between weakness and neurologic disease, FEC on excreta (especially in cases of sudden death or diarrhoea), PCR testing and postmortem examination with histology. ☺

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