

Paws claws and judder things



February 2023

Heavy rain and leptospirosis

ALICE FRASER

A warm, wet summer certainly brings an abundance of feed for livestock grazing or to harvest, but it also brings more disease issues to consider.

For sheep farmers, such conditions can cause increased cases of fly strike, early bouts of Haemonchus and the threat of an upcoming high sporidesmin challenge, to name a few. In addition, this wet summer season, Gribbles Veterinary have seen an increased incidence of leptospirosis in weaned lambs.

Background:

Leptospirosis is a globally important disease which can affect all mammalian species as well as man, caused by pathogenic species of *Leptospira*. These slender, helical, motile, spirochete bacteria, harbour in the kidneys of chronically infected, carrier animals and are shed intermittently in their urine into the environment. Animal reservoirs of the disease include livestock as well as wildlife, typically rodents. In addition, leptospires survive in wet soils, leading to contamination of surface water following heavy rainfall and flooding.

There are multiple species of *Leptospira* and numerous serovars (the complexity of the classification is beyond the scope of this article). The most significant serovar causing acute, severe clinical disease in young lambs is *Leptospira interrogans* serovar Pomona. Pigs as well as wildlife are considered to be the maintenance host of this serovar but some outbreaks in lambs have no apparent association with pigs; environmental contamination being the likely source.

Leptospira borgpetersenii serovar Hardjo, the most common serovar in cattle and deer (and host-adapted in these species) showed a high higher seroprevalence in sheep than serovar Pomona in an epidemiological study (Dreyfus et, 2018). However, serovar Hardjo in sheep is more often subclinical and not associated with the acute severe clinical disease observed in lambs with serovar Pomona.

Serovar Pomona outbreaks in lambs:

The typical picture is sudden deaths and malaise in several lambs in a mob, with possibly 10 to 20 dead lambs. On opening an affected lamb carcase (usually in good body condition), the key necropsy findings are: marked jaundice of the omental fat, generalised carcase pallor, yellow/brown liver, dark urine and dark swollen kidneys.

Necropsy samples for *Leptospira* diagnosis:

- > fresh kidney and urine (pooled for PCR);
- > fresh liver if copper/zinc toxicities are possible causes to differentiate (see below);
- > fixed kidney and liver for histopathology (and a range of other tissues if other differentials are considered).

Sick lambs:

- > collect urine for PCR;
- > EDTA blood for haematology (including blood film analysis) for differential diagnoses;
- > serum samples for the first of paired serology tests for *Leptospira* serovar Pomona (collect the second serum sample approximately two weeks later to

Typical diagnostic results confirming *Leptospira* serovar Pomona:

demonstrate rising titres).

Histopathology:

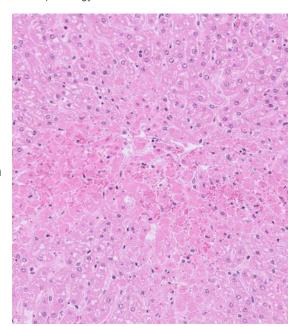
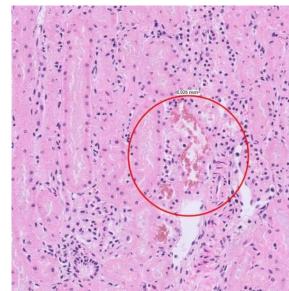


Figure 1: Liver, centrilobular degeneration, necrosis. H&E 20x.

Figure 2: Kidney, haemoglobinuria (intratubular haemoglobin casts), H&E 20x.



Leptospira PCR on fresh kidney tissue and/or urine:

> False negatives can occur with urine samples alone as the pathogens are intermittently shed from the kidneys.

Serology:

- > Leptospira serovar Pomona: rising titres observed.
- > Leptospira serovar Hardjo, often low background titres (probably maternal).

Differential diagnoses:

Other causes of haemolytic anaemia in sheep include:

- Copper, zinc toxicity, chronic or acute.
 There is usually a history of administration. Haematology with blood smear evaluation (which may illustrate RBC Heinz bodies resulting from RBC oxidant injury). Also test copper or zinc levels in fresh liver or kidney.
- Brassica toxicities grazing history.
- Mycoplasma ovis infection parasites in RBCs, seen on evaluation of blood smears.
- Bacillary haemoglobinuria (Clostridium haemolyticum), check clostridial vaccine history. Usually triggered by lesions of liver necrosis.
- Heavy sporidesmin challenge can directly cause acute haemolysis in some cases.
 Diagnosis usually relates to the season and high spore counts, confirmed by testing serum liver biochemistry (raised GGT enzyme).

Pathogenesis of leptospirosis:

Outbreaks of leptospirosis affecting weaned lambs occur after exceptionally heavy summer rainfall, causing accumulation of surface water to which stock have access. Transmission can be direct through infected urine splashing and post-abortion discharges but indirect transmission through contamination of soil and water sources via infected urine, is more likely for grazing lambs.

Leptospires penetrate exposed mucous membranes or through abraded or water softened skin and then disseminate throughout the body. After a brief leptospiraemia the development of opsonising and agglutinating antibodies clears the leptospires from all sites (in subclinical or chronic cases), except those poorly penetrated by antibodies including the renal tubules, CSF, vitreous humor (and, certain serovars, the genital tract). In subclinical cases leptospiral infections are detected only via serology or lesions of interstitial nephritis at slaughter/necropsy.

However, the acute and often severe form of the disease occurs during the leptospiremic phase, particularly in young animals, as observed in the outbreaks of serovar Pomona in grazing lambs this season. In these cases, anaemia is initially due to the production of the haemolysin toxin and later is caused by an antibody mediated reaction against leptospiral antigen-coated erythrocytes. Jaundice may result from both the haemolysis as well as toxic and ischaemic hepatocellular injury.

Chronic disease in older sheep can occur in the post-septicaemic phase in the form of abortion, stillbirth, infertility and interstitial nephritis. Localisation of leptospires in the kidney is associated with focal or diffuse interstitial nephritis and with acute transient tubular degeneration.

Prompt treatment of sick lambs with injections of Streptomycin is reported to be highly effective and good protection is afforded by *Leptospira* vaccination, recommended in high-risk situations (West et al, 2009; Vermunt et al, 1994).

Zoonosis:

Leptospirosis is an important zoonosis and is the most common occupational infectious disease. Most at risk are those working with livestock or in meat processing plants. Also at risk are those working in wet or flooded environments. Infection, as for animals noted above, can be direct via urine splashing or indirect via contaminated water. Anyone working with livestock should ensure cuts/ abrasions are properly covered.

References:

Dreyfus A, Wilson P, Benschop J, Collins-Emerson J, Verdugo C, Heuer C. Seroprevalence and herd-level risk factors for seroprevalence of Leptospira spp, in sheep, beef cattle and deer in New Zealand. *NZVJ*, 66: 302-311, 2018

Vermunt JJ, West DM, Cooke MM, Alley MR, Collins-Emerson J. Observations on three outbreaks of Leptospira interrogans serovar pomona infection in lambs. *NZVJ*, 42:133-136, 1994

West DM, Bruere AN, Ridler AL. The Sheep: Health, Disease and Production. 3rd Edition. *Vetleam Foundation*, Wellington, New Zealand, 2009

Meet our newest sales team member

We are very pleased to welcome Peta Schiessel to our sales team. Peta is the new Territory manager for the Northland, Auckland, Taranaki and Manawatu regions.

Peta has a strong background in animal care, having been a Veterinary Nurse in a mixed practice vet clinic for a number of years before becoming a Pet Nutrition Advisor at Royal Canin ANZ and then a Customer Development Executive within the same

company.

With a passion for learning and people she loves to get out and about meeting clients and finding ways to help them. She is really looking forward to providing great customer care to the clients in her area.

Based in Auckland Peta enjoys spending time with her horse Zephyr and two cats, Nikau and Officer Dibble.



I see red, I see red!

We just love getting customer feedback, especially suggestions for improvement that will benefit many of our clients. The most recent improvement is to molecular test reporting.

You will now find positive, pool positive, detected, weak detect, high positive etc.

results for molecular and BVD milk tests are highlighted in **red** font on reports. This will ensure that positive results are much easier to spot, especially on cases with there are a large number of animals with negative results.

See example report (right).



Hypoadrenocorticism - a diagnostic challenge

REBECCA ALLAN

A recent case through the laboratory highlighted the challenges when diagnosing hypoadrenocorticism and in differentiating the typical from atypical form of the disease.

Hypoadrenocorticism or Addison's disease is an uncommon endocrinopathy that occurs when there is destruction or atrophy of layers of the adrenal cortex. The most common form is when both the zona glomerulosa (the outer layer which makes mineralocorticoids) and zona fasciculata (middle layer which makes glucocorticoids) are affected, leading to glucocorticoid and mineralocorticoid-deficient hypoadrenocorticism.

In about 30-40% of cases only the zona fasciculata is affected, leading to glucocorticoid deficient hypoadrenocorticism. The ensuing glucocorticoid and/or aldosterone deficiencies result in clinical signs that are frequently vague, episodic and nonspecific, including anorexia, vomiting, weight loss and diarrhoea, but can sometimes be dramatic with patients presenting recumbent and in acute circulatory collapse.

Laboratory findings can be similarly nonspecific and attributable to a number of different underlying disease processes. Commonly seen are pre-renal azotemia, hyperkalemia, hyponatremia and lack of a stress leucogram (lymphocyte concentration in the normal range). Other abnormalities may also include hypochloremia, hypercalcaemia, hypoglycaemia, hypoalbuminemia and a mild nonregenerative anaemia.

Clinical history:

The patient in question, a 1-year-old entire female Kelpie dog "Fern" (not her real name) had collapsed and was nonresponsive with haemorrhagic gastroenteritis. Initial in-clinic laboratory results revealed a mild azotaemia, marginal hyponatraemia, marked panhypoproteinaemia and hypocholesterolaemia with an unremarkable CBC and mid-normal range lymphocyte concentration.

Supportive treatment, including intravenous fluids, was initiated and an internal medicine specialist consulted. Atypical Addison's disease was suspected and the veterinarian performed an ACTH stimulation test.

Further laboratory testing:

Resting and 1-hour post ACTH stimulation serum cortisol concentrations were both <4.04 nmol/L (<55 nmol/L at both times consistent with hypoadrenocorticism).

With the absence of typical electrolyte disturbances and marginal hyponatremia attributed to gastrointestinal loss, a presumptive diagnosis of atypical hypoadrenocorticism was made and Fern was started on prednisone therapy. She made an excellent recovery with resolution of the majority of clinical signs over the following two weeks, other than reports of lethargy and tiring easily.

The possibility that electrolyte disturbances not seen at initial presentation were now apparent couldn't be discounted so repeat

bloods were taken, this time revealing hyperkalemia, hyponatremia and hypochloremia, consistent with typical hypoadrenocorticism. At the time of writing, Fern had just started on mineralocorticoid replacement therapy (Florinef).

Discussion:

Gastrointestinal haemorrhage can be a presenting sign in dogs with hypoadrenocorticism as glucocorticoids have an important influence on epithelial integrity and vascular permeability. It is proposed that low concentrations predispose gastrointestinal mucosa to erosion, ulceration and consequent haemorrhage. More common rule outs for GI haemorrhage in a young dog like Fern, might include acute haemorrhagic gastroenteritis, acute pancreatitis, parvovirus, severe hookworm infection, rodenticide toxicity, and ulceration/perforation due a GI foreign body.



While Fern's gastrointestinal signs had an acute onset, it was interesting to read a paper published by Hauck et al, JVIM 2020, that determined a 4% prevalence of hypoadrenocorticism in dogs with signs of chronic gastrointestinal disease compared to 0.3% in the general population. They also established there was no significant difference in history, physical examination and laboratory variables between dogs with hypoadrenocorticism and those with chronic enteropathies and recommended an ACTH stimulation test be part of the standard workup for dogs presenting with gastrointestinal signs.

Some patients initially diagnosed with atypical Addison's disease, go on to develop electrolyte abnormalities during follow up,

leading to the hypothesis that destruction of the adrenal cortex is first limited to the zona fasciculata but spreads to the zona glomerulosa over time. This may have been the explanation for the development of electrolyte abnormalities in Fern's case.

In summary, the diagnosis of hypoadrenocorticism can be tricky to make due to the considerable overlap in clinical signs and laboratory results between this and other conditions, in Fern's case haemorrhagic gastroenteritis. This case also demonstrated the importance of careful monitoring following a presumptive diagnosis of atypical Addison's disease, due to the possibility of progression to typical hypoadrenocorticism and development of electrolyte abnormalities.

Thank you to Beth Campbell from Vetlife Wanaka for this interesting case and follow up information.

References:

Hauck C, Schmitz SS, Burgener IA, et al. Prevalence and characterisation of hypoadrenocorticism in dogs with signs of chronic gastrointestinal disease: a multicenter study. J Vet Intern Med. 34:1399-1405, 2020,

Hatoya S, Kanegi R, Nabetani T, et al. Atypical hypoadrenocorticism with intact zona glomerulosa of the adrenal cortex after long-term observation: a case report of a dog. J Vet Med Sci. 85:9-13, 2023.

We haven't forgotten the cows!

RACHEL WHITEHEAD

We are seeing a continual emergence of drench resistance in cattle. The importance of monitoring the efficiency of drenches in cattle, particularly dairy, is key to halt the production limiting effect of resistant parasite burden.

Faecal egg counting (FEC) alone does have its limits when used for cattle. The host immune response can suppress Ostertagia ovulation, which effects the ability to detect Ostertagia burdens via FEC. Larval culture is a recommended add-on test that will improve the detection and identification of species present, and when interpreted with FEC results and clinical signs, gives a more complete picture of parasite status.

> A significant driver of drench resistance is the continued use of ineffective drench products.

This season Gribbles Veterinary have released the faecal egg count reduction test (FECRT) report for cattle (in addition to the FECRT for sheep released in 2022).

When sending samples to Gribbles Veterinary for FEC and larval culture pre- and post-drench, you will receive a customised, farm-specific report, highlighting any areas of concern. This means that there is no calculation or data entry required by the veterinarian. Our FECRT report is in a format that is farmer-ready, to support on-farm planning.

To receive this free of charge report, samples must arrive with the correct submission form. The bovine FECRT form can be found on our website here.



If you have any questions or require any further information, please contact your local Territory Manager or Gribbles Veterinary laboratory.



Species:

13/02/2023 14:25 Date Sent: Date Received: 13/02/2023 14:25 Date Tested: 13/02/2023 14:28

All client, owner and animal details supplied by the submitter.

Pre-Drench Case No: PN23T0009 / Post-Drench Case No: PN23T0027

Days between samples:

Age:

0 YEAR(S)

The following estimates of the faecal egg count reductions (FECRs) obtained for the various strongylid worm genera represented in this case, are based on the application of the results obtained from pooled larval cultures to their group mean egg counts. In general, pre-treatment counts of 150 epg and 50 epg are required to provide reliable measures of the FECRs of combined genera and individual worm genera, respectively. Where these criteria are met, resistance is indicated by a FECR of <95%. If they are not met, the resistance is classified as NA.

MATRIX	PN23T0009	PN23T0027			
Parasite	Pre- epg (T1 or C2)	Post- epg (T2)	FECR %	Resistance status	Post- LC %
Nematodirus	5	0	100	NA	
Haemonchus	2219	0	100	Susceptible	0
Ostertagia	380	43	89	Resistant	92
Trichostrongylus	317	4	99	Susceptible	8
Cooperia	190	0	100	Susceptible	0
Oesoph/Chabertia	63	0	100	Susceptible	0
Total *	3170	47	99	Susceptible	

False alarm

During January we were advised by MPI of concern about reports of a positive heartworm test in a New Zealand born dog.

The overseas laboratory has confirmed that

the initial result was a false-positive and that the dog is negative for heartworm. This test was part of a routine investigation for export of a dog. This represents very fast work by the investigators and laboratory involved. On behalf of MPI, we would like to reiterate that there is no heartworm in New Zealand, this is a false positive result at the overseas laboratory and has been stood down.

Diarrhoea and gut histo

ROB FAIRLEY

We have received several lamb and calf cases recently with post mortem samples from animals with diarrhoea. Here are some recommendations to ensure you get the most out of your cases, especially when sacrificing an animal for histological sampling of the gut.

1. Immerse your samples of gut into formalin as soon as possible - within minutes,

- especially in a sacrificed animal. Make sure the formalin bathes the mucosa. Long tubes of gut without contact with formalin will keep autolysing. We want the autolysis process to be arrested immediately.
- Sample throughout the GI tract take large intestine, small intestine, abomasum, and forestomach.
- For abomasum take your samples from the body of the stomach, not the pylorus. We can't assess for metaplastic change due to

- parasites in the abomasum if the sample is from the pylorus.
- 4. Take multiple samples of small or large intestine. Lesions of some diseases can be multifocal and a single piece of small intestine may miss lesions of yersiniosis, for example. How do we know? We often see cases of yersiniosis where multiple samples of small or large intestine have been taken and some of those samples do not have characteristic lesions.

In brief

- > **Need an up-to-date copy of our price list?** We have recently made a couple of corrections and updates to our current price book. <u>Download an updated copy here.</u>
- > **Our facial eczema Lab-Portal** is open for business any time you do pasture spore counts. <u>Login or register here</u> or view real-time facial eczema trends <u>here</u> (no user account required).
- > **Serum electrophoresis testing** will now be carried out on Tuesdays (instead of Fridays). Results will be available on Tuesday afternoons.









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