

Recognising and diagnosing leptospirosis in lambs

By Alice Fraser

Our wet summer provided prime conditions for *Leptospira* to thrive – here's what you need to know.

A warm, wet summer brings an abundance of feed for livestock grazing and preserving, but it also brings livestock disease challenges. For sheep farmers, the wet conditions this summer caused increased fly strike, early bouts of *Haemonchus* infestation and the threat of an upcoming high sporidesmin challenge, to name a few. In addition, during this wet summer season Gribbles Veterinary saw a significant increase in the incidence of severe acute leptospirosis in young ruminants, particularly in weaned lambs, which was likely related to the season's heavy rainfall.



Background

Leptospirosis is a globally important disease that can affect all mammalian species, including humans, and is 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, not only do leptospires survive in wet soils but researchers have found they multiply in waterlogged soils (Yanagihara et al., 2022), leading to an increased contamination of surface water with leptospires following heavy rainfall and flooding.

There are multiple pathogenic 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 and wildlife are considered to be the maintenance hosts of serovar Pomona, but outbreaks in lambs often have no apparent association with pigs. The environmental multiplication of the pathogen under the aforementioned conditions is a likely source.

Other young ruminants, including calves and fawns, can similarly be affected by acute severe leptospirosis. With the greater degree of routine *Leptospira* vaccinations in the dairy industry, we tend not to see such a stark increase in the incidence of acute leptospirosis in calves in wet summers. Dogs, particularly rural/farm dogs, are also susceptible to severe acute leptospirosis (Thompson, 2018).

Leptospira borgpetersenii serovar Hardjo, the most common serovar in cattle and deer (and host-adapted in these species), showed a higher seroprevalence in sheep than serovar Pomona in a New Zealand epidemiological study (Dreyfus et al., 2018). However, serovar Hardjo in sheep is more often subclinical and not associated with the acute severe clinical disease observed in lambs infected by serovar Pomona, on which this article focuses.

Serovar Pomona outbreaks in lambs

In a wet summer, particularly following heavy rains, a typical incident involves sudden deaths and malaise in several lambs in a mob; sometimes

10–20 lambs are found dead in a mob, often on hill-country farms/stations where there has been surface water or flooding. On necropsy, an affected lamb carcass, usually in good body condition, typically shows marked jaundice of the omental fat (unless it is a peracute case that may just be anaemic), generalised carcass pallor elsewhere, yellow/brown liver, dark urine and dark, swollen kidneys.

Histopathology

Necropsy samples for *Leptospira* diagnosis

Fresh kidney should be used for *Leptospira* polymerase chain reaction (PCR) (occasionally false negatives arise, particularly in peracute cases). This is a generic *Leptospira* test and does not differentiate the serovars. Also, collect fresh liver if copper/zinc toxicities need to be investigated. The key fixed samples for histopathology relating to leptospirosis are kidney and liver.

Microscopic changes in a typical acute case provide evidence of an acute haemolytic crisis (strongly supportive of the clinical suspicions of leptospirosis). Findings include centrilobular hepatocellular necrosis in the liver (figure 1) and haemoglobinuric nephrosis in the kidney (as a result of an intravascular haemolysis, figure 2). On occasion, in very well-preserved renal tissue samples, leptospirae are visible with silver stains (although as a diagnostic test for leptospirosis, this is of low sensitivity owing to often sparse pathogen numbers visible in renal tissue, particularly in acute cases, and to any tissue autolysis).

To allow for differential diagnoses, it is of course always recommended that all tissues be examined, including by opening the abomasum (examine for *Haemonchus* in particular). Open the rumen to note the contents and its lining and open the small intestines. A range of other fixed-tissue samples (as well as liver and kidney) can be harvested, including samples of the spleen, lung and gastrointestinal tract, to investigate differential diagnoses.

Sick lambs

Collect urine for *Leptospira* PCR (false negatives can occur in urine as the leptospirae are intermittently shed). Take EDTA blood samples for haematology (including blood smear analysis); a severe haemolytic anaemia is observed with acute leptospirosis (see below for differential diagnoses). Take a serum sample for the first of two serology tests for *Leptospira* serovar Pomona (collect the second serum sample one to two weeks later to show a four-fold increase in antibody titres).

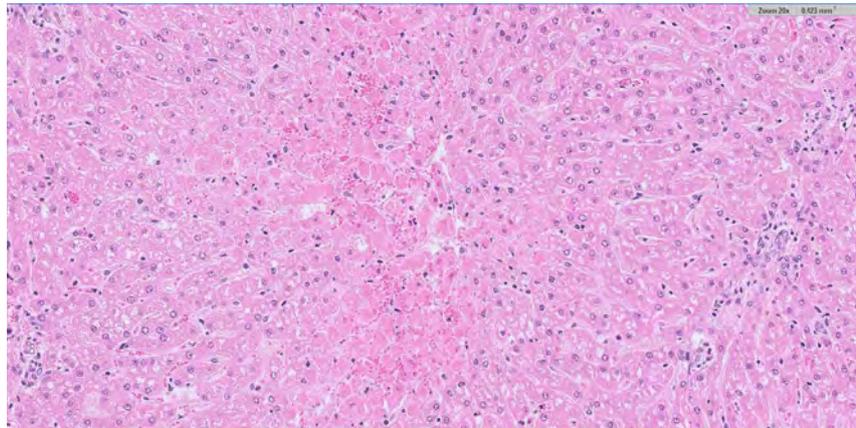


FIGURE 1: Liver, centrilobular degeneration,

Serology

Leptospira serovar Pomona titres increase markedly once seroconversion occurs. Initial serum samples, if collected early/in the acute phase of the disease, may show no evidence of seroconversion or low titres. Follow-up samples after 7–14 days in affected or in-contact lambs are required to show seroconversion, often revealing very high titres. *Leptospira* serovar Hardjo: lambs often show low background titres (probably maternal source).

Differential diagnoses

Other causes of haemolytic anaemia in sheep include:

- copper, zinc toxicity: there is usually a history of administration. Haematology with blood smear evaluation is required (Heinz bodies on red blood cells [RBCs] are seen as a result of RBC oxidant injury). Also, test copper or zinc levels in fresh liver or kidney
- brassica crop toxicities: these relate to grazing history. Again, an evaluation of a blood smear usually reveals Heinz bodies on RBCs. Liver and kidney histopathology is also useful
- *Mycoplasma ovis* infection: this relates to haemoparasites in RBCs, seen on evaluations of (fresh) blood smears. It predominantly causes an extravascular haemolytic anaemia (so haemoglobinuria is less commonly seen). If there is any delay in making a blood smear (causing the RBC parasites to ‘fall off’), a PCR test on EDTA blood is available. The distribution of *M. ovis* in New Zealand is not known (Hulme-Moir, 2018). Reports of effects are variable, from subclinical cases to anaemia and ill-thrift or outbreaks of anaemia and deaths in lambs

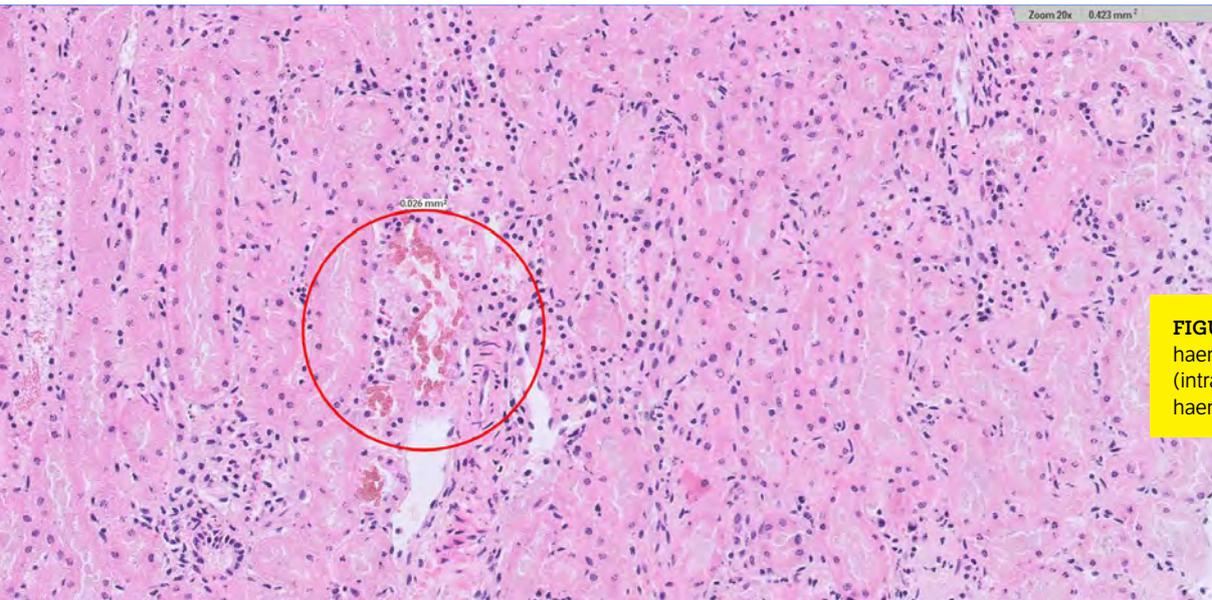


FIGURE 2: Kidney, haemoglobinuria (intratubular haemoglobin casts).

- bacillary haemoglobinuria (*Clostridium haemolyticum*, check clostridial vaccine history). This is usually triggered by lesions of liver necrosis
- a very heavy sporidesmin spore challenge, which can directly cause acute haemolysis in some cases. Diagnosis usually relates to the facial eczema season with high paddock spore counts, supported by testing serum liver biochemistry in several affected lambs in a mob (raised gamma-glutamyl transferase enzyme).

Pathogenesis of the leptospirosis outbreak

As previously mentioned, outbreaks of leptospirosis affecting weaned lambs (and other young ruminants, including calves and fawns) often occur after exceptionally heavy summer rainfall, causing an 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 the contamination of soil and water sources via infected urine is often the case for more extensively farmed stock.

Leptospire penetrate exposed mucous membranes or enter 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 leptospire from all sites (in subclinical or chronic cases), except those poorly penetrated by antibodies, including the renal tubules, cerebrospinal fluid and vitreous humour (and, for some serovars, the genital tract). In subclinical cases, leptospiral infections are detected only via serology or lesions of interstitial nephritis at slaughter/necropsy.

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 bacterial haemolysin toxin and later is caused by an antibody-mediated reaction against leptospiral antigen-coated erythrocytes. Jaundice may result from both the haemolysis and 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. The localisation of leptospire 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, which is recommended in high-risk situations (West et al., 2009; Vermunt et al., 1994). However, routine *Leptospira* vaccination in sheep flocks may not be cost effective from the farm business perspective.

Zoonosis

Leptospirosis is an important zoonosis and is reported by the New Zealand health system to be the most common infectious occupational disease. Most at risk are those working with livestock and in meat-processing plants. Also at risk are those working in wet and 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.

The disease in people can appear in its early stages to be very similar to an acute viral infection, such as influenza, but prompt antibiotic treatment for leptospirosis is effective and is key to the prevention of a progression to severe or chronic disease; ongoing awareness of the disease by general practitioners is therefore of great importance. Collaborative initiatives involving the veterinary, farming and medical industries have been in motion for a number of years to reduce the incidence of leptospirosis cases in the workplace. The introduction of 'Leptosure' – a programme developed by the NZVA's Society of Dairy Cattle Veterinarians special interest branch as a national risk-management programme, with steps to reduce the incidence in the workforce – has seen an increase in *Leptospira* vaccination programmes within the dairy industry. However, vaccination rates for other livestock classes, including sheep, beef cattle and deer, are much lower owing to debate on the economic advantages of a regular vaccination programme for these livestock industries (apart from during high-risk seasons). ⁵⁹

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Indicators of exotic disease in poultry



Images courtesy of the USDA-APHIS Foreign Animal Disease Diagnostic Laboratory at the Plum Island Animal Disease Centre.

Exotic diseases in poultry should be considered when signs include:

- Sudden, unexplained deaths
- Rapid spread of disease throughout the flock
- An unexpected drop in egg production
- Unusual signs/combination of unusual signs such as:
 - Nervous signs
 - Respiratory signs
 - Facial swelling, cyanosis
 - Depression, loss of appetite
 - Diarrhoea

Report an exotic pest or disease



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