

OVERVIEW

- **Ragwort (*Senecio jacobaea*) toxicity in suckler cows at grass**
- **Abomasal emptying defect in a Charolais ewe**
- **Listerial meningoencephalitis in both cattle and sheep**

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in October 2024. Given similar climatic and production conditions, they could also be important this year.

- **Syngamus trachea infection in pheasant poults**
Birds are at risk once they have access to outdoors and particularly following the move to release pens. Infection occurs via ingestion of larvated eggs, hatched larvae or larvae within earthworms. The eggs can survive in the soil for nine months and as L3s within earthworms for many years. Wild birds such as corvids or blackbirds can also act as a source of infection.
- **Klebsiella pneumoniae septicaemia in piglets**
Klebsiella pneumoniae causes acute death in piglets pre-weaning with postmortem examination findings consistent with bacterial septicaemia. Outbreaks of disease are most common in outdoor breeding herds between May and September. The bacterium can be

GENERAL INTRODUCTION

Sunshine and rainfall figures for July were comparable to the thirty-year average being 107 and 99 per cent respectively. Consistent with 2025 so far, the mean temperature was increased being 1.5°C above the 1991 to 2020 average.

CATTLE

Toxic conditions

Three cows from a group of 44 were found dead over the course of three weeks and a fourth presented with neurological signs. The animals were at grass, and calves at foot were unaffected. The carcass of a six-year-old Limousin cross cow was submitted after being found dead in the river. This history was consistent with a previous submission from the farm in September 2024, where ragwort toxicity was diagnosed. Postmortem examination identified an enlarged, pale, firm liver with a mottled appearance to the cut surface and a markedly distended gall bladder (Fig 1). Histopathology revealed very severe chronic liver pathology, characterised by fibrosis, biliary proliferation, loss of hepatic tissue, and hepatocyte enlargement supporting a diagnosis of pyrrolizidine alkaloid (ragwort) toxicity. Due to the challenging weather conditions causing the grass to bolt and seed, the field had been topped. Combined with an overall shortage of grass, it is likely that the cows ingested an excessive amount of the more palatable dried ragwort, precipitating the losses.

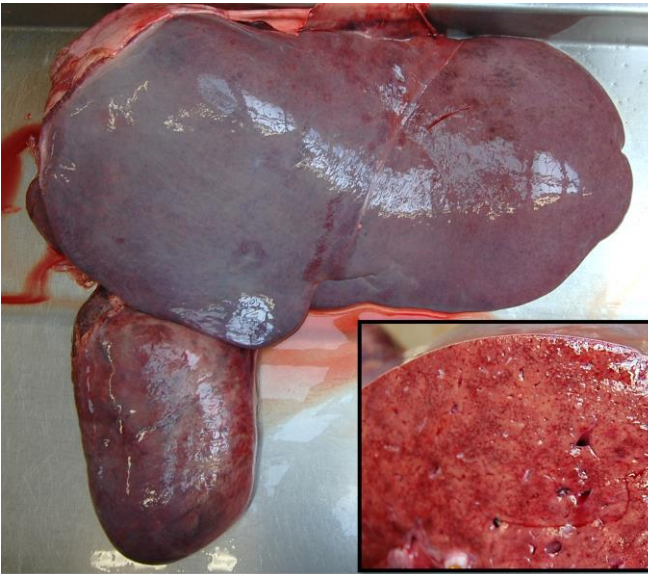


Figure 1 – Enlarged, pale, firm liver with a lobular pattern on the cut surface and markedly distended gall bladder due to ragwort poisoning.

A 400-head dairy herd found 16 milking cows dead at grass close to a water trough. The field had been strip grazed for three days, and no signs of illness had been observed. The weather was hot and due to concerns over slow trough refilling, extra water had been supplied in plastic tanks earlier that day. Screening for anthrax was negative, and the carcass of a Friesian cow was submitted 36-hours later to investigate the losses. There were no significant findings on postmortem examination, rumen pH was 6 and vitreous humour calcium and magnesium results were within normal limits. It was reported that one of the tanks had previously contained a concentrated solution of urea and sulphur used to treat grain. It had not been fully drained before being filled with water raising the possibility of urea toxicity. Vitreous humour urea was 7.1 mmol/l which is within the serum reference range (4 – 8 mmol/l) but consistent with results from previous urea toxicity cases. Brain histopathology was carried out, but the pathology associated with urea toxicity can be very subtle and interpretation was hampered by autolysis. A diagnosis of urea toxicity was recorded based on the history and exclusion of other causes.

Parasitic diseases

A three-year-old Luing cow was euthanased after losing condition and becoming unwell. The carcass was poorly muscled with no body fat and several other cows in the group were reported to be ill thriven. Haemorrhagic fluid was found within the abomasum and the mucosa appeared thickened and red. Histopathology described severe abomasitis characterised by marked inflammation and mucosal fibrosis plus loss of glands and acid producing parietal cells. Nematode larvae were observed embedded within the mucosa and abomasal glands. The findings were consistent with severe, chronic abomasal parasitism, most likely due to ostertagiosis, and fully explained the loss of body condition. Despite the pathology no strongyle eggs were detected in the faeces. It is not uncommon for faecal egg counts from adult cattle to return negative or very low egg count results. In cases where abomasal parasitism is still suspected plasma pepsinogen can be a useful follow-up test to confirm the diagnosis.

Alimentary tract disorders

A four-month-old Friesian cross heifer was submitted for postmortem examination after being euthanased on farm due to ill thrift, diarrhoea and abdominal pain. The group of 150 calves had been turned out at eight-weeks of age and were receiving 1.5kg concentrates/head/day. A trace element bolus had been given at turn out and they were on a paddock grazing system moving every three weeks. Twenty per cent were thinner than expected and had been treated for coccidiosis. A pour-on anthelmintic had been administered to the whole group three weeks earlier. Rumen fill was good however the rumen papillae were not well developed. There was a small number of 2–3 mm diameter circular lesions in the abomasal mucosa suspected to be early ulcers and the mesenteric lymph nodes were prominent. The mucosa of the mid to distal jejunum and particularly the ileum was reddened. There were no significant findings on bacteriology

however histopathology identified superficial ulceration and inflammation associated with colonies of bacteria in the small and large intestine consistent with acute yersiniosis. A moderate increase in overall cellularity of the small and large intestinal mucosa suggested possible chronic dysbiosis which may have predisposed to *Yersinia* sp infection.

Reproductive tract conditions

The carcass of a Holstein Friesian cow was submitted for postmortem examination after it became the fifth cow to present with blood clots in the milk since turn out at the end of April. The herd history indicated that bleeding began in one quarter progressing to three or four quarters over 24 hours. Affected cows continued to eat with no other clinical signs apart from a slight reduction in milk yield. Two cases resolved on their own and two were culled due to continuing haemorrhage. Blood was detected in the milk from the back right quarter of the fifth case the day before it was found dead. No pattern was found with regards to the age/breed, stage of lactation or field and there was no evidence of haemorrhage at other sites. The carcass was generally pale with a thick layer of yellow subcutaneous oedema ventrally, particularly on the left. Haemorrhage was evident mainly overlying the left udder, and on incision a large quantity of clotted blood was present within the left udder (Fig 2). A small area of haemorrhage was found within the right side. Bacteriology produced a growth of *Streptococcus uberis* and *Streptococcus parauberis*, mixed with coliforms. No evidence of mastitis had been noted prior to death nor on histopathology which did not identify any underlying pathology or inflammation that could explain the haemorrhage. Since coagulopathies, mastitis and issues with blood vessel walls had been ruled out, discussions focussed on other possible aetiologies such as trauma and milking machine function.



Figure 2 – Large quantity of blood in the left udder. No cause was found.

Nervous system disorders

A housed, four-month-old Holstein Friesian calf presented with neurological signs and two similarly affected calves were reported in different groups at grass. Pre mortem haematology detected a marked leucocytosis ($59.63 \times 10^9/l$, reference range $4.0-12.0 \times 10^9/l$) due to a marked neutrophilia ($50.1 \times 10^9/l$, reference range $0.6-4.12 \times 10^9/l$) and monocytosis ($7.75 \times 10^9/l$, reference range $0.025-0.84 \times 10^9/l$). Postmortem examination was unremarkable, and bacteriology did not detect any significant isolates. Neuropathology revealed a severe, multifocal to coalescing, non-suppurative meningoencephalitis with microabscesses consistent with a diagnosis of listeriosis. Diagnoses of listerial encephalitis in both cattle and sheep usually peak in spring most likely associated with supplementary feeding. It is less commonly diagnosed in cattle, and the disease tends to be slower in progression.

SMALL RUMINANTS

Alimentary tract disorders

A flock of 30 charollais sheep reported that in previous years three ewes had lost condition and developed abdominal distension with death occurring three to four-weeks later in spite of nursing and symptomatic treatment. The carcase of a three-year-old ewe with a typical history was presented to investigate the issue. An abdominal girth of 170cm was a result of the accumulation of 21.8 kg of malodorous, dry, fibrous content within the abomasum (Fig 3). There were scattered irregular areas of mucosal ulceration, marked dilation of the omaso-abomasal orifice and moderate distension of the forestomachs. There was no evidence of an obstruction, and no foreign material was identified. Histopathology of the abomasal wall showed a reduction in the number of submucosal and myenteric ganglia. Where present they appeared shrunken with increased cytoplasmic basophilia and karyomegaly suggestive of neuronal degeneration. The findings were consistent with a diagnosis of abomasal emptying defect which is believed to be a toxin-related, acquired dysautonomia. A genetic predisposition is likely with most reports involving Suffolk sheep.² The condition has also been reported in the Hampshire, Dorset, and Texel breeds but no references to charollais sheep were found.



Figure 3 – Mild distention of the forestomachs (left) and massive distention of the abomasum (right) due to abomasal emptying defect in a charollais ewe.

The carcases of two, three-month-old Texel cross lambs were submitted following the death of 11 lambs in seven days from a group of 300. A cobalt drench had been administered two weeks before and a doramectin injection the week prior to that. The group had recently been dipped and moved to a reseeded field. Postmortem examination of lamb 1 revealed severe cellulitis with extensive tissue necrosis in the upper neck. The carcase of lamb 2 was pale with some blood in the mouth and large clots within the forestomachs. There was tissue necrosis in the caudal pharynx with a distinct circular perforation through the wall. The findings in both lambs were consistent with a dosing gun injury. Strongyle egg counts of 4450 and 10850 eggs per gram suggested either possible treatment failure and/or massive reinfection from pasture. Further investigation of anthelmintic efficacy was recommended.

Nervous system disorders

A small herd of 40 mixed age goats running on rough ground reported neurological signs in a castrated male. Treatment with antibiotics and corticosteroids was attempted but it deteriorated and was euthanased the following day. The herd came inside each day in order for 12 animals to be milked. Two lead batteries were known to be present in the field, and it was noted that poor quality food had been fed for a 24-hour period after the concentrates ran out. A second goat became pyrexemic and continued to deteriorate despite treatment with B vitamins, antibiotics and corticosteroids. It was euthanased for postmortem examination. No ticks were found on the carcase and there was no evidence of cerebellar coning or fluorescence under ultraviolet light. A pure growth of *Listeria monocytogenes* was cultured from a brain swab confirming listerial meningoencephalitis as the cause of the problem.

A five-month-old, housed Valais blacknose ram lamb was found dead and submitted for

postmortem examination along with a fixed brain from an 18-month-old ram. The latter had a history of recumbency and paddling prior to death and was reported to be the second animal from a group at grass to exhibit those clinical signs. Listerial meningoencephalitis was confirmed on histopathology in both cases, and a pure growth of *Listeria monocytogenes* was isolated from the spleen of the lamb.

A flock of 1100 had lost 44 ewes since lambing time at a rate of one to two per week. A three-year-old mule ewe was found dead and submitted for investigation however the carcass was very autolysed. Pleural adhesions were found between the right lung and the thoracic wall with three to four abscesses up to 1cm in diameter present in each lung. Rumen fill was poor with watery content but there was no evidence of diarrhoea. The brain was very autolysed however a pituitary abscess was identified. Bacteriology produced mixed growths with no significant organisms isolated. This was likely to be a one-off case and did not explain the ongoing deaths. Further investigation was advised.

BIRDS

The carcasses of three adult pheasants were submitted to investigate increasing mortality in a flock of 200 ex-laying females. The birds had been housed prior to being introduced to a release pen one month earlier. Affected birds were anorexic with fluffed up plumage prior to death. Postmortem examination revealed coelomitis and salpingitis (egg peritonitis) in two while the third had renomegaly plus white crystalline material on the hepatic capsule and pericardium, consistent with visceral gout (Fig 3). Histopathology confirmed severe nephritis in all cases and PCR testing of kidney detected infectious bronchitis (IB) virus in two. Nephritis due to gamma-coronaviruses (including IB virus) occurs sporadically in adult pheasants particularly associated with stress. Visceral gout and egg peritonitis can also be stress related.

Advice provided included minimising stress, provision of appropriate diet, and ensuring clean palatable water was always available in the release pen.

MISCELLANEOUS

Twenty per cent neonatal calf mortality was reported in a group of 93 red deer hinds. The carcasses of three one-week-old calves were submitted all of which had evidence of diarrhoea. No milk was present in the abomasa and mucosal ulceration was a common feature. Unfortunately, samples of blood collected at postmortem examination were too haemolysed to allow ZST testing. Analysis of vitreous humour in one case revealed significant uraemia (urea 102.7 mmol/l; serum reference range 4 – 8 mmol/l). Screening for enteric pathogens detected cryptosporidia in all cases however histopathology did not support this as the cause of death. The presence of fibrinous alveolar oedema was suggestive of septicaemia and it was hypothesised that inadequate transfer of maternal colostral antibodies, perhaps due to maternal desertion had predisposed to septicaemia and death.

References:

- 1 Bidewell CA, Williamson SM, Rogers J. *et al.* Emergence of *Klebsiella pneumoniae* subspecies *pneumoniae* as a cause of septicaemia in pigs in England. *PLoS One* 2018;13(2): e0191958
- 2 Pruden SJ, McAllister MM, Schultheiss PC, O'Toole D. Abomasal emptying defect of sheep may be an acquired form of dysautonomia. *Vet Path* 2004; 41(2): 164–9