

SRUC Veterinary Services

Monthly Report for January 2025



OVERVIEW

- Hypomagnesaemia in a well grown three-month-old suckled calf
- Abortion outbreak due to bovine herpesvirus 1 in a pedigree beef herd
- Diarrhoea and deaths due to acute rumen fluke in ewes
- Ovine foetal deformities as a result of in utero infection with Schmallenberg virus

GENERAL INTRODUCTION

The mean temperature for January was 2.3°C which is 0.6 degrees below the 1991 to 2020 average. It was a dry sunny month with 51 per cent of average rainfall and 140 per cent of average sunshine. Wind speeds of up to 100 miles per hour were recorded in southern Scotland during storm Eowyn on 24th January.

DISEASE ALERTS

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

Abortion due to *Aspergillus fumigatus*

Exposure to mouldy or spoiled forage/bedding increases the overall abortion risk in cattle due to a range of pathogens including *Aspergillus fumigatus*. Following experimental infection lesions in the cotyledons can be detected after 7 days with abortion occurring at around 4 weeks post-inoculation.¹ The intercotyledonary placenta can appear thickened and leathery and the cotyledons necrotic. Irregular lesions are occasionally present on the foetal skin. Isolation of a pure growth of *A. fumigatus* from the foetal stomach contents confirms the diagnosis.

DISEASE ALERTS

Congenital swayback

Swayback is associated with low dam copper status in mid-gestation. Outbreaks can be more common following mild winters when less supplementary feed is provided. The severity of clinical signs varies and some lambs may be stillborn. Affected lambs are ataxic or unable to stand and occasionally have a fine head tremor. Liver copper concentrations in affected lambs are usually well below the reference range, but can be higher if the dam has received copper supplementation in late pregnancy. For this reason the brain should always be removed and formalin fixed in case histopathology is required to confirm the diagnosis.

CATTLE

Nutritional and metabolic disorders

A well grown three-month-old Limousin heifer calf weighing 140 kg was found dead. It was submitted for investigation as the herd had a history of sporadic sudden deaths in healthy calves and low selenium status had previously been diagnosed. Postmortem examination found haemorrhages on the thymus and around the heart base with moderate numbers of tiny blood clots within the tracheal lumen. A small amount of fibrin and oedema was present around the brainstem. The abomasum contained a good milk clot confirming it to be a true sudden death. Liver selenium content was below the reference range at 0.51 mg/kg dry matter (DM) (reference range 0.9 – 1.75 mg/kg DM) but histopathology did not detect any evidence of nutritional myopathy, or lesions indicative of clostridial disease. It confirmed multifocal endocardial haemorrhage plus haemorrhage within the adventitia of the aorta. Reactive change in the overlying

mesothelium suggested this had been present for a short period prior to death. A rib Ca:Mg ratio of 102 (reference range 30 – 70) was consistent with a diagnosis of hypomagnesaemia. Mineralised creep feed was available to the calves, and it was assumed that this animal had not been eating enough to meet its magnesium requirement.

Alimentary tract disorders

A one-week-old Holstein heifer became the fourth calf to die over a two-month period was submitted for further investigation. It had been off colour for 48-hours prior to death and was described as dehydrated with a “fluidy” abdomen. Diarrhoea was not a feature, and it had been treated with antibiotics, NSAIDs and oral electrolytes. Postmortem examination identified a fibrinous peritonitis centred on the caecum. The mid-jejunum was adhered to the caecum obstructing the flow of ingesta resulting in fluid distension of the proximal small intestine and abomasum. The serosa of the blind end of the caecum was coated in a layer of fibrin and there were irregular patches of mucosal necrosis in this area. Differential diagnoses included ischaemia or bacterial typhlitis due to *Salmonella* spp, *Listeria* spp or *Clostridium sordellii*. There were no significant findings on bacteriology. Histopathology confirmed that the caecal mucosa had been replaced with necrotic debris mixed with large numbers of neutrophils and colonies of coccobacilli. Necrosis and inflammation extended through the submucosa to the serosa resulting in perforation and secondary peritonitis. The lesions were considered to be most consistent with salmonellosis. Prominent thrombosis was absent but could have been masked by the severity of the pathology. It was suggested that more information on the herd’s salmonella status could be obtained by carrying out *Salmonella enterica* serotype Dublin serology on youngstock and bulk tank milk.

Respiratory tract diseases

A dairy herd with a two-month history of poor calf health euthanased a 20-day-old Holstein heifer to investigate the problem as all but one calf in the shed had developed diarrhoea between the ages of four and 18 days. None had died but affected calves took at least two weeks to recover. The herd was closed with a negative BVD status and prophylactic treatment with paromomycin had been tried without success. Calves were routinely stomach tubed with 4 litres of dam colostrum after birth and then received five feeds of colostrum via a teat before moving on to milk replacer fed from a bucket. The submitted calf was a typical case that had been diarrhoeic with a poor appetite for one week. Postmortem examination found thymic atrophy and minor bilateral areas of lung consolidation. A small amount of straw and concentrate feed was found in the rumen, the abomasal and small intestinal contents were fluid and the mesenteric lymph nodes were slightly enlarged. The faeces were diarrhoeic and cryptosporidial oocysts were detected. No other enteric pathogens were identified however PCR testing of lung produced a strong positive result for bovine respiratory syncytial virus (RSV) with a CT value of 10.49. Histopathology confirmed cryptosporidiosis to be the cause of the diarrhoea with evidence of repair in the jejunum and ongoing mucosal damage in the ileum. The lung changes appeared minor and not typical of RSV however, when compared to older calves this may reflect a contrasting immune response in neonatal calves.² There was a slight hypoalbuminaemia of 28.9 g/l (reference range 30 – 40 g/l) explainable by the diarrhoea. However, the globulin result of 15 g/l (reference range 30 – 50 g/l) was disproportionately low. Respiratory virus infection in horses is known to cause low globulin levels, and despite the minimal lung pathology RSV infection was considered significant in relation to the ongoing calf health issues. Trial intranasal RSV vaccination was initiated.

A nine-month-old Limousin cross calf was presented to investigate the cause of a respiratory disease outbreak in a group of 80 weaned calves. Three animals had died within 48-hours, and a further three were ill. The submitted calf had been treated with tulathromycin and meloxicam prior to death. Postmortem examination found a marked pleuritis and consolidation of the cranial and accessory lung lobes with interlobular fibrinous exudate (Figure 1). The pericardium was thickened and oedematous with a mild fibrinous pericarditis. Screening for respiratory pathogens by PCR detected *Histophilus somni* (CT 13.45) together with a low level of parainfluenza-3 virus (CT 31.09), which may have predisposed to the bacterial infection. Bacterial cultures of lung and liver remained sterile perhaps due to the fastidious nature of *H. somni*, or premortem antibiosis. *H. somni* is also capable of acting as a primary pathogen, causing both pneumonia and/or septicaemia. As a result the clinical presentation can be variable and may include sudden death, recumbency, pyrexia, respiratory signs, neurological signs or lameness.

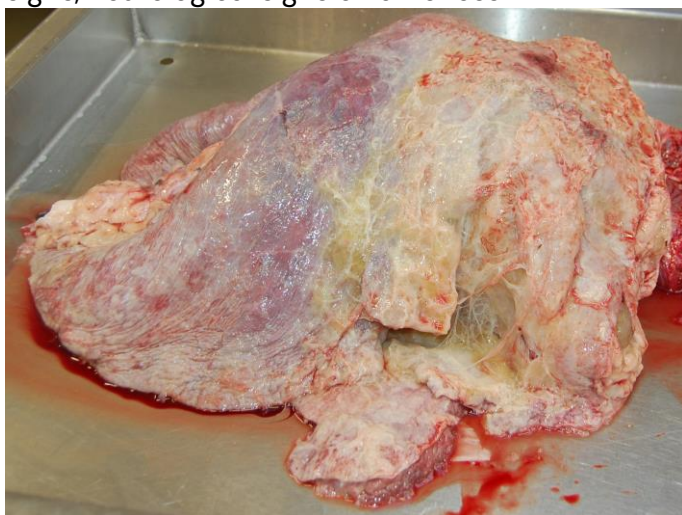


Figure 1 – Fatal *Histophilus somni* pneumonia in a Limousin cross stirk

Reproductive tract conditions

A pedigree beef herd with 20 Aberdeen Angus cows experienced a series of four abortions

between 2 and 8-months of gestation. The herd used natural service, bred its own replacements and was BVD vaccinated. Two fetuses were examined with no evidence of infectious disease on initial investigation. Histopathology revealed multifocal necrotising hepatitis in both plus necrotising placentitis with vasculitis in one. Subsequent PCR testing of stored foetal liver proved positive for bovine herpesvirus 1 (BoHV-1) with CT values of 17.46 and 11.2 confirming a diagnosis of foetopathy due to BoHV-1. This is an infrequently diagnosed cause of bovine abortion in Scotland. Screening for BoHV-1 is not part of the routine bovine abortion diagnostic package and this case highlights the need to collect a range of fresh and fixed tissues in order to thoroughly investigate abortion outbreaks without a diagnosis. The source of the virus was not known.

Nervous system disorders

Four Aberdeen Angus cross dairy calves presented with nystagmus, head pressing and opisthotonus. Veterinary assistance was sought for the third and fourth cases which were treated with vitamin B12, corticosteroids and antibiotics. One died later the same day, and the other failed to improve and was euthanased 24-hours after the onset of clinical signs. Both carcasses were submitted for postmortem examination which detected an area of abomasal ulceration in the first and localised lung microabscessation in the second. These were considered incidental findings in relation to the cause of death. Rumen pH results of 6.4 and 6.1 ruled out acidosis as a contributing factor. Cerebrocortical necrosis (CCN) was suspected, both brains fluoresced under ultraviolet light and histopathology findings of laminar polioencephalomalacia confirmed the diagnosis. The group consisted of four to five-month-old calves on a diet of calf nuts and hay. No obvious predisposing factors for CCN were apparent in the history suggesting that a more detailed investigation was required to identify any underlying issues.

SMALL RUMINANTS

Parasitic diseases

A flock lost six ewes from a group of 30 over the course of one week. Two were found dead and the others were reported to be quiet with poor appetites and rapid weight loss in the three to four days prior to death. Treatment with triclabendazole/ivermectin and oxytetracycline did not halt the losses and deaths continued after the group were moved to a different field. The carcase of a three-year-old Suffolk ewe was submitted and was thin and dehydrated. Large deposits of internal fat remained and there was no evidence of pneumonia or liver fluke associated pathology. Rumen fill was adequate, but the contents were abnormally wet and frothy. A few small adult rumen fluke were noted. The intestines contained profuse liquid content and uncountable numbers of juvenile rumen fluke (*Calicophoron daubneyi*) were found in the proximal small intestine (Fig 2). Treatment with oxclozanide was advised with follow up monitoring of faeces for liver and rumen fluke eggs in late spring/early summer to inform if further treatment is required to reduce pasture challenge in autumn.



Figure 2 – Immature *C daubneyi* recovered from the small intestine of a store lamb in a previous case of fatal acute rumen fluke

Respiratory tract diseases

Four thin mule ewes were submitted from a lowland flock of around 1000 bought in ewes. They had been selected for culling pre-tupping but failed to gain body condition prior to sale. Postmortem examination found varying degrees of broken mouth plus a range of lung pathology in all four sheep. Ovine pulmonary adenocarcinoma (OPA) was suspected in two however the pathogenesis of the other lesions was not clear (Figure 3 and 4). Histopathology described lesions of OPA in one with changes indicative of maedi visna (MV) in all four including increased alveolar macrophages with marked smooth muscle hyperplasia and increased lymphocytes in the alveolar septa. Positive MV serology confirmed the diagnosis. Lung lesions in sheep can be difficult to assess and additional testing, particularly histopathology, is essential in confirming a diagnosis particularly where there may be far reaching implications for flock management. The possibility of underlying infection with MV should be considered in all cases of OPA and when investigating issues with respiratory disease in adult sheep.

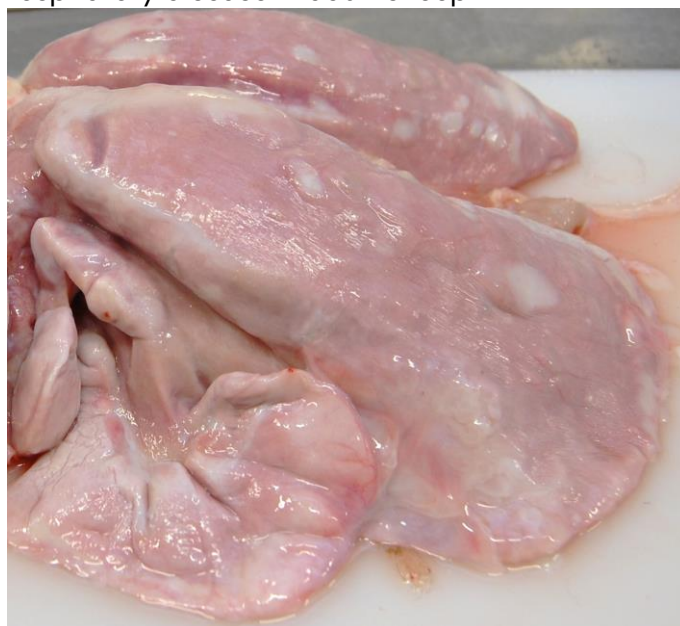


Figure 3

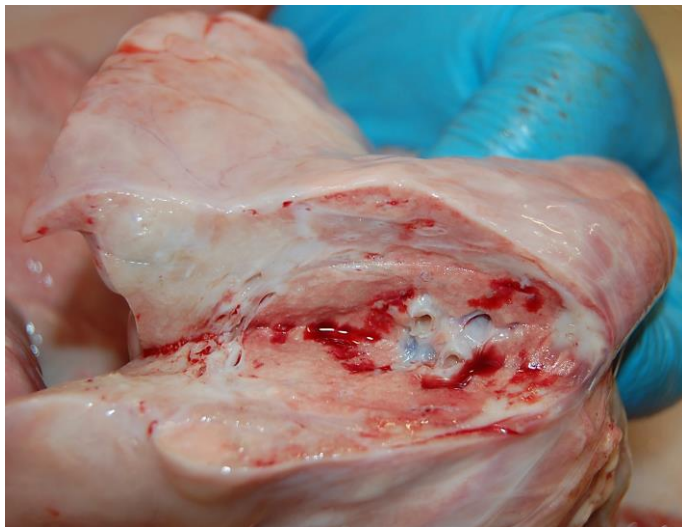


Figure 4

Figure 3 and 4 – Non-diagnostic lung lesions in a ewe with maedi visna

Reproductive tract conditions

Two sets of deformed twins were born from a group of 16 Dutch spotted Texel ewes lambing in early January. Both dams were homebred, one was a gimmer and the other a ewe. One set was submitted for investigation and found to have arthrogryposis of all four limbs plus kyphosis and scoliosis of the cervical and thoracic spine. Brain and spinal cord abnormalities were not observed. PCR testing of brainstem proved positive for Schmallenberg virus confirming midge-borne infection during the second month of gestation which equated to September in this case.

Skin diseases

A group of 40 north country Cheviot gimmers were treated for suspected contagious ovine digital dermatitis after several developed foot lesions and became lame. They responded to treatment, however one animal was found in lateral recumbency. A single ulcerative lesion was present on the heel of its right hind foot. It was reluctant to eat and was euthanased a few days later. Postmortem examination detected extensive sloughing of the dorsal lingual mucosa and erosion of the oesophageal mucosa particularly distally. Histopathology confirmed

multifocal areas of ulceration with a fibrovascular response in the underlying tissue. Differential diagnoses included orf, border disease, uraemia and traumatic/caustic injury. PCR testing for parapox virus was positive confirming orf as the cause. The absence of proliferative lesions was unusual in this case. It was not clear if orf virus was involved in the foot lesions as no further testing was carried out.

References:

1. Hill MWM, Whiteman CE, Benjamin MM, Ball L. Pathogenesis of experimental bovine mycotic placentitis produced by *Aspergillus fumigatus*. *Vet Path* 1971; 8: 175–192
2. Antonis AFG, de Jong MC, van der Poel WHM *et al*. Age-dependent differences in the pathogenesis of bovine respiratory syncytial virus infections related to the development of natural immunocompetence. *J of Gen Virol* 2010; 91(10):
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