SRUC Veterinary Services Monthly Report for February 2021

OVERVIEW

- **Malignant catarrhal fever and cerebellar abiotrophy in a Limousin calf.**
- **Congenital erythropoetic porphyria in a Limousin cross calf.**
- **Ill thrift and deaths due to chronic fasciolosis in sheep.**
- **Basilar empyema in tups.**

GENERAL INTRODUCTION

The mean temperature for February was 0.2 °C below the long-term average, and it was coldest relative to normal towards the north-east. Rainfall was 105 per cent of average with 116 per cent of average sunshine. Some southern/eastern areas were very wet, while the far north was very dry and sunny.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in May 2020. Given similar climatic and production conditions, they could also be important this year.

- **Coccidiosis in suckled calves**

  In beef herds the incidence of coccidiosis clearly peaks at one to two months of age making it easier to target prophylactic treatments if required. The number of diagnoses in younger calves increases in years where prolonged, cold spring weather and poor grass growth delays turn out.

- **Yolk sac infection in gamebird chicks**
  Chicks with yolk sac infections usually die in the first ten days after hatching with peak mortality between days four and six. Infection may occur at the breeding farm, hatchery, during transport or at the rearing site. A range of bacteria can be involved including *Escherichia coli*, *Staphylococcus aureus* and *Salmonella* spp.

CATTLE

Generalised and systemic conditions
Two six-month-old pedigree Limousin calves from a herd of 30 were noted to have neurological deficits. One was more mildly affected with ataxia and a short choppy gait. The other was initially ataxic and collapsed when moved or handled. It seemed to improve briefly before deteriorating very rapidly. A postmortem was carried out on farm, and samples submitted for histopathology. This revealed an extensive moderately severe necrotising enteritis and portal hepatitis which was indicative of malignant catarrhal fever (MCF). PCR testing carried out on liver confirmed this diagnosis. Examination of the brain revealed a nonsuppurative angiocentric encephalitis and cerebellar fibrinous meningitis consistent with MCF. However, there was also widespread cerebellar cortical degeneration consistent with cerebellar abiotrophy which is the most likely explanation for the neurological signs in the second calf.

Congenital erythropoietic protoporphyria was diagnosed as the cause of rapid deterioration and death in a one-month-old Limousin cross calf, which was the sixth to die in a herd of 40 suckler cows. It was not known if the deaths were related and history was limited, but the calf selected for postmortem examination was reported to exhibit neurological signs. Fixed tissues were received, and histopathology described deposition of abundant brown pigment that exhibited bright red birefringence with Maltese cross formation when viewed under polarised light, mainly within the hepatic portal areas and, to a lesser extent, within the alveolar septa. These findings were considered typical of congenital erythropoietic protoporphyria. Advice on sampling further cases for porphyria analysis, and information on genetic testing to identify carriers was provided. The condition is thought to have an autosomal recessive mode of inheritance and has been reported in both pedigree and cross Limousin cattle.[1]

Four animals from a group of 53 were either found dead or died following a short period of recumbency. A six-month-old Hereford stirk became recumbent and unresponsive and was euthanased for investigation of the problem. Postmortem examination revealed extensive bilateral blackening of the epaxial lumbar and mid-gluteal muscles. Fluorescent antibody testing proved positive for *Clostridium chauvoei* and the organism was also isolated on anaerobic culture confirming a diagnosis of blackleg. Clostridial vaccination was advised.

Two, one-week-old suckler calves present with mucopurulent oculo-nasal discharge, increased upper respiratory noise, frothing at the mouth and weakness. A nasopharyngeal swab was submitted from one calf and bovine herpes virus type 1 (BoHV1) was detected by PCR. SRUC VS commented that neonatal IBR is usually seen when naïve animals are introduced into an infected herd in late pregnancy. In this case the calves were born to homebred dams, the herd did not vaccinate and its BoHV1 status was unknown. However, a batch of store cattle had been introduced onto the farm six weeks previously and had shown signs consistent with IBR in the month prior to the birth of these calves. SRUC VS considered it possible that animals in the herd were naïve to BoHV1 and thus their calves did not receive antibody protection from circulating virus. Assessing passive transfer in the herd was also discussed.

Musculo-Skeletal conditions

A nine-month-old Highland heifer was euthanased after a month-long history of reducing motility, increased spells of recumbency and weight loss. Severe chronic active purulent/necrotising arthritis was identified as the cause of the clinical signs and histopathology of the synovial membrane identified changes consistent with a primary pyogenic infection with more limited necrotising lesions typical of those associated with *Mycoplasma bovis*. Cultures remained sterile however PCR/DGGE testing of synovial fluid proved positive for *M bovis.* Necrotising bronchiolitis typical of *M bovis* bronchiolitis was found in two cranioventral lung sections and it was postulated that this may have been the source of the secondary infection.

Circulatory system disorders

Two youngstock from group of 35 died in the three weeks following purchase. The carcase of the second, a 13-month-old Aberdeen Angus cross was submitted for postmortem examination and a severe necrotising myocarditis and fibrinous pericarditis was found (Fig 1). The cardiac form of blackleg was suspected but testing detected no evidence of *Clostridium chauvoei* infection. Histopathology suggested *Histophilus somni* myocarditis as a differential diagnosis and mycocardial tissue tested PCR positive for a high DNA load of *H somni*. The fibrinous pericarditis seen in this animal was considered an uncommon finding in the myocardial form of H. somni septicaemia.

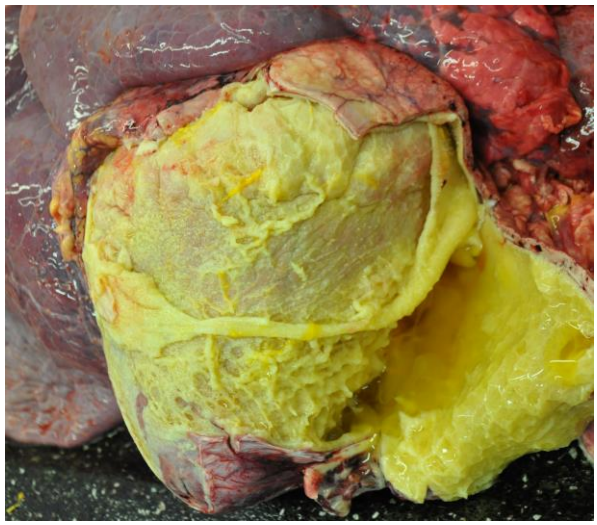

Figure 1 – Fibrinous pericarditis in association with *Histophilus somni* myocarditis

SMALL RUMINANTS

Parasitic diseases

Chronic fasciolosis was diagnosed as the cause of ill thrift and/or death in sheep from three flocks over a ten-day period in late February. All five submitted carcases were anaemic (Fig 2) with large numbers of *Fasciola hepatica* in the bile ducts and gall bladder. There was evidence of submandibular oedema in two. In all cases there was a history of treatment with triclabendazole in autumn 2020 and January/February 2021. Triclabendazole resistance was suspected but for welfare reasons testing to confirm this was not appropriate at the time. An information note was issued advising routine monitoring of untested flocks, particularly those that had used triclabendazole in the last six months. Undiagnosed liver fluke in late gestation ewes could lead to problems with metabolic disease, colostrum quality and lamb viability.

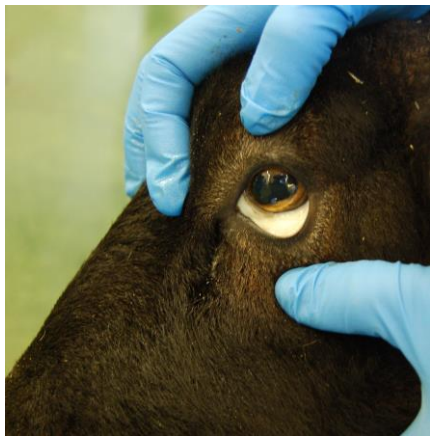

Figure 2 – Anaemia secondary to chronic fasciolosis

Alimentary tract disorders
A farmer reported the sudden death of two gimmers stocked on kale with access to silage. An on-farm postmortem examination was carried out and petechiae noted on the abomasal mucosa. Histopathology confirmed a bacterial septicaemia and detection of necrotising lesions in the abomasal mucosa and muscularis mucosa raised the possibility of listerial abomasitis. *Listeria monocytogenes* was isolated from liver and lung confirming a diagnosis of listerial abomasitis and septicaemia. Both silage and soil were considered possible sources of infection in this case.

Respiratory tract diseases
A flock of 60 pedigree ewes of mixed breeds lost three Texel ewes (all sired by the same tup) in a three-month period. Laryngeal chondritis was suspected in the first and confirmed on postmortem examination in the second. The carcase of the third Texel ewe was submitted with a history of recurrent respiratory signs prior to death. Bilateral laryngeal chondritis lesions were identified and abdominal haemorrhage originating from the diaphragm was confirmed as the cause of death. Histopathology revealed acute myofibre degeneration in the diaphragm and tearing of connective tissues and blood vessels leading to haemorrhage (Fig 3). Chronic damage and repair by scar tissue formation was also identified. The findings are similar to those seen in previous cases of acute abdominal haemorrhage in Texel sheep.[2]

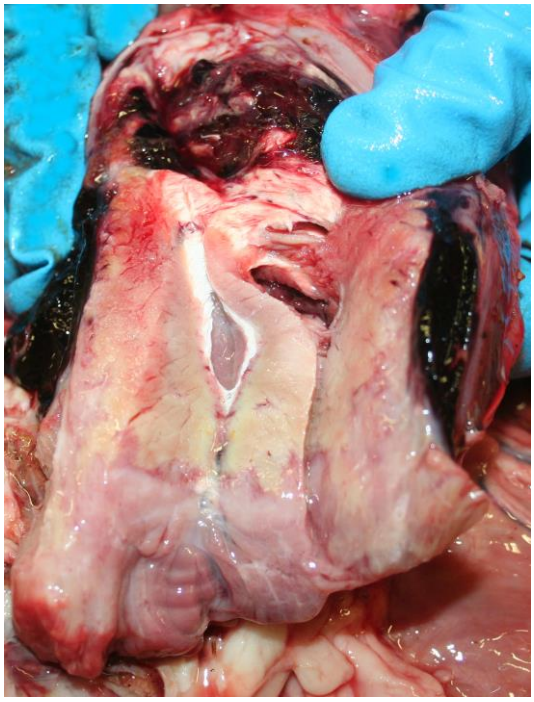

Figure 3 – Degeneration and tearing of the diaphragm in a Texel ewe with laryngeal chondritis

Nervous system disorders
Two cases of basilar empyema were diagnosed during February affecting Texel tups on two separate holdings. An 11-month-old tup lamb was noted to be dull and anorexic and was found dead the following day. Postmortem examination revealed an abscess in the pituitary fossa (Fig 4) and purulent exudate extended into the ventral meninges, the third ventricle and the lateral ventricles. The second case involved a three-year-old blue Texel tup that was treated with antibiotics after becoming ataxic but failed to respond and died within 48 hours. It has been suggested that seeding of bacteria in the carotid rete mirabile can lead to basilar empyema and possible sources of bacteria were identified in both animals. The tup lamb had laryngeal chondritis while a subcutaneous abscess was found at the poll of the older tup.

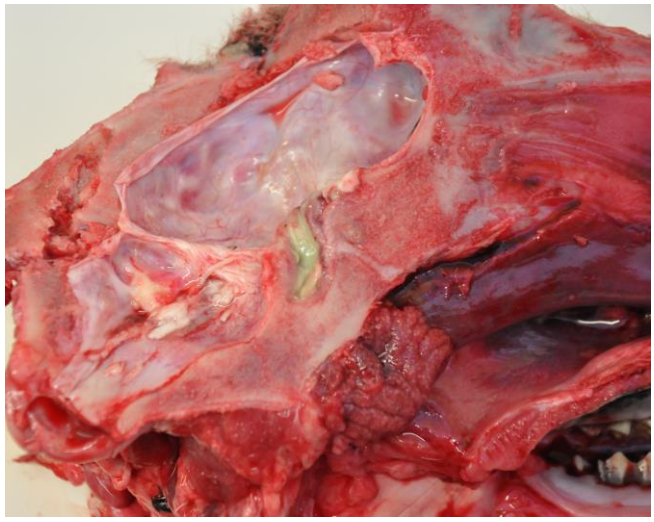

Figure 4 – Basilar empyema in a Texel tup lamb

Skin diseases
Squamous cell carcinoma (SCC) was diagnosed in a six-year-old ewe with a horn-like growth on the right side of the head. The lesion had first been noticed three months earlier. The histopathology findings of a densely cellular and keratinising epithelial neoplasm were consistent with a SCC. Potential risk factors for such lesions include papillomavirus infections, UV radiation (particularly in breeds with little skin pigmentation) and skin trauma such as ear tagging or punching.

BIRDS

Poultry
Ovarian adenocarcinoma was diagnosed on postmortem examination of two aged ex-battery hens that were euthanased following loss of condition. Multiple white, circular lesions consistent with adenocarcinoma were

present on the ovaries of both birds. Similar lesions were present in the mesentery and intestinal serosa (Fig 5).

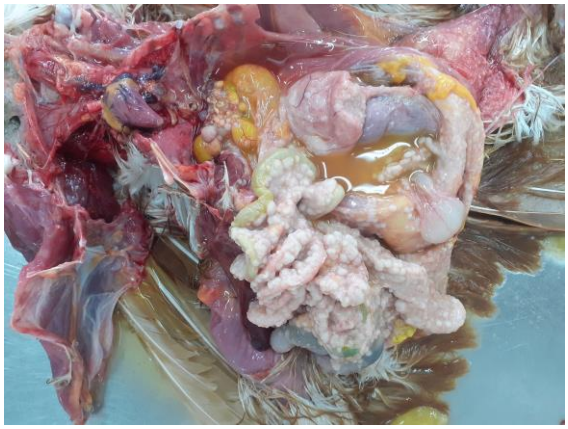

Figure 5 – Adenocarcinoma in an ex-battery hen

Wild Birds

A female buzzard (*Buteo buteo*) in very good condition was found dead in woodland adjacent to a garden fence and submitted to establish the cause of death. Bruising and swelling was found around the right eye and extending slightly down the neck (Fig 6). Clotted blood was found within the orbit but the globe was intact, there were no fractures and the brain was grossly normal. Trauma, perhaps caused by collision with the fence, was considered the likely cause. Liver analysis carried out at the SASA (Science and Advice for Scottish Agriculture) laboratory detected high levels of anticoagulant rodenticide residues (brodifacoum 0.307 mg/kg, bromadiolone 0.023 mg/kg, difethialone 0.002 mg/kg). This can lead to excessive bleeding following trauma and may have contributed to the death of the bird.

Figure 6 – Bruising secondary to trauma in a buzzard

MISCELLANEOUS

Deer

A five-year-old farmed red deer hind was submitted after being found dead following a seven-day history of ataxia. It appeared to be the only animal affected in the group of 220. Postmortem examination was unremarkable but neuropathology described symmetrical white matter degeneration in the brainstem and symmetrical myelopathy of the spinal cord typical of hypocuprosis. Liver copper was low at 80 umol/kg dry matter (DM) (reference range 180 to 300 umol/kg DM) despite supplementation with a trace element bolus four months previously, confirming a diagnosis of enzootic ataxia.

References:

1 McAloon CG, Doherty ML, O'Neill H *et al* Bovine congenital erythropoietic protoporphyria in a crossbred Limousin heifer in Ireland. *I Vet J* 2015; 68(15): DOI 10.1186/s13620-015-0044-3

2 Waine K, Strugnell BW, Howie F *et al* Diaphragmatic lesions and fatal haemorrhage in Texel sheep. *Vet Rec Case Rep* 2018 7(2) (e000745) https://doi.org/10.1136/vetreccr-2018-000745

Black Disease in Cattle

Necrotic hepatitis due to *Clostridium novyi* type B (black disease) is typically described as a cause of sudden death in cattle and sheep, often in association with *Fasciola hepatica* infection. It has been asserted that the condition is more common in sheep than cattle, however Scottish VIDA data from 2016 to 2020 identified 33 diagnoses in cattle and only 16 in sheep. It was diagnosed most frequently in the south-west which accounted for 31 of the 33 cattle diagnoses. In common with other *Clostridium* species, the bacteria exist as spores in the soil which are ingested and transported to the liver via the portal circulation. They persist in a dormant state until local anaerobic conditions allow germination, multiplication and production of toxins.

The clinical history and pathology in cases of bovine black disease was reviewed. It was diagnosed in cattle from ten months to thirteen years of age, but animals aged between one and two years accounted for 64 per cent of diagnoses. Two thirds of diagnoses were made in beef herds, with the remaining third in dairy herds. Although reported as a cause of sudden death, 17 of the 33 cases were seen alive and described as lethargic and dull with a reduced appetite. More specific clinical signs were also reported and are summarised in Figure A. In some cases blood samples were taken prior to death and biochemistry revealed glutathione dehydrogenase (GLDH) results up to 100 times the top end of the reference range. Only 27 per cent of animals had active *Fasciola hepatica* infection, indicating that *Clostridium novyi* can proliferate in the absence of parasitic damage. The initiating factor in these cases was not evident.

The classical hepatic pathology in cases of black disease is a firm pale lesion, bordered by an area of haemorrhage (Fig B). Other findings including subcutaneous oedema, pulmonary oedema, petechial haemorrhaging, chemosis or pericardial effusion are related to toxaemia. Carcases tend to autolyse rapidly which can provide a diagnostic challenge as, while the hepatic lesion is obvious in a fresh liver, it becomes more subtle as the carcase autolyses. The diagnosis can be confirmed by fluorescent antibody testing, anaerobic cultures and/or histopathology.

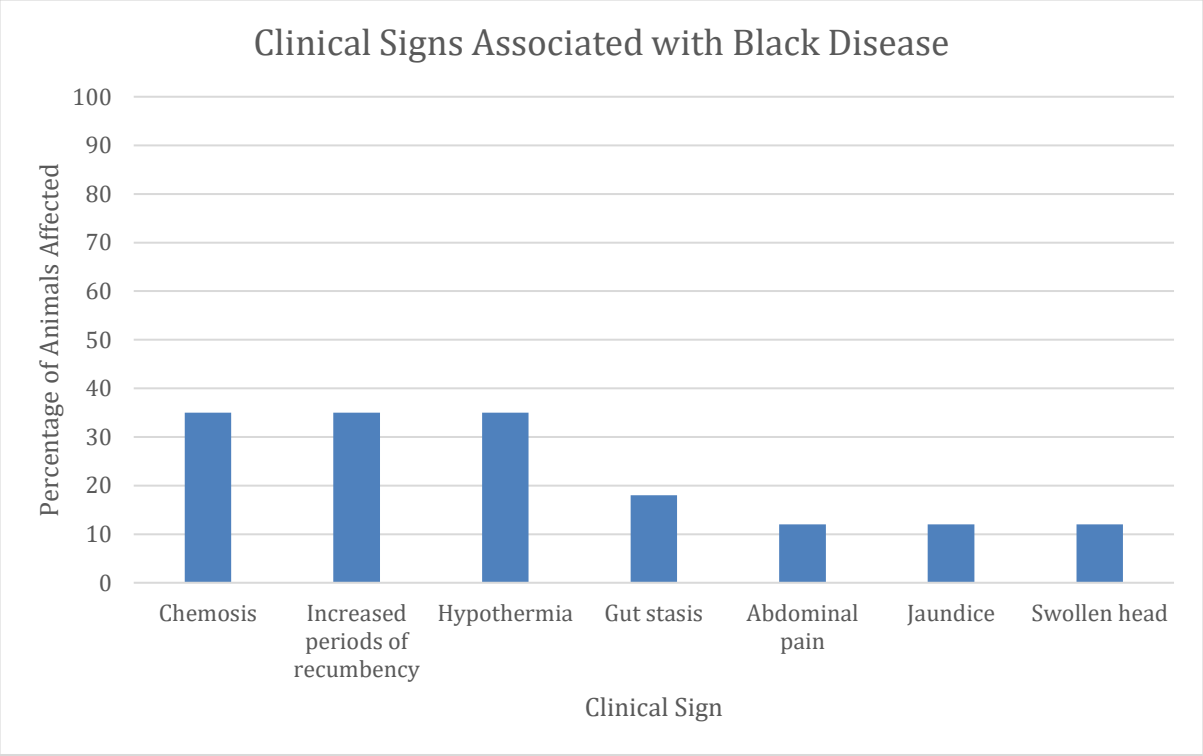

Figure A: Clinical signs in animals diagnosed with black disease

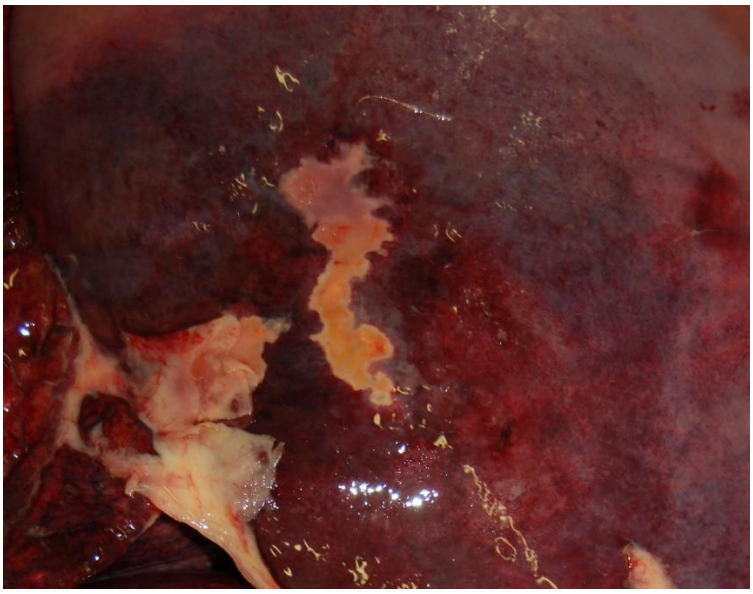

Figure B: A black disease lesion in a fresh carcase