# SRUC Veterinary Services Monthly Report for January 2021

## OVERVIEW

- **Congenital hepatic fibrosis in a cross bred foetus**
- **Detection of multiple abortifacient agents in a spring calving dairy herd**
- **Ovine pulmonary adenocarcinoma in eight-month-old lambs**
- **Death due to hepatic lipidosis in a domestic goose**

## GENERAL INTRODUCTION

The mean temperature was 2.1 °C below the long-term average making it the coldest January since 2010. It was a dry sunny month with 80 per cent of average rainfall and 136 per cent of average sunshine. This made it Scotland's fourth sunniest January in a series from 1919.

## DISEASE ALERTS

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

- **Colisepticaemia secondary to hypogammaglobulinaemia in neonatal beef calves**
  Dystocia and bradytocia associated with issues such as relative foetal oversize, excessive cow body condition and subclinical hypocalcaemia are predisposing factors that result in delayed or insufficient intakes of colostrum. In addition, the presence of acidosis following a difficult calving reduces the absorption of immunoglobulins. Colostrum should be administered to all calves with a poor suck reflex.
- **Outbreaks of ovine pasteurellosis secondary to infection with *Anaplasma phagocytophilum* (tick borne fever)**
  Deaths are seen following the movement of ewe replacements to hill pastures in spring. Both bought in and homebred animals may be affected including hoggs that have returned from wintering. Significant losses are common. Limited supervision and the extensive nature of the grazing mean good quality diagnostic material can be difficult to source. The need for tick prophylaxis should be considered in advance.

## CATTLE

### Alimentary tract disorders

An aborted foetus from a crossbreed heifer was examined following an assisted delivery. Postmortem examination revealed anasarca, ascites and a mottled liver with an irregular red reticular pattern on the surface (Fig 1). Histopathology confirmed marked portal-bridging fibrosis in the liver that was considered severe enough to have altered hydrostatic and osmotic pressures. The findings were consistent with congenital hepatic fibrosis, a rare and sporadic cause of death in foetuses and calves. The condition is thought to be due to abnormal biliary development and may have a genetic basis. This was the only case at the time in the herd.

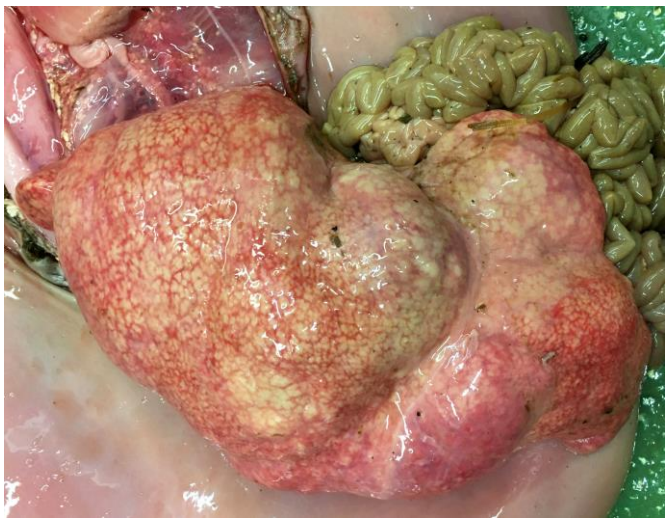


**Figure 1 – Congenital hepatic fibrosis in an aborted foetus**

### Reproductive tract conditions

Twenty abortions occurred in an 800 cow spring calving dairy herd over a six week period and material from seven foetuses was submitted for investigation. A slightly higher barren rate had been noted, at 13.8 per cent compared to 12 per cent in 2020, but this was attributed to the use of sexed semen. A mixture of artificial insemination and natural service was used. Cow condition was reported to be good and none of the aborted dams showed any clinical signs. Bacteriology of foetal stomach contents yielded three cultures of *Campylobacter fetus* and three of *Trueperella pyogenes.* The seventh foetus had histopathological changes

consistent with a diagnosis of neosporosis. SRUC VS commented that *T pyogenes* is not of herd level concern, and that due to the fastidious nature of campylobacter, it may have been present but not cultured. *C fetus* was considered to be the most significant finding and one isolate was typed as *C fetus venerealis intermedius*. The herd plan to only use artificial insemination in future.

A 740 cow suckler herd reported twelve abortions over a two-week period. The cattle were managed on two farms and both holdings were affected. Mixed ages of cows aborted between four and eight months of gestation. Three foetuses were submitted, and PCR testing of spleen proved positive for BVD virus in two. Of the two that were virus positive, one exhibited a congested brain with flattened gyri and histopathology confirmed a suppurative meningitis. Extensive haemorrhage within the abomasal wall was noted in the second (Fig 2) and histopathology confirmed a suppurative abomasitis. The third foetus tested positive for antibodies to BVDV. The diagnosis of BVDV abortions was consistent with the fact that both holdings had recently failed BVDV check tests having previously held Scottish Government BVDV negative status. Virus testing of all calves born in the previous 12 months detected a persistently infected (PI) animal on each holding. The herd had bought in a number of heifers during summer 2020 and it was advised that the dams of the PI calves and the purchased cattle were also screened for BVD virus.

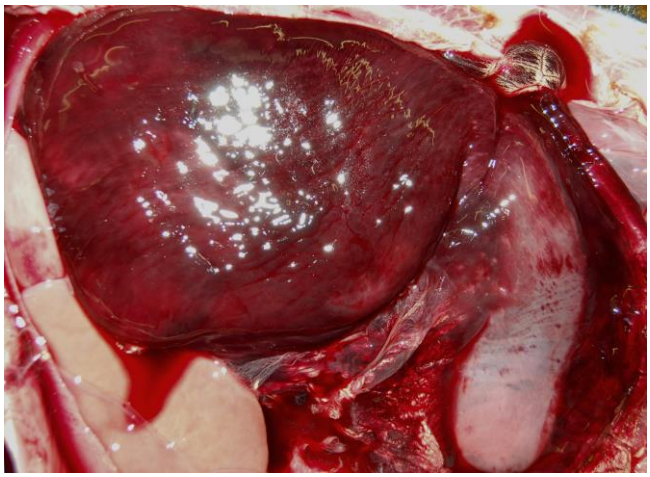


**Figure 2 – Abomasal haemorrhage in a foetus with BVD viraemia and a secondary suppurative abomasitis**

**Circulatory system disorders**

A two-month-old Limousin bull calf was found dead a few days after it had been purchased as a calf at foot. Postmortem examination revealed a severe restrictive pericarditis giving rise to marked left ventricular myocardial deformity (Fig 3) with secondary cardiac failure. The ventricular myocardial changes were considered to be a result of fibrous adhesions fixing the apex of the heart to the pericardial sac and restricting its movement. The original cause of the pericarditis was not detected on bacteriology.

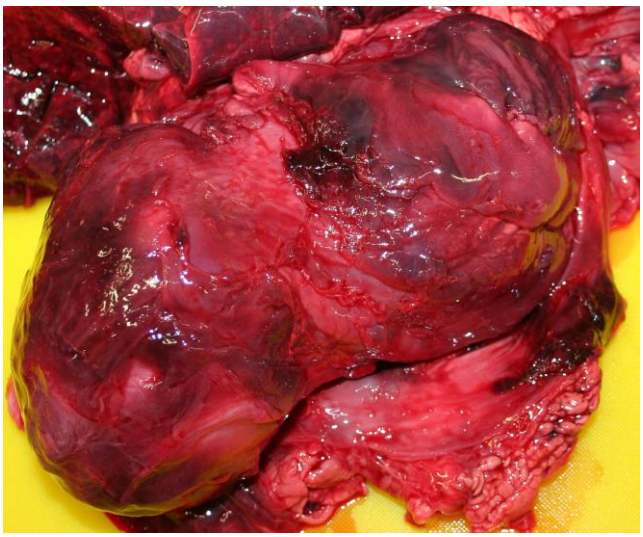


**Figure 3 – Marked distortion of the left ventricle in a two-month-old Limousin calf**

A two-year-old finishing heifer was treated for suspected pneumonia and appeared to have recovered until two weeks later when it rapidly deteriorated and died. A 7 cm long piece of wire was found penetrating from the reticulum into the pericardium, with associated adhesions in the cranial abdomen and caudal thorax. A purulent pericardial effusion together with a fibrinous pericarditis (Fig 4) had caused fatal restrictive congestive cardiac failure. The initial episode of illness was considered likely to represent pain associated with penetration of the wire.

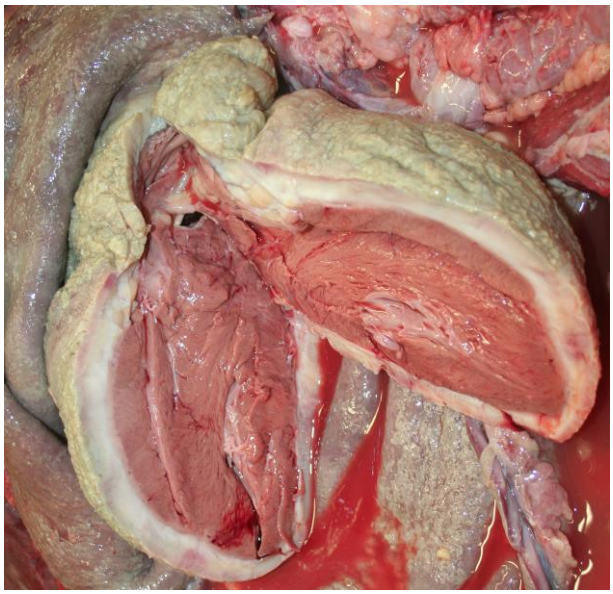


**Figure 4 – Traumatic pericarditis in a fattening heifer**

## SMALL RUMINANTS

### Alimentary tract disorders

Four crossbred ewes were submitted for postmortem examination in order to investigate ill thrift. Molar loss and impaction of food was found to be the issue in two cases. Extensive fibrinous adhesions were noted between the liver, diaphragm, omentum and body wall of the third ewe and numerous adult *Fasciola hepatica* were detected. Ovine pulmonary adenocarcinoma was diagnosed in the fourth. All four animals were seronegative for maedi visna virus and no evidence of Johne's disease was found. The range of findings illustrates the value of postmortem examinations in identifying issues that are relevant to both individual animals and at the flock level.

A yearling pygmy goat died after being found in lateral recumbency and fitting. The group of eight were noted to be lacklustre and there had been one previous death. Postmortem examination confirmed poor condition with little body fat, pulmonary oedema and a clear pericardial effusion. Faeces were diarrhoeic and the colonic mucosa was thickened. The coccidial oocyst count was moderate at 4300 oocysts per gram and *Yersinia pseudotuberculosis* was cultured from the ileum. Histopathology confirmed a severe purulent, necrotising enteritis consistent with yersiniosis. There was some evidence of coccidial damage which may have been a predisposing factor allowing bacterial colonisation.

### Respiratory tract diseases

Two eight-month-old Scottish blackface ewe lambs were submitted for postmortem examination after being found dead at grass. Both had pneumonia with grey focal lesions within the consolidated areas. *Mannheimia glucosida* was isolated from the lung of one lamb and *Mannheimia haemolytica* from the other. Histopathology confirmed underlying ovine pulmonary carcinoma in both cases. SRUC VS commented that OPA is unusual in this age of lamb and would tend to indicate a high challenge during the rearing period.

## BIRDS

### Poultry

The second goose to die from a flock of five over a four-day period was submitted for postmortem examination. The birds had been housed since November 2020 in order to comply with avian influenza regulations and were fed a diet that included maize, barley, peas, corn, goose mix, vitamin powder and bread. The two deaths did not appear to be related. The first goose was euthanased after exhibiting anorexia, wasting, head shaking and a nasal discharge, but a diagnosis was not pursued. The second bird presented differently and became recumbent one week prior to death. Postmortem examination revealed large deposits of subcutaneous and internal fat that encased the kidneys and intestines (Fig 5). The liver was pale, greasy and friable consistent with a diagnosis of hepatic lipidosis. SRUC VS commented that geese are susceptible to accumulating fat in their livers as an evolved mechanism to support long distance migration. Grass should form the majority of their diet and overfeeding with carbohydrates, particularly maize, can lead to accumulation of triglycerides in the liver. It was advised that any reduction in feeding to the surviving geese should be carried out slowly in order to avoid a metabolic crisis.

**Figure 5 – Hepatic lipidosis in a domestic goose**

## MISCELLANEOUS

### Camelids

A three-year-old female alpaca from a herd of 16 died following a three-day period of increasing tachypnoea and hyperpnoea. Despite this no abnormalities were detected on auscultation. Firm, brown material was found adhered to the endocardium of both ventricles reducing the functional lumen (Fig 6). The atrio-ventricular valves were unaffected. Ascites and hepatomegaly with a nutmeg appearance were considered secondary to cardiac insufficiency. Histopathology confirmed a fibropurulent mural endocarditis with the presence of marked granulation tissue indicating that it was chronic in nature. There was no evidence of infection with *Fasciola hepatica* which has previously been reported in association with endocarditis in alpacas.

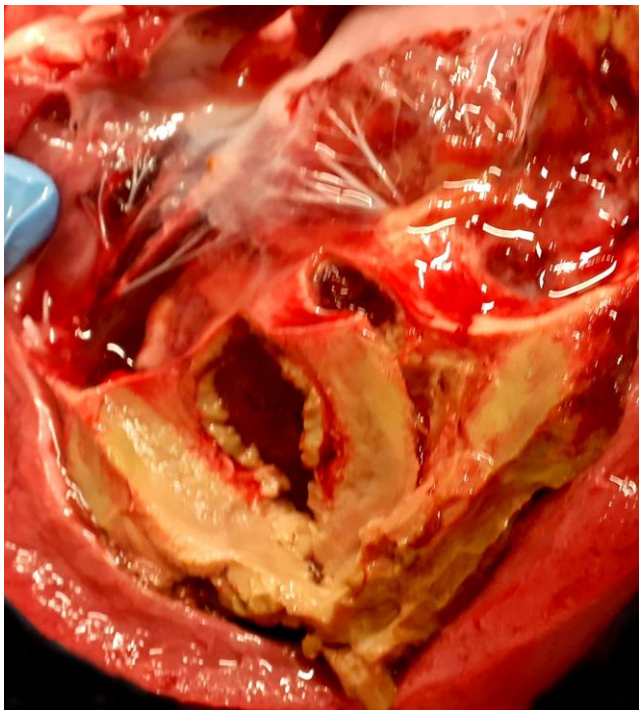


**Figure 6 – Mural endocarditis in an alpaca**

In addition to superficial laminar cerebrocortical neuronal necrosis (as outlined in part 1), in utero copper deficiency (congenital swayback) and the teratogenic effects of transplacental Border disease virus and other viral infections, are established causes of congenital neurological deficits in lambs. Additional causes include genetically determined conditions and in utero or intrapartum hypoxia.

Congenital swayback

Swayback is associated with low copper status of the dam at mid-gestation, and hence her lambs in utero. The disease tends to occur in areas in which the soil may have a low copper content, or copper uptake or utilisation may be affected by other factors, most notably high levels of molybdenum in the soil. Breed differences are apparent with hill breeds generally being more susceptible. The severity of clinical signs varies greatly, and some lambs may be stillborn. The most severely affected lambs are dull, weak and limp, occasionally with a fine head tremor. These clinical signs are associated with characteristic lesions that include extensive porencephaly and/or gelatinous transformation of cerebral white matter together with more subtle cerebellar dysgenesis and motor neurone degeneration. In contrast, the latter is the main neuropathological finding in less severe cases in which progressive ataxia is the most notable clinical finding, but the presence of motor neurone degeneration alone is not sufficient for a diagnosis of swayback; other lesions such as cerebellar cortical degeneration have more specificity. The hepatic copper concentrations in affected lambs are usually well below the reference range, however occasionally copper concentrations may increase in late gestation if the dam has received copper supplementation towards the end of pregnancy. In these latter cases, further flock history may be useful (see box), together with assessment of copper status of the ewes prior to tupping.

Border disease, and other teratogenic infections

Two main presentations are associated with in utero Border disease virus (BDV) infection. The most characteristic presentation is continuous tremor which is the result of delayed myelination of the central nervous system, sometimes associated with fleece abnormalities ('hairy shakers'), in lambs that are exposed to BDV in early gestation resulting in persistent infection. If the lambs survive the postnatal period, the clinical deficits may slowly resolve, however as these lambs are persistently infected, they may act as a reservoir of infection in the flock. Midgestational BDV infection may result in CNS malformations, most commonly involving the cerebellum and less frequently the cerebrum (porencephaly, hydrocephalus) and hence usually present with cerebellar signs and variable degrees of dullness. Most lambs with BDV associated malformations have cleared the virus and have seroconverted to BDV, therefore detection of BDV antibody in a pre-colostral serum sample is required for confirmation. In most cases a precolostral blood sample is not available and the diagnosis depends on demonstration of the characteristic neuropathology and high BDV antibody titres in the dam. However, some lambs with BDV malformations are persistently infected, therefore screening for BDV RNA also is recommended.

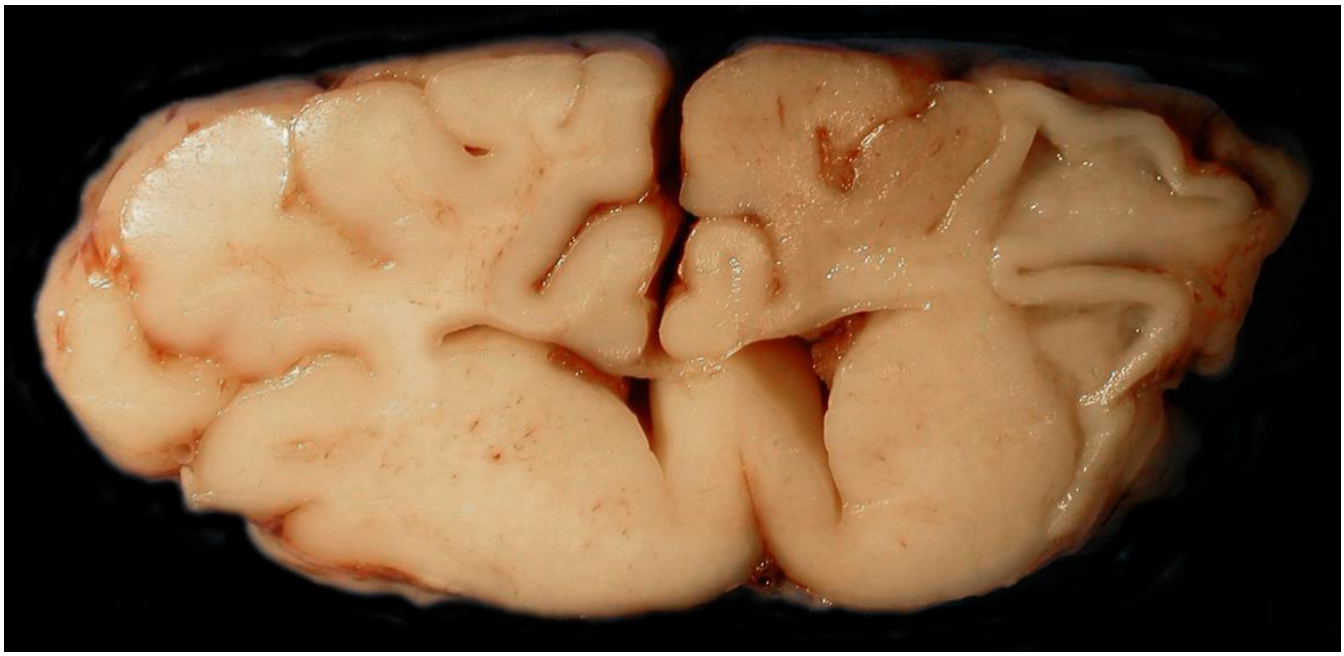
Transplacental bluetongue virus (BTV) infection, including BTV8, may also result in CNS malformations in lambs, although this is much less common in sheep compared with cattle. The finding of hydranencephaly (where the cerebrum is replaced by fluid filled sacs, within a skull that is normal or slightly reduced in size) in ruminants should be reported to APHA. The large majority of lambs with teratogenic Schmallenberg virus (SBV) infection present with arthrogryposis however a minority may present with only brain malformations. For example, rare lambs with isolated cerebellar dysgenesis that present with non-progressive signs of cerebellar dysfunction.

Other in utero infections, for example *Toxoplasma gondii*, may result in neurological presentations, mainly dullness, in non-viable lambs. These usually occur against a background of reproductive losses in the flock.

Inherited conditions such as daft lamb disease in a range of breeds, prenatal onset cerebellar abiotrophy, lysosomal storage diseases and other inherited metabolic disorders also affect the CNS as do malformations such as hydrocephalus and rhombencephaly, vertebral malformations and occult spina bifida. Finally, it should not be forgotten that dystocia can result in intrapartum hypoxia and cerebral injury.

**Clinical history**

- Has the problem coincided with a change in management compared to previous years? This may include differences in trace element supplementation, feeding or pasture that may potentially affect copper and energy status of pregnant ewes.
- Has a sire been used for the first time in the flock? Examination of breeding records with particular regard to possible in-breeding can be helpful.
- Were biosecurity precautions taken when introducing replacement stock? Is it the offspring of homebred and/or bought in stock that are affected?
- Are all lambs in multiple births affected? If so to a similar or differing degree? Some lambs with swayback lesions may have siblings that are much less severely affected but generally thrive less well than expected (and may eventually develop swayback signs). In contrast, lambs less severely affected with SLCCN (putative late gestational energy deprivation) will recover with adequate care.



Unilateral congenital swayback