

OVERVIEW

- **Q fever as a cause of stillbirths in dairy heifers**
- **Outbreak of cerebrocortical necrosis in housed fattening bullocks**
- **Congenital hepatic arteriovenous malformation in a Highland calf**
- **Rickets in hogs on a grass re-seed**

GENERAL INTRODUCTION

Rainfall and sunshine figures for February were similar to the thirty-year average for the period between 1990 and 2020. In contrast the mean temperature was 1.1°C higher.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in May 2023. Given similar climatic and production conditions, they could also be important this year.

- **Malignant catarrhal fever**
Cases in Scotland occur predominantly in the late spring, peaking in May likely reflecting increased contact between cattle and sheep at lambing time. The association with sheep is emphasised by the fact that the majority of diagnoses are in beef cattle; with most beef farms in Scotland having both cattle and sheep. Detection of ovine herpesvirus 2 from EDTA blood samples or spleen from suspected cases confirms the diagnosis.
- **Clostridial enterotoxaemias in neonatal lambs**
Both lamb dysentery and pulpy kidney disease can be a cause of sudden death in neonatal lambs. Cases are predominantly seen in flocks where clostridial vaccination is not routinely practised. In vaccinated flocks screening lambs for adequate transfer of maternal colostral antibodies may detect hypogammaglobulinaemia. The pathology in lamb dysentery is usually characteristic and laboratory confirmation of the diagnosis requires histopathology and/or detection of beta toxin in small intestinal contents. Making a diagnosis of pulpy kidney disease can be more challenging. Postmortem examination findings are very variable and histopathology is often the most cost effective way to investigate suspect cases. Collecting a range of tissues (including brain) into formalin is advised.

CATTLE

Respiratory tract diseases

A two-month-old Aberdeen Angus cross heifer calf was found recumbent, kicking and roaring but died before it could be examined. It had been treated twice for pneumonia in January and postmortem examination confirmed bilateral lung consolidation affecting 50 per cent of the parenchyma. Additional findings included multiple haemorrhages within the tissues of the neck, blood clots in the distal trachea plus blood splashing and fibrin deposits on the parietal pleura. No abnormalities were observed on direct examination of the brain or on histopathology. *Pasteurella multocida* was isolated from lung tissue and PCR testing detected significant levels of *P. multocida* and *Mycoplasma bovis*. The rib calcium:magnesium ratio was high at 120 (reference range 30 -70) indicating terminal hypomagnesaemia that explained the clinical signs. Access to creep feed had been withdrawn over the previous two days and this combined with reduced dry matter intake in a sick calf was considered to have predisposed to hypomagnesaemia in this case.

Reproductive tract conditions

The first 12 Jersey heifers in a large seasonally calving dairy herd gave birth to full term stillborn or weak calves. The group of 20 were in calf to synchronised AI, there was no history of bradytocia, and calves born to cows were unaffected. Four stillborn calves, three placentas and one live born calf were examined postmortem. A suggestion of placentitis was noted in one, and suspected inclusion bodies were seen on a modified Ziehl-Neelsen stained smear of placenta. This triggered PCR testing for *Coxiella burnetii* (Q fever) which proved positive. Routine testing failed to produce a diagnosis in the other four cases but PCR testing of the other two placentas also returned a positive Q fever result. Histopathology was inconclusive with only placental congestion and oedema detected. Nonetheless, it was proposed that the detection of *Coxiella burnetii* was significant because the heifers had been reared away from home before returning to mix with dry cows in late gestation. This has previously been shown to be a risk factor for Q fever associated stillbirths.

A beef herd comprising 175 cows reported three abortions and submitted a Saler cross foetus and placenta for investigation. Postmortem examination identified pericarditis and thickening of the intercotyledonary placenta. *Bacillus licheniformis* was cultured from the foetal stomach contents and histopathology confirmed widespread suppurative changes in the foetal tissues and placenta. The herd was due to start calving two weeks later, and the crown

rump length indicated that the abortion had occurred at around seven months gestation. Abortions due to *B licheniformis* are most common in housed, pit silage fed, spring calving beef cows in the last two months of gestation. Feeding the best available silage and regularly cleaning out water troughs can help to minimise challenge with this environmental bacterium.

Musculo-Skeletal conditions

Blackleg was confirmed as the cause of death in housed beef calves from two holdings. A five-month-old Charolais calf was found recumbent and comatose after appearing well the previous day. It died rapidly, on farm postmortem examination identified typical blackleg lesions in the right hind leg and *Clostridium chauvoei* was isolated from the affected muscle. The farm had reported issues with blackleg for the first time during the previous grazing season and the affected animal was from a cohort of later born calves that had not been vaccinated. In the second case a 12-month-old Simmental cross bullock was reported to be dyspnoeic the day before death. Pneumonia was suspected as there had been eleven presumed respiratory disease related deaths since housing. On farm postmortem examination found an area of dark haemorrhagic muscle in the brisket and a small volume of serosanguinous pericardial fluid. *Clostridium chauvoei* was isolated from the myocardium and histopathology confirmed acute necrosis, inflammation, and surface fibrin exudation. Bacteria consistent with *C chauvoei* were present but not particularly associated with the myocardial necrosis. Nonetheless cardiac blackleg was considered the most likely cause of death based on the history, postmortem findings, and rapid carcass autolysis. No evidence of pneumonia or inflammation in the discoloured muscle from the brisket was detected.

Nervous system disorders

Five fattening animals from a group of 50 presented with blindness, hypermetria and ataxia over the course of a six-week period. Two affected animals progressed to recumbency prior to death and a recumbent 15-month-old Luing cross was euthanased for investigation of the problem. Postmortem examination was mostly unremarkable however localised areas of the brain were found to fluoresce under ultraviolet light (Fig 1). This suggested a diagnosis of cerebrocortical necrosis (CCN) confirmed on histopathology which described severe multifocal laminar necrosis in the cerebral cortex. The two remaining affected animals recovered following treatment with vitamin B1. Proliferation of thiaminase producing bacteria within the rumen is thought to induce CCN, however the conditions that promote their multiplication remain uncertain. Predisposing factors

may include diet change, high cereal inclusion rates, lead poisoning, chronic copper toxicity and high levels of dietary sulphur all of which were excluded on history and diagnostic testing. The affected animals were fed a total mixed ration comprising silage and 4kg barley/head/day. A second group of cattle in the same shed received 10kg barley/head/day and were unaffected. It was hypothesised that the outbreak may have been caused by inadvertently feeding a higher than intended quantity of barley to the affected group resulting in an abrupt increase in cereal intakes on at least two occasions.

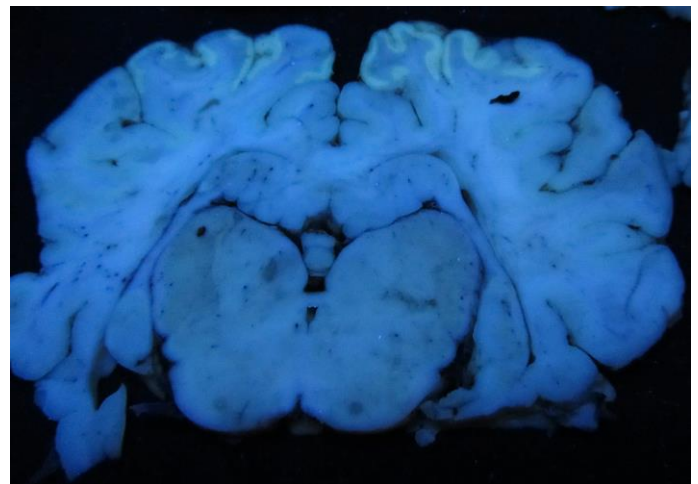


Figure 1 – Fluorescence of the cerebrum under ultraviolet light in a case of cerebrocortical necrosis

Circulatory system disorders

A Highland calf that had been born unassisted failed to stand and died within 24 hours. Postmortem examination detected an unusual cluster of torturous dark blue blood vessels embedded in the parietal peritoneum cranial to the right kidney (Fig 2). These extended to the liver which was firm and distorted with dilated blood vessels within some areas of the parenchyma. Histopathology identified an area of fibrosis and dense vasculature within the hepatic parenchyma likely representing a fistula between the arterial and venous circulation. This has been described in cases of hepatic arteriovenous malformation, in which there are congenital communications between the hepatic artery and portal vein. High pressure arterial blood enters the venous portal circulation resulting in retrograde flow, arterialisiation of the portal circulation and development of

portal hypertension. Ultimately this leads to the formation of extrahepatic portosystemic shunts. A similar case was recently reported in the literature and the authors

commented that arteriovenous malformations have been widely studied in humans and can have both congenital or acquired aetiologies.¹ They are rare in animals and occur congenitally without a defined cause.



Figure 2 – Tortuous blood vessels in a Highland calf with a congenital hepatic arteriovenous malformation

SMALL RUMINANTS

Musculo-Skeletal conditions

Fifteen easycare hogs from a group of homebred animals at grass were reported to be lame with five severely affected. There had been no response to treatment, one animal continued to deteriorate and was submitted for euthanasia and postmortem examination. It was able to stand but reluctant to walk with a tendency to hold its hind feet under the body and take small steps. It was thin and weighed 19kg, although some body fat remained. Bone quality was poor, and the ribs were easy to break. No lesions were noted in the muscles or joints however the growth plates of the femur, radius and metacarpal bones were examined and considered wider than expected for a ten-month-old animal (Figure 3). The hogs had spent the previous six weeks in a reseeded field with access to large amounts of grass and no supplementary feeding. The grazing history together with the appearance of the growth plates suggested a diagnosis of rickets which was confirmed on histopathology. This showed thickening of the growth plates and persistence of unmineralized cartilage within the primary spongiosa. Frequent microfractures indicated weakness of newly formed spongiosa. It was

advised that the rest of the group were treated with an injection of vitamin D and/or introduced to concentrate feed.



Figure 3 – Expansion of the distal femoral growth plate in an easycare hog with rickets

Nervous system disorders

120 homebred Scottish blackface hogs were housed in autumn on a diet of hay plus concentrates twice a day. Three, ten-month-old animals were found recumbent and paddling in February. Circling, blindness and opisthotonos were not observed. Two carcasses were submitted, and examination of the brains detected no evidence of cerebellar coning or fluorescence under an ultraviolet light. Histopathology confirmed listerial meningoencephalitis in both. An incidental finding was the presence of fresh-looking haemorrhagic tracts several centimetres in length on the liver surface (Fig 4) and hepatic migration of *Cysticercus tenuicollis* was considered to be the cause. This takes place from four weeks following infection indicating that this had occurred during the housing period. Preventing dogs scavenging carcasses and/or contaminating feed with faeces was advised.



Figure 4 – Haemorrhagic tracts in an ovine liver following migration of *Cysticercus tenuicollis*

Renal diseases

A full-mouthed Texel tup became dyspnoeic three months after arriving at an animal rescue centre. It was treated for suspected pneumonia but died three days later. External examination of the carcass identified extensive scab material at the base of the fleece over the thorax and abdomen and *Psoroptes ovis* mites were detected triggering a report to the local Animal Health Office under the terms of The Sheep Scab (Scotland) Order 2010. The scrotum was oedematous, the carcass smelled uraemic (aqueous humour urea 91.2 mmol/l), and a large volume of light red serous fluid was present in the abdomen. The bladder was full and there was bilateral hydronephrosis with marked accumulation of fluid within the renal capsules (Fig 5). A 2 x 1 cm necrotic lesion was found on the preputial mucosa. The vermiform appendage was intact, but the urethra appeared necrotic along most of its length. Histopathology confirmed a fibrinosuppurative urethritis and revealed large numbers of spermatozoa, with associated necrosis, in the kidney. This was presumed to be a result of vesiculoureteral reflux and was considered to have increased the inflammatory response due to the immunogenic nature of spermatozoa.

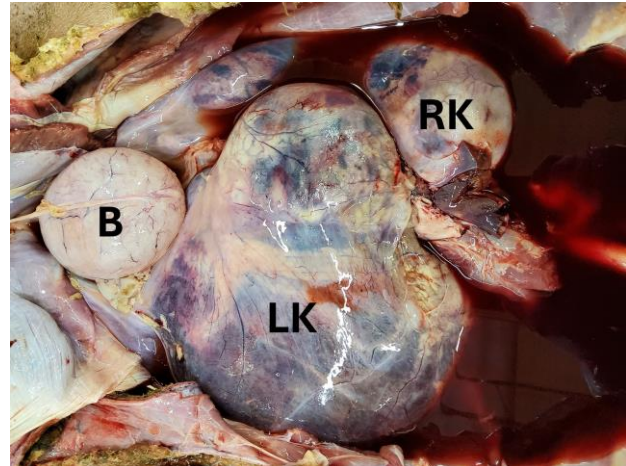


Figure 5 – Marked accumulation of urine within the renal capsules of a Texel tup with urethritis (B - bladder, RK - right kidney and LK - left kidney)

Skin diseases

The carcass of a thin, 11-month-old beltex tup was submitted for investigation of severe, chronic orf that had failed to respond to treatment. The group had received a parapox virus vaccine and were stocked on stubble turnips with supplementary concentrate feed. Postmortem examination found extensive proliferative dry crusting lesions over the head and ears. There was hair loss and thickened skin on the distal limbs and a 2 to 3 cm diameter raised red granular lesion on the anterior aspect of the left foreleg adjacent to the coronary band. The draining lymph nodes were enlarged, and PCR testing of lesion material was positive for orf virus. Bacteriology produced a mixed growth including *Trueperella pyogenes*, *Streptococcus dysgalactiae* and *Fusobacterium necrophorum* all of which will have been contributing to the lesion following secondary infection. No bacteria resembling *Dermatophilus* sp were observed. Screening for border disease virus was negative and there was no evidence of trace element deficiency ruling both out as a cause of immunosuppression. The flock owner reported small numbers of similar cases in previous years. Carcasses with severe chronic orf are examined periodically by SRUC VS and cases have been described in the literature², but no evidence of an underlying immunodeficiency has been detected.

References:

1 - Journal of Comparative Pathology: Soares YGS, Ferreira JS, Campos ÉM, Alves RC, Leite IM, Soares LA, Ferreira MAS, Dantas AFM, Riet-Correa F, Galiza GJN. Hepatic arteriovenous malformation in a calf. *J Comp Pathol.* 2023 Aug;205:27-32.

2 – Smith GW, Scherba G, Constable PD *et al.* Atypical parapoxvirus infection in sheep. *J Vet Intern Med* 2002;16:287-292