SRUC Veterinary Services Monthly Report for April 2025

OVERVIEW

- **Congenital calf deformities unassociated with Schmallenberg virus infection**
- **Losses due to water deprivation/salt poisoning in housed beef cows**
- **Neonatal lamb mortality associated with dam undernutrition**
- **Squamous cell carcinoma affecting C1 in an alpaca**

GENERAL INTRODUCTION

April 2025 was the third warmest in a 142-year series with mean temperatures across the whole of Scotland between 1.5 and 2.5˚C above the thirty-year average. It was the second sunniest April on record (190-year series) with 146 per cent of average sunshine hours. Forty-eight per cent of average rainfall made it the 19th driest in a 116-year series.

DISEASE ALERTS

Cardiac blackleg

The resistant spores of *Clostridium chauvoei* are found in soil and other organic material, where they can survive for years. Following ingestion spores are distributed to tissues, including muscle, where they may survive for long periods. Any conditions which compromise the oxygen supply to muscles harbouring latent spores will create an anaerobic environment, spore reactivation, and development of blackleg. Outbreaks of blackleg can occur in association with excessive exercise, soil disturbance or flooding. Cardiac blackleg most often presents as sudden death with postmortem findings of pericarditis and myocarditis. Typical muscle changes can be less obvious than those found in skeletal muscles. Histopathology is a reliable way to confirm the diagnosis.

DISEASE ALERTS

Idiopathic necrotising enteritis

Idiopathic necrotising enteritis (INE) is a disease of spring born suckled calves that usually occurs between the ages of 6 and 12 weeks. It is characterised by multifocal necrosis and ulceration of the gastrointestinal tract but lesions may also be found in the oropharynx, larynx, oesophagus and abomasum. Affected calves rarely respond to supportive treatment and have a poor prognosis with death after approximately 2 to 10 days. The cause remains unknown, and confirmation of the diagnosis requires histopathology and exclusion of BVD infection.

CATTLE

Alimentary tract disorders

A three-month-old Limousin cross heifer calf was sedated and disbudded uneventfully. It was dull for the next two days and was then treated with antibiotics and NSAIDs for suspected early pneumonia a week later. It improved slightly but became recumbent and was found bellowing with pricked ears, starey eyes and no menace response. It died within 20 minutes and was submitted for investigation. Postmortem examination found a generalised lymphadenopathy and a single focus of necrotic consolidation in the right lung from which *Mannheimia haemolytica* was cultured. The liver was swollen and orange with a white mottled appearance to the parenchyma, and histopathology confirmed severe diffuse bridging portal fibrosis with marked proliferation of the bile ducts (Fig 1). Bile lakes with an associated

Part-funded by the Scottish Government as part of its Public Good Veterinary Advice Services

granulomatous response were also noted indicating damage or rupture of bile ducts. These findings suggested that hepatic encephalopathy was the cause of the neurological signs, further supported by marked bilateral white matter vacuolation in the substantia nigra. Congenital hepatic fibrosis (due to embryonic ductal plate malformation) or congenital biliary atresia were the most likely explanations, ingestion of ragwort during pregnancy considered less likely. A five-day -old calf was reported to have presented similarly earlier in the year however a different aetiology, such as bacterial meningitis, could not be excluded. Information was sought regarding possible in-breeding.

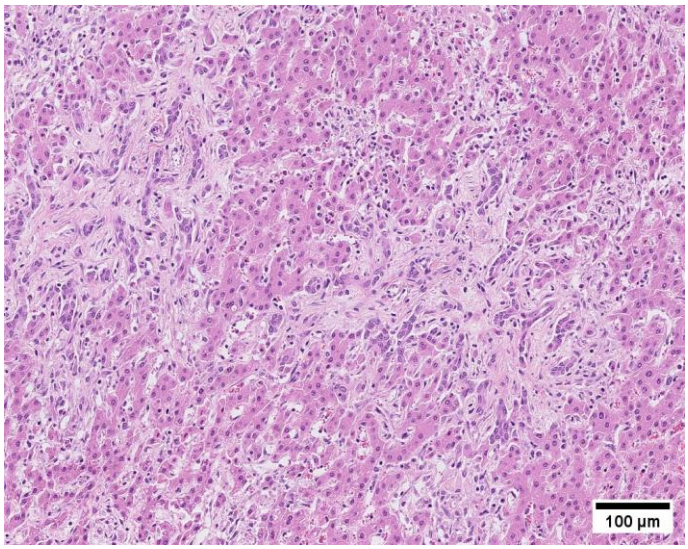

Figure 1 – Liver section from a calf showing severe diffuse bridging portal fibrosis with marked proliferation of the bile ducts

A well grown six-week-old simmental cross bull calf weighing 123 kg was found dead. It was the only loss in the group and had been clostridial vaccinated and disbudded the previous week. Postmortem examination found a generalised peritonitis secondary to rupture of an abomasal ulcer. The 1cm diameter defect was surrounded by an area of blackened mucosa and the mesenteric lymph nodes were reactive. The liver selenium result was within the reference range and there were no significant findings on bacteriology. The most likely explanation was that repeated ingestion of large volumes of milk had resulted in repeated distension of the abomasum with mucosal compromise due to reduced perfusion.

Respiratory tract diseases

A herd reported that they had treated more than ten, three-to eight-week-old suckled calves at grass for suspected pneumonia. Two died and the submitted Limousin cross calf had been dull in the evening and found dead the following morning despite treatment with antibiotics and NSAIDs. External examination identified a single tick in the inguinal region. Internally there was a fibrinous peritonitis with enlargement of the mesenteric lymph nodes and adhesions between the jejunal loops. A thick layer of fibrin coated the thoracic surface of the diaphragm and there was a severe fibrinous pericarditis and minor areas of lung consolidation. Bacteriology was unrewarding but histopathology confirmed a severe fibrinous polyserositis associated with Gram-negative bacilli consistent with a *Pasteurellaceae* species. Spleen tested PCR positive for *Anaplasma phagocytophilum*. It was considered that immunosuppression secondary to tick borne fever could have been responsible for this outbreak of respiratory disease in young calves. Moving the group to a field with no tick habitat was advised if possible.

Reproductive tract conditions

Calves with congenital limb and spine deformities were submitted from a range of holdings in April in order to investigate the possibility of foetal exposure to Schmallenberg virus (SBV). A single calf from an 80-cow spring calving herd was found to have multiple deformities including arthrogryposed forelimbs, a malformed pelvis, a single kidney, abnormally shaped liver, and a blind ending jejunum with aplasia of the distal

intestines and anus. A slightly bulging, hairless area covered by a thin red membrane was found dorsally at the level of the sacrum. This communicated directly with the spinal canal and the spinal cord protruded at this point. These findings are consistent with spina bifida with failure of neural tube closure resulting in formation of a meningocele. The additional abnormalities were also indicative of defective development during early embryogenesis. No evidence of infection with SBV was found.

Two abortions occurred in a group of 50 housed Simmental cross cows. Scoliosis, cleft palate, brachygnathism and a reduced range of movement in the forelimb joints were identified in the first. Microphthalmia and doming of the cranium were noted in the second and examination of the brain found dilation of the lateral and third ventricles. In this case histopathology revealed a severe suppurative placentitis which was the likely cause of abortion but incidental in relation to the deformities. There was no evidence of in utero infection with SBV in either case. Testing to exclude Bluetongue virus was initiated in the second case and also proved negative.

The cause of the congenital abnormalities remained unknown with no further cases reported.

Musculo-Skeletal conditions

A large beef herd reported that 25 calves from 250 cows calved so far had been stillborn with limb deformities. Other affected calves had been born alive with offspring from multiple dam breeds and a range of sires and groups affected. Examination of three typical cases identified different degrees of long bone deformity with the femurs and humeri being disproportionately short (Fig 2). Hyperextension of the metacarpophalangeal joints was evident in the most severely affected calf. Sectioning of the bones revealed that the growth plates were abnormally wide. Histopathology confirmed chondrodystrophy and it was noted that this can be genetic/nutritional or of unknown aetiology. Initially it was reported that there had been no changes in feeding management compared to previous years. However, it later transpired that first cut silage had been fed via a trailer prior to the cows being housed. This was suggested as the trigger for the outbreak as feeding good quality silage on its own during months four to five of gestation is a known risk factor for long bone deformity although the precise aetiology remains to be elucidated.

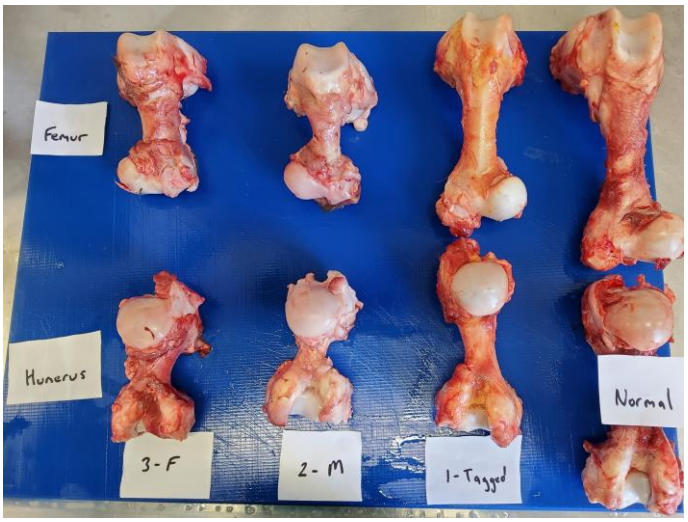

Figure 2 – Reduced length of the femoral and humeral shafts in calves with long bone deformity

Nervous system disorders

Botulism was strongly suspected to be the cause of clinical signs and death over a two-week period in thirteen, 10-month-old weaned calves from a group of 400. The cattle had been purchased in October and were at grass on well drained sandy soils with access to a mix of silage, brewers draff, crimped barley and minerals via a trailer. Affected animals initially appeared stiff or lame and became sternally recumbent within 24to 48 hours. They deteriorated further into

lateral recumbency over next 24 hours and the majority were euthanased due to lack of response to symptomatic treatment. One animal in a shed housing 700 cattle with access to the same diet presented similarly. Postmortem examinations were carried out on eight cattle with no specific pathological findings and histopathology of the brain and spinal cord was not diagnostic. There was no evidence of hypomagnesaemia or hypocalcaemia and no evidence of nutritional myopathy. Hen litter had been spread on the silage fields after the first cut of silage was taken in 2024 and second/third cut silage was being fed when the problem arose. ELISA testing of four samples of small intestinal content was negative for botulinum toxin, but this does not exclude a diagnosis of botulism as the toxin may become undetectable. The history, clinical signs and absence of an alternative diagnosis was suspicious for botulism. The feed areas were thoroughly cleaned of any waste silage and the remainder of the second and third cuts was discarded. Deaths subsided rapidly after changing to first cut silage. *Clostridium botulinum* vaccination was recommended if hen litter was going to be used as fertiliser in the future.

Ten cows in a group of 30 housed suckler cows died over a weekend in April with deaths in two adjacent pens of 45. Cattle were either found dead or in some cases became recumbent and showed opisthotonos for a short period before death. They had been housed on slats since November on a total mixed ration of silage, straw, pot ale syrup plus minerals and were due to calve in April. The water supply to these pens was suspected to have been interrupted for 48-hours. The cattle were then given free access to water which they rapidly drank. Losses started the following day, and two cows were submitted for postmortem examination. Ocular fluid analysis showed slightly elevated urea in both, and calcium and magnesium levels were unremarkable. There were no significant findings

on culture and no significant abnormalities on brain histopathology. The clinical history was strongly suggestive of water deprivation/salt toxicity. Diagnosis of the condition in the acute stages is difficult as the main finding is cerebral oedema which can be difficult to assess. This can progress to cerebrocortical necrosis if the animal survives long enough. The farmer was aware of water pressure issues on the farm, and the two affected pens were furthest away from the supply. This case is a reminder that in these situations, cattle should be gradually re-introduced to water over a few days.

SMALL RUMINANTS

Nutritional and metabolic disorders

An emaciated adult pygmy goat was submitted from a herd of eight with access to both indoors and out. Hay and pygmy goat mix were also available. The carcase was very pale with white conjunctivae. Rumen fill was fine with normal content and no evidence of diarrhoea. An area of inflammation was found on the serosa of the proximal colon which had perforated at this point. *Oesophagostumum* sp worms were visible through the defect (Fig 3). The abomasum and small intestines were washed out, but only small numbers of nematodes were recovered and not considered significant in relation to the ill thrift. The liver copper result was below the lower detection limit of the test confirming severe hypocuprosis as the problem. Histopathology was carried out to determine if the *Oesophagostumum* sp worms were responsible for the perforated colon however this proved not to be the case. Findings indicated a subacute course with possible previous damage at the site, but the cause was not clear.

Figure 3 – Ruptured colon in a pygmy goat with *Oesophagostomum* sp worms visible

Sixty neonatal lambs were reported to have died from a group of 600 twin bearing Cheviot ewes in lamb to a Romney tup. Equivalent losses were not seen in any other group, all of which were managed similarly. The ewes had been fed concentrates from scanning until one week before lambing when they were turned onto better grass with access to feed buckets. A mineral drench had been given four-to-six weeks earlier. There had been no issues on the same ground in previous years, and the same tups had been used. The ewes were thought to have sufficient milk and no mastitis was reported. Often one lamb from a set of twins appeared hungry or mis-mothered. Four lambs were submitted and there was no evidence of ticks or diarrhoea. Three of the four were considered light for twin lambs weighing 2.2, 2.4 and 2.9kg. The brown fat had been metabolised in all cases and only lamb 1 had any evidence of milk within the abomasum. The rumen and abomasum of lamb 2 were impacted with grass and the abomasums of lambs 3 and 4 were empty. ZST testing was possible in three cases with hypogammaglobulinaemia confirmed in two (6.1 and 10 units; reference range >14 units). The findings were consistent with starvation/ hypothermia and it was suggested that the abrupt change in feeding so close to lambing may have adversely affected colostrum/milk supply. (S54000254)

A Cheviot mule flock lambing from mid-March reported 25 to 35 per cent lamb losses with both stillborn and weak lambs seen. Lambs born to twin-bearing ewes were most often affected, and some displayed a tremor. The mean BOHB result from five twin-bearing ewes was 3.25 mmol/l with a mean of 1.25 mmol/l for five triplet-bearing ewes (reference range 0-1 mmol/l). The latter were receiving a small amount of concentrate feed while the twin-bearing ewes had access to energy blocks and treacle. Supplementary feeding had been reduced in response to oversized lambs in spring 2024. Inadequate dam nutrition was considered to be the cause of the losses. Hypoglycaemic encephalopathy can be seen in lambs born to energy deficient dams and was the likely cause of the neurological signs reported.

Reproductive tract conditions

A flock of 760 ewes experienced an abortion storm with 46 abortions in one group of 240. The ewes were EAE vaccinated and on a stubble field with access to a grass run back. Supplementary feeding was via a snacker, and water was available in troughs and from a burn. Aborted ewes were not ill and standard investigations found no evidence of EAE, toxoplasma, campylobacter, salmonella, Border Disease or SBV. Pure growths of a range of bacteria were isolated from foetal stomach contents including *Escherichia coli, Bibersteinia trehalosi* and *Streptococcus uberis*. Six aborted ewes were retrospectively blood sampled and were all negative for Q fever with two pools of three PCR positive for *Anaplasma phagocytophila* the cause

of tick-borne fever (TBF). Four foetal spleens also tested positive for *A phagocytophila*. Further investigation by the practice vet revealed that raw human sewage was draining into the burn from the septic tank of the adjacent property. Water samples were collected and extremely high levels of *E coli, Bacillus cereus/thuringiensis, Yersinia intermedia* and *Aeromonas bestiarum* were identified. It was proposed that high bacterial challenge combined with possible immuno- suppression associated with TBF was the cause of the abortion storm.

Musculo-Skeletal conditions

Two, 11-month-old Texel cross ewe hoggs were submitted from a group of 30 for investigation of ill thrift and ataxia. Affected animals were reported to stop growing with muscle loss noted over the hind quarters and progressive deterioration over a period of six to eight weeks. Administration of NSAIDs temporarily ameliorated the clinical signs. Both submitted sheep were reluctant to stand and appeared weak on all four limbs with fine muscle tremors. Varying degrees of valgus in the metacarpal and metatarsal phalangeal joints were noted. No joint effusions or neurological deficits were evident, and no significant lesions were detected on postmortem examination. However, splitting of the long bones revealed multifocal thickening of the physis. Histopathology detected multifocal retention of hypertrophic chondrocytes beneath the physis and marked irregularities of the growth plates consistent with a diagnosis of rickets. Vitamin D deficiency is the most common cause with rapid growth in lambs grazing good quality grass with no supplementary feeding during winter most at risk.

A "short scrotum castrated" Romney lamb accidently mated 500 ewes over a period of three months. Congenital limb deformities were present in around 80 per cent of the progeny with a quarter requiring euthanasia due to the severity of the deformity. Two lambs were examined postmortem both of which had bilateral carpal arthrogryposis. A valgus deformity of the distal forelimbs was also noted in one. The brains and spinal cords were unremarkable. Screening for both border disease and Schmallenberg virus proved negative. No similarly affected lambs were born to the stock rams. A similar limb deformity has been reported in Suffolk lambs which was shown to be autosomal recessive.[1] In the current case it is likely that a high incidence of in-breeding has accounted for the large number of affected lambs.

Nervous system disorders

A neonatal lamb was euthanased to investigate issues with small eyes. The dam was a Texel cross ewe and the exlana sire had produced offspring with small eyes for three consecutive years. The palpebral opening measured 1cm in length compared to 1.8cm in a normal lamb, and the eyeball weighed 1g compared to 3.5g. Histopathology revealed that the anterior chamber had been replaced with fibrous tissue and occasional developmental abnormalities were noted in the retina. The findings were consistent with primary microphthalmia with lens agenesis and anterior segment dysgenesis. A sample of spleen was tested for the mutation that is known to cause microphthalmia in Texel sheep[2] however this proved negative. It was advised that the tup should not be used again and that this may be a unique hereditary issue rather than a wider breed problem.

MISCELLANEOUS

A 15-year-old alpaca was submitted after being discovered dead in the field surrounded by regurgitated food. Postmortem examination detected mucosal ulceration affecting the proximal two thirds of the oesophagus. There were a number of large diameter circular ulcerative lesions with a proliferative surface of

soft white papillae-like projections on the mucosa of C1 (Fig 4). A 5 to 7 cm diameter firm cream coloured irregularly shaped mass was identified adherent to the uterine body. Histopathology revealed an aggressive squamous cell carcinoma of C1, explaining the regurgitation and secondary oesophageal ulceration. The carcinoma could be spontaneous given the animal's age, but prolonged exposure to dietary carcinogens such as bracken fern, can predispose to forestomach neoplasia. The uterine mass was a benign leiomyoma and considered incidental.

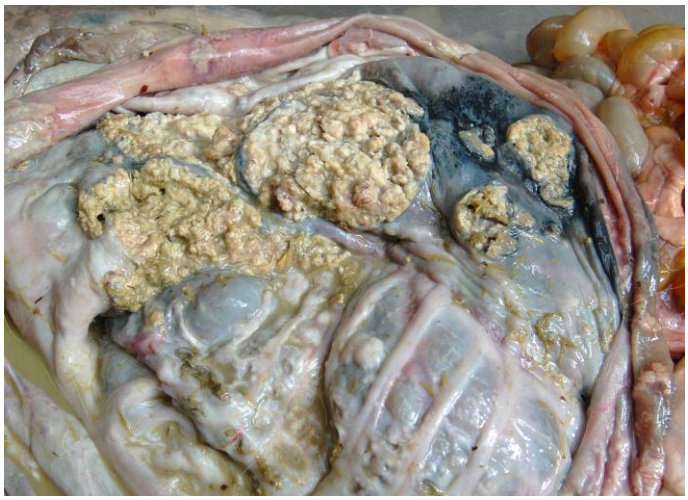

Figure 4 – Squamous cell carcinoma affected C1 in an alpaca

References:

1 - Doherty ML, Kelly EP, Healy AM. *Et al.* (2000) Congenital arthrogryposis: an inherited limb deformity in pedigree Suffolk lambs. *Vet Rec*; *146*(26): 748-53

2 - Becker D, Tetens J, Brunner A. *et al.* (2010) Microphthalmia in Texel sheep is associated with a missense mutation in the paired-like homeodomain 3(PITX3) gene. *PloSOne:* 5(1): e8689.doi:10.1371/journal.pone.0008689