

OVERVIEW

- **Arthrogryposis and torticollis consistent with in utero Schmallenberg virus infection in a calf born to an imported heifer.**
- **Congenital goitre and alopecia in a salers calf.**
- **Swaledale encephalopathy in lambs from five flocks.**

GENERAL INTRODUCTION

The mean temperature for May was 1.2 °C below the long-term average, with more frosts than usual. Scotland as a whole had 130 per cent of average rainfall, with more than double the average in some eastern parts but below average in the west. Sunshine totals were 80 per cent of average.

DISEASE ALERTS

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

- **Parasitic pneumonia in cattle**
Purchased cattle, unvaccinated calves in their first grazing season and older cattle with a limited previous grazing history are at highest risk of parasitic pneumonia. Any outbreaks where the response to treatment is poor should be investigated further and discussed with the relevant pharmaceutical company. Under-dosing can be an issue especially when treating heavier cattle.
- **Spironucleosis in pheasant poults**
Outbreaks of disease can occur shortly after the transfer of birds from rearing to release pens and are triggered by the stress of handling, transportation and introduction to a new environment. Pens that have been used in previous years are higher risk. Affected birds are dull, diarrhoeic and can lose weight rapidly.

CATTLE

Nutritional and metabolic disorders

A six-year-old Ayrshire cow became anorexic with milk drop and malaise three weeks after calving. A ketotic smell was noted and blood calcium and phosphorous levels were low, but there was little response to treatment with corticosteroids, multivitamins, calcium and phosphorous. After being relatively stable for a week the cow then deteriorated rapidly and died. Externally the cow did not appear excessively fat but postmortem examination revealed a large amount of intra-abdominal fat. The liver was swollen, greasy and pale with petechial haemorrhages on the serosa. Sections of tissue floated in water and the findings overall were consistent with a diagnosis of severe hepatic lipidosis. A vitreous humour beta-hydroxybutyrate result of 10.7 mmol/l confirmed significant acetonemia. A 25 x 12 cm diameter firm mass with a necrotic centre was detected within the fat adjacent to the pylorus. This was restricting pyloric outflow and was the likely cause of the anorexia. Histopathology confirmed that the mass was an area of fat necrosis.

Parasitic diseases

A group of 10 beef heifers at grass that were in poor condition after winter were reported to be losing weight and two developed profuse watery diarrhoea. Blood samples were submitted and pepsinogen results of 4.9 and 3.5 iu/l (reference range < 1.5 iu/l) were consistent with a diagnosis of abomasal parasitism due to ostertagiasis.

Generalised and systemic conditions

A three-year-old suckler cow was submitted for investigation of sudden deaths associated with one field. Three cattle had been found dead in the month since turnout and several sheep and a donkey had also died. Inspection of the field failed to detect any potential source of toxins. The carcass was markedly autolysed despite being examined within 12 hours of death. Chemosis and vulval oedema were noted and two pale firm areas surrounded by a zone of haemorrhage were found in the liver. A fluorescent antibody test on tissue from the liver lesion proved positive for *Clostridium novyi* confirming a diagnosis of Black disease. Clostridial vaccination and the postmortem examination of any further animals to die in this field were advised.

A ten-week-old suckled calf was treated for diarrhoea but became recumbent and died. On-farm postmortem examination detected haemorrhagic intestinal contents, oedematous intestines and enlarged lymph nodes. Only

50 coccidial oocysts per gram were detected in caecal contents however histopathology of the large intestine identified a chronic and severe typhlocolitis with entrapped coccidial oocysts consistent with a diagnosis of coccidiosis. Prominent perivascular aggregations of lymphocytes were detected in the renal cortex, lung and brain raising the suspicion of malignant catarrhal fever which was confirmed by PCR detection of ovine herpes virus-2 DNA in kidney tissue. The calf had been housed since birth and, as this period coincided with lambing time, periparturient ewes were considered to be a potential source of infection.

Alimentary tract disorders

A one-week-old suckled calf became lethargic and died one week later after failing to respond to treatment with antibiotics, vitamins and corticosteroids. Small nodular lesions overlying ulcers were found in the distal oesophagus. The rumen contained grey liquid digesta with a pH of 4.4, and areas of ulceration were present on both the rumen and abomasal mucosae (Fig 1). Histopathology confirmed a fungal rumenitis and abomasitis and *Candida albicans* was cultured from the oesophagus. The dam was described as highly strung and a ZST result of 3 units (reference range >20 units) indicated a failure of maternal colostral antibody transfer. The primary problem was not clear in this case. Rumen acidosis secondary to rumen drinking will predispose to fungal infection, however fungal infection secondary to antimicrobial therapy would be an alternative explanation. The lesions will have impaired the functioning of the oesophageal groove perpetuating rumen drinking and ensuring a terminal outcome.



Figure 1 – Fungal abomasitis in a ten-day-old suckled calf

Reproductive tract conditions

A dairy farm imported 12 heifers from Germany in mid-April. One month later an assisted calving produced a calf with clinical signs of torticollis and arthrogryposis. An on-farm postmortem examination was performed, samples were submitted to SRUC VS. Seroconversion to Schmallenberg virus (SBV) was demonstrated in the dam. Histopathology of the calf brain and spinal cord identified micromyelia with a marked reduction in the number of ventral horn neurones and multifocal absence of ependyma, including within the mesencephalic aqueduct, with gliosis in the subventricular neuroparenchyma. These lesions are typical of the consequences of in utero teratogenic orthobunyaviral (Schmallenberg) infection towards the end of the risk period of 30 to 150 days gestation. SBV RNA was not detected in the unfixed CNS (cerebellar) sample submitted but this does not preclude the diagnosis.¹ The dam of the affected calf would likely have been infected with SBV between September 2020 and January 2021 and would not have been viraemic at the time of importation. In addition, April was considered to fall within the vector free period with lower than average temperatures. Overall the importation of these heifers was considered to represent a negligible risk of introducing SBV to the area. Screening of blood samples from the remainder of the imported group revealed the presence of antibodies to SBV in a further two animals one of which originated from the same holding as the dam of the deformed calf.

Musculo-Skeletal conditions

A dairy herd reported ongoing issues with lameness in four- to six-week-old calves most of which recovered after a prolonged course of antibiotics. The herd had a history of *Mycoplasma bovis* infection and there had been a recent increase in pneumonia cases. A six-week-old Aberdeen Angus-cross bull calf was euthanased for investigation of the problem. It had been lame for three days and treated only with NSAIDs. Septic arthritis was confirmed in the left shoulder (Fig 2) along with evidence of pneumonia. A pure growth of *Streptococcus ruminantium* (previously *S suis* serotype 33) was isolated from the joint. This has been reported as a cause of septic arthritis in calves and has also been isolated from cases of pneumonia and endocarditis.² Lung cultures were sterile and histopathology confirmed chronic bacterial pneumonia however no evidence of infection with *M bovis* was detected. As *S ruminantium* can be isolated from the tonsils of healthy cattle focussing on the hygiene of stomach tubes and feeding equipment was suggested as a means of trying to prevent further cases.



Figure 2 – Septic arthritis due to *Streptococcus ruminantium*

Skin diseases

A neonatal shorthorn cross calf was euthanased and submitted to investigate areas of hair loss over the dorsum, distal limbs and head (Fig 3). A similar calf had been born in 2020 but no testing was carried out. Epidermolysis bullosa was suspected and confirmed on histopathology. This revealed extensive absence of epidermis often associated with haematoidin and haemosiderin pigments in the dermis indicating previous episodes of haemorrhage linked to separation of overlying epidermis. In sections where epidermis remained there was multifocal separation from the dermis. The epithelia of the tongue and oesophagus were also affected. Further history is being sought regarding the breeding of the calf.



Figure 3 – Epidermolysis bullosa

A 140 cow suckler herd submitted a stillborn three-quarter salers-cross calf to investigate the cause of congenital alopecia. A similar calf had been born to a different cow two years previously. The calf was believed to be full term but weighed only 16 kg with a crown rump length of 71 cm.

Hair was only present around the eyes and on the tail, ears, forehead and distal limbs. The thyroid was enlarged (30g) and oedematous (Fig 4). Histopathology confirmed diffuse hyperplastic goitre with no colloid present and the thyroid iodine content was very low at 70 mg/kg dry matter (DM) (reference range > 1200 mg/kg DM). Hair follicles were present in usual numbers but were under-developed without hair shaft formation. Similar cases have been recorded in salers cattle whereby they fail to synthesise thyroid hormones despite adequate dietary iodine.³ Hypothyroidism can also delay myelination and hypomyelination was an additional finding in this case.



Figure 4 – Hyperplastic goitre in a Salers calf with congenital hypothyroidism

SMALL RUMINANTS

Nutritional and metabolic disorders

A three-week-old Texel cross lamb was noted to be off colour before being found dead the next day. Postmortem examination revealed free blood in the abdomen originating from a 9 cm tear in the liver. The liver appeared large and pale and histopathology detected diffuse macro- and microvesicular hepatic vacuolation consistent with widespread lipid accumulation. Ovine white liver disease was considered as a differential diagnosis but the lamb was unusually young and ceroid pigment was not detected. Despite this liver analysis confirmed low cobalt levels; <0.02 mg/kg DM (reference range \geq 0.06 mg/kg DM). Other differential diagnoses included toxicity, which was deemed unlikely, a metabolic defect or a congenital enzyme deficiency.

Generalised and systemic conditions

A flock reported the death of 15 lambs from a group of 60 over a short period and the carcass of a three to four-week-old Texel lamb was submitted for investigation of

the problem. A fibrinous pleurisy, pericarditis and peritonitis was detected on postmortem examination. An organism suspected to be a non-haemolytic strain of *Mannheimia haemolytica* was isolated and considered to be the cause of death. Concentrate feed had been introduced one week earlier, the rumen pH was 4 and this was considered to have predisposed to the outbreak. A flock of Shetland sheep reported ataxia and neurological signs in neonatal lambs with 30 per cent of the lamb crop dying at one to two days of age. Lambs appeared healthy at birth and colostrum management was good. Postmortem examination of a typical case revealed a pericardial effusion containing a fibrin clot but no other abnormality. Epsilon toxin was detected in small intestinal contents and histopathological examination of kidney and brain confirmed lesions consistent with a diagnosis of neonatal pulpy kidney (clostridial enterotoxaemia type D). This condition is preventable by clostridial vaccination of ewes which was advised for the future.

Nervous system disorders

Swaledale encephalopathy was suspected as the cause of neurological signs in two-week-old swaledale lambs from five separate flocks. Affected lambs were reported to be ataxic with rapid clinical deterioration. There were no significant findings on postmortem examination. Screening for Border disease virus and Louping Ill virus was carried out in one flock and proved negative. Neuropathology revealed a polioencephalopathy/ interface encephalopathy typical of Swaledale encephalopathy in all cases.⁴ In flock one all the affected lambs were known to have been sired by a new tup. Information on breeding history was not available for all flocks however this condition is believed to be hereditary.

PIGS

Generalised systemic diseases

The carcasses of three, eight- to ten-day-old piglets from different litters were submitted to investigate an ongoing problem with a low level of sudden deaths in neonatal piglets from a 560-sow herd. Postmortem examination findings included marked distention of the small and large intestines with liquid yellow contents but no evidence of diarrhoea. A suspect isolate of *Clostridium difficile* cultured from the small intestines of one piglet was tentatively identified as such by MALDI-TOF. *Clostridium difficile* antigen was detected by ELISA in the faeces of two. Histopathology identified widespread acute bacterial necrotising colitis in one case. When considered in combination the findings were highly suggestive of enterotoxaemia associated with *Clostridium difficile*. No other neonatal enteropathogens were identified, however

one piglet was diagnosed with secondary septicaemia due to *Mannheimia varigena*, an organism occasionally associated with disease in cattle and pigs. Clostridium difficile-associated disease (CDAD) is most common in neonatal piglets between one and seven days of age and spores shed in sow faeces are the primary source of infection. Good colostrum management and a clean environment are essential in preventing this condition. All piglets received an injection of tulathromycin as a prophylactic measure against joint ill. However, such antimicrobial prophylaxis may encourage overgrowth of *C. difficile* by disrupting the natural gut flora. The client was encouraged to submit further cases with an emphasis on culturing *Clostridium difficile* on selective media for potential autogenous vaccine production.

BIRDS

A dead buzzard (*Buteo buteo*) submitted for routine Avian Influenza screening was found to be thin with a prominent keel and no fat stores. Multiple spherical creamy-white abscesses up to 0.5 cm in diameter were found scattered throughout the liver (Fig 5). A Ziehl–Neelsen stained impression smear of liver tissue revealed acid-fast bacilli supporting a diagnosis of avian tuberculosis. Avian tuberculosis affects a wide range of bird species and was also confirmed as the cause of chronic wasting in a backyard khaki Campbell duck. Postmortem examination findings in this case were atypical and included multiple abdominal masses of varying colour, size and shape plus countless, dark 1 mm oval structures attached the ovary by a cord of fibrous tissue. Adenocarcinoma of the oviduct was suspected, but histopathology revealed multiple granulomatous lesions with prominent epithelioid macrophages and giant cells consistent with tuberculosis.



Figure 5 – Avian tuberculosis lesions in the liver of a buzzard

MISCELLANEOUS

Wild animals

A female mountain hare (*Lepus timidus*) was submitted after being found moribund prior to death. The carcass was thin and numerous ticks and lice were present. The strongyle egg count was high (10,950 eggs per gram) and considered significant. There was no evidence of louping ill and histopathological examination of tissues found only mild changes in the liver and kidneys suspected to be secondary to the intestinal parasitism.

References:

- 1 De Regge N, van den Berg T, Georges L, Cay B. Diagnosis of Schmallenberg virus infection in malformed lambs and calves and first indications for virus clearance in the fetus. *Vet Microbiol* 2013; 162(2): 595-600
- 2 Okura M, Maruyama F, Ota A *et al.* Genotypic diversity of *Streptococcus suis* and the *S. suis*-like bacterium *Streptococcus ruminantium* in ruminants. *Vet Res* 2019; 50: 94-109
- 3 Watson PJ, Scholes SF. Congenital goitre and alopecia in pedigree Salers cattle. *Vet Rec* 2010; 166(1): 29-30
- 4 Scholes SFE, Higgins RJ, Holliman A *et al.* Subacute symmetrical necrotising encephalopathy in young lambs. *Vet Rec* 2007; 160(22):775

Perforated Abomasal Ulcers In Suckled Calves

SRUC VS recorded 102 diagnoses of perforated abomasal ulcers in suckled calves between January 2011 and June 2021. Cases were most commonly seen between May and July and in calves between one and two months-of-age (Figs A and B; data for dairy calves included for comparison). There is no evidence of second peak following autumn calving. 80.4 per cent of calves were in good body condition with only 2 per cent described as thin. Males were slightly over-represented accounting for 56.7 per cent of cases.

Losses due to perforated abomasal ulcers are usually isolated incidents and the majority of affected calves are found dead (Fig C). The diagnosis is straightforward but, as the aetiology is not understood, it is difficult to provide advice on how to prevent further cases. Suggested risk factors are listed below but, even when identified, proving cause and effect is difficult.

- Concurrent disease or stress.
- Mechanical trauma due to abnormal contents such as grit/sand or hair balls.
- Viral infection such as BVD.
- Trace element deficiencies including vitamin E, selenium and copper.
- Bacterial or fungal infection.
- Hyperacidity.

Further review of postmortem examination reports found that seven of the calves had pneumonia (described as mild/incidental in four), and five had evidence of navel ill and/or liver abscesses. Hair balls were detected in six and the presence of sand/silt was noted in the abomasal contents of four. Twenty-four calves were screened for BVD virus with negative results. These findings suggest that these proposed risk factors are of minimal or no importance. Liver trace element analysis was rarely carried out but, of the five calves tested, selenium results were low in all cases. These numbers are too small to draw any conclusions, but further investigation may be warranted.

It has been suggested that clostridial species bacteria have a role in the pathogenesis of abomasal ulceration and perforation. Bacteriology and/or fluorescent antibody testing was carried out in around two thirds of cases and results do not support any link between the two. In addition, histopathology failed to detect any evidence of clostridial abomasitis in the 41 cases examined. A primary bacterial abomasitis was suspected in two cases implying that infection is also of little importance overall. *Sarcina* species bacteria were observed histologically in 15 cases indicating an abnormal abomasal bacterial flora. *Sarcina* sp proliferate when there is an excess of fermentable carbohydrate within the abomasum. This could result if a degree of tympany develops following ingestion of a large volume of milk. *Sarcina* sp. ferment sugars producing carbon dioxide which can prolong tympany potentially reducing mucosal perfusion. Other products of fermentation such as acetate, hydrogen and ethanol can cause chemical damage to the mucosa and may reduce the pH which further slows abomasal emptying. It is hypothesised that a repeated cycle of mucosal damage triggered by large intakes of milk may predispose to abomasal ulceration and perforation. Providing advice on how to reduce the wastage caused by this condition remains problematic as the initiating cause remains unclear in many cases.

Fig A: SRUC VS diagnoses of perforated abomasal ulcers in calves up to six months of age 2011 to 2021

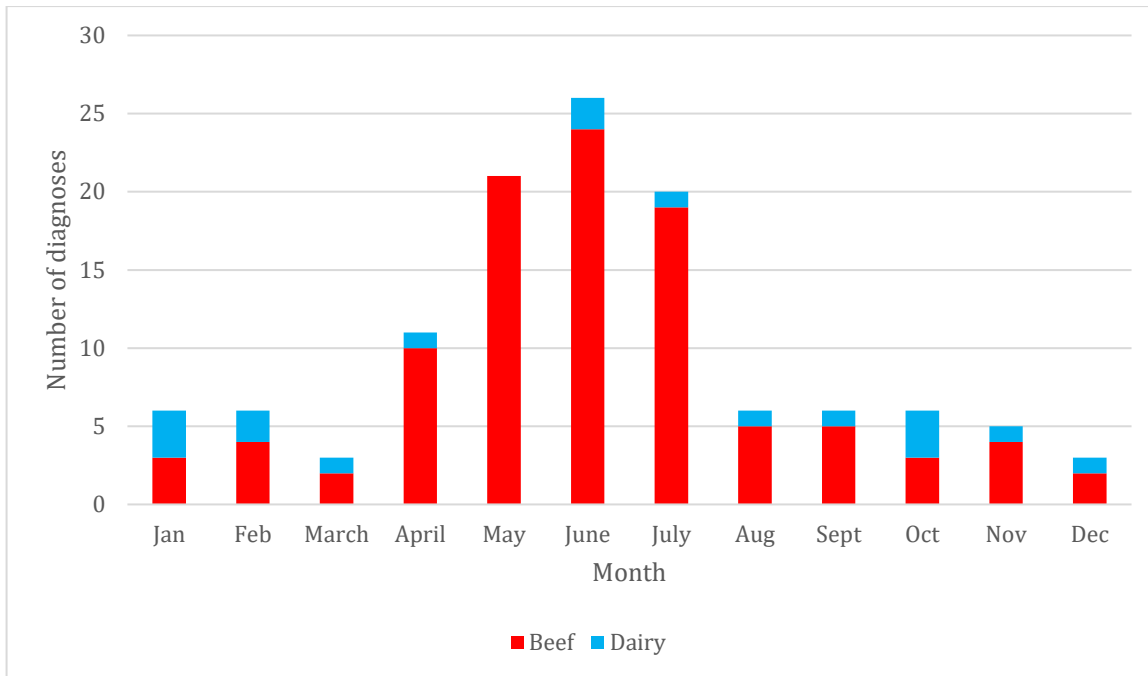


Fig B: Age of calves diagnosed with perforated abomasal ulcers 2011 to 2021

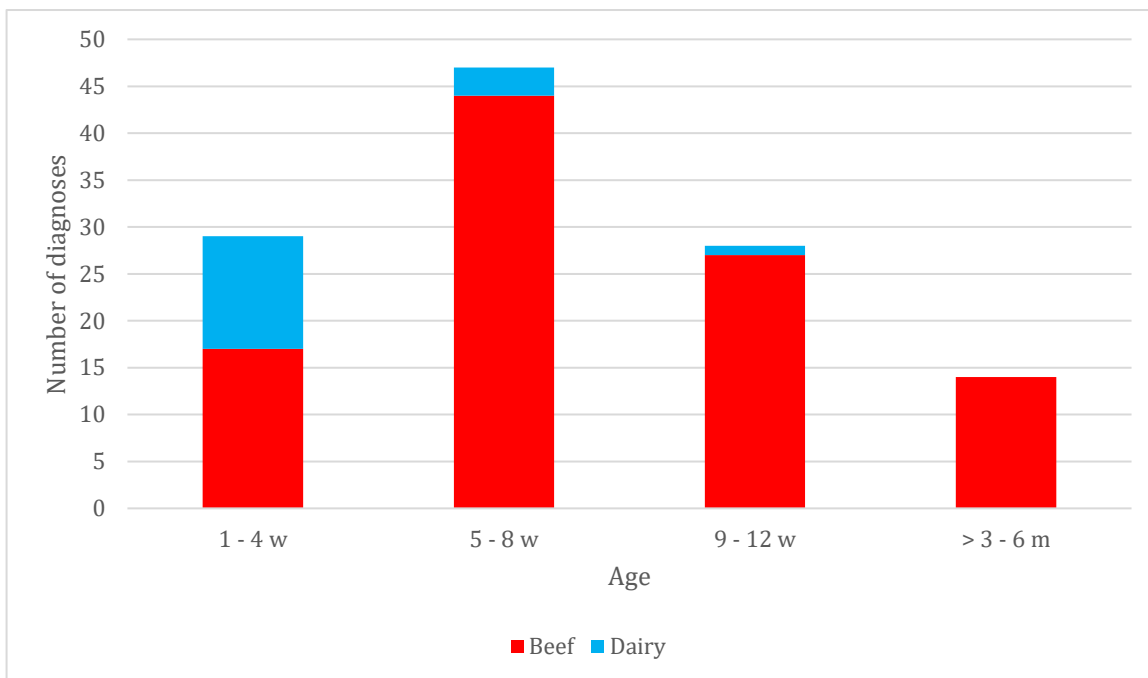


Fig C: Ruptured abomasal ulcer in a four week old suckler calf

