SRUC Veterinary Services Monthly Report for October 2022

OVERVIEW

- **Fog fever as a cause of suckler cow deaths.**
- **Abomasal emptying defect in a Texel tup.**
- ***Mycoplasma gallisepticum* causing "bulgy eye" in pheasant poults.**
- **Multiple deaths due to hepatic lipidosis in an alpaca herd.**

GENERAL INTRODUCTION

Monthly mean temperatures were 1.5 °C above the average for October. Rainfall was well above average in parts of the west and south-west, and slightly lower than average in the north-east, amounting to 116 per cent of average for Scotland as a whole. Sunshine was below average in most western areas, but it was brighter in the east, giving 93 per cent of the long-term average overall.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in January 2022. Given similar climatic and production conditions, they could also be important this year.

- **Schmallenberg virus as a cause of deformed lambs**
  Congenital deformities including arthrogryposis, vertebral column and central nervous system abnormalities can occur following infection of naïve ewes with Schmallenberg virus (SBV) during the second month of gestation. There are many other causes of foetal deformities with only a single confirmed case of SBV in Scotland during 2022. The timing of this suggested that SBV transmission from midges had occurred during autumn 2021.

- **Yersiniosis in deer calves**
  Yersiniosis causes acute diarrhoea and death typically in the winter months. It most often involves groups of four to eight-month-old deer with up to 20 per cent morbidity. *Yersinia* spp are found in soil and in the intestinal tracts of carrier animals which include both livestock and wildlife. Outbreaks are usually precipitated by stressors such as weaning, transport, bad weather or inadequate nutrition.

CATTLE

Parasitic diseases
A group of 100 stirks was handled for treatment with a levamisole/oxyclozanide drench and the best 25 animals were selected to run as a separate group. Three weeks later barley was added to the concentrate ration they had been receiving all summer. One week after this two animals were found dead, two were recumbent and one was diarrhoeic. The others rapidly lost condition over the next 48 hours and became weak and reluctant to stand. The carcase of an eight-month-old wagyu cross bullock was submitted to investigate the possibility of rumen acidosis however the rumen content was dry with plentiful fibre and no visible cereal grains. The abomasal mucosa was thickened and necrotic (Fig 1) and the intestinal contents were liquid. The strongyle egg count was 2000 eggs per gram (epg) and *Bibersteinia trehalosi* and *Trueperella pyogenes* were isolated from the abomasum. Histopathology confirmed severe abomasal parasitism with bacterial invasion and necrosis secondary to increased pH as a consequence of *Ostertagia ostertagi* damage to the gastric glands. The rest of the group improved following housing and anthelmintic treatment. The other 75 stirks were unaffected suggesting that the smaller group had inadvertently been stocked on a field with high infectious larval challenge.

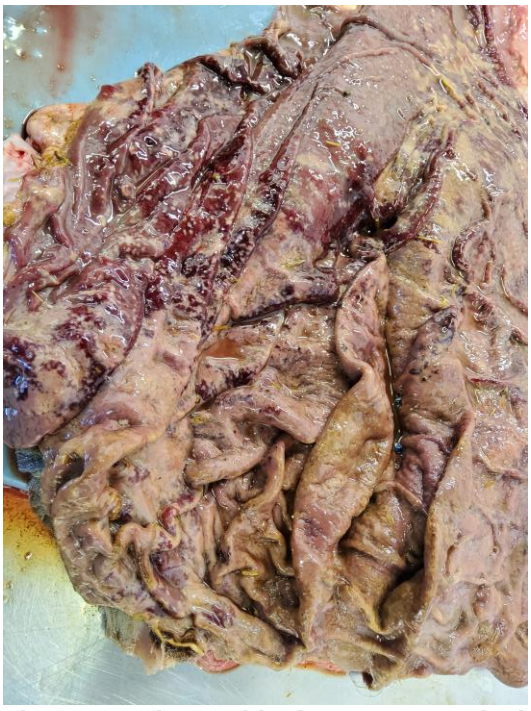

Figure 1 – Abomasitis due to ostertagiasis and secondary bacterial infection

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Alimentary tract disorders
A calf rearing unit sourcing animals from two separate dairy herds reported that small numbers of calves were reluctant to drink and regurgitated milk if stomach tubed. Three of six affected calves had been euthanased and the other three were chronically ill thriven. The seventh affected calf, a three-week-old Aberdeen Angus cross heifer was euthanased three days after it arrived in order to investigate the problem. Postmortem examination detected a severe oesophagitis and inflammation along the edges of the rumen pillars (Fig 2). Histopathology revealed that the changes were subacute to chronic indicating that they had occurred on the farm of origin and were most likely associated with either a physical or chemical insult. It was recommended that all stomach tubes should be checked for damage and the disinfection protocol for calf feeding equipment reviewed.

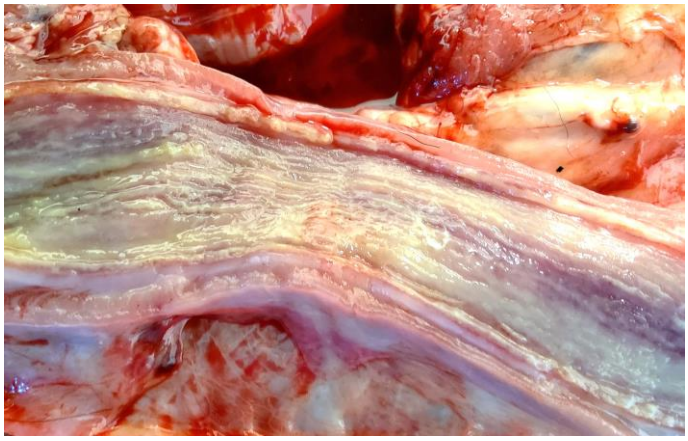

Figure 2a

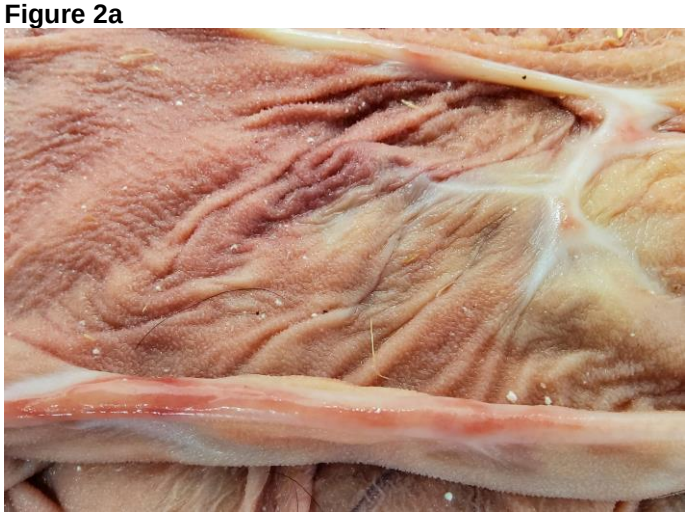

Figure 2b

Figure 2 – Oesophagitis (a) and reddening of the rumen pillars (b) in a three-week-old Aberdeen Angus cross calf

Respiratory tract disorders
Five simmental cross cows from a group of 40 with calves at foot developed acute respiratory distress and two died. The lungs failed to collapse when removed from the thorax and showed widespread emphysema and interlobular oedema typical of interstitial pneumonia. Thick mucus and froth were present in the airways and there was a moderate pericardial effusion. Histopathology described severe proliferative pneumonia with extensive type 2 pneumocyte hyperplasia and multifocal areas of hyaline membrane formation. No lungworms were seen on postmortem examination and histopathology ruled out the possibility of re-infection syndrome. No respiratory viruses were detected by PCR. The group had been moved to a silage aftermath two weeks before the onset of clinical signs. The history of dyspnoea with minimal coughing in beef cattle following introduction to better grazing in the autumn was considered consistent with a diagnosis of fog fever and the animals were removed from the pasture.

SMALL RUMINANTS

Nutritional and metabolic disorders
A two-month-old boer kid described as dull and anorexic since weaning was found dead and submitted for postmortem examination. The carcase was thin with pulmonary oedema and marked serous body cavity effusions. Pale discolouration of the papillary muscles of the left ventricles was observed (Fig 3) and histopathology confirmed polyphasic cardiac myofibre degeneration, necrosis, fibroplasia and mineralisation consistent with a diagnosis of nutritional myopathy. Neither liver vitamin E nor selenium concentrations appeared low, and it was proposed that a pre-existing deficiency had been corrected after the initial insult which was believed to have occurred at least three weeks prior to death. Although no other animals were clinically affected at the time the findings had implications of the rest of the herd.

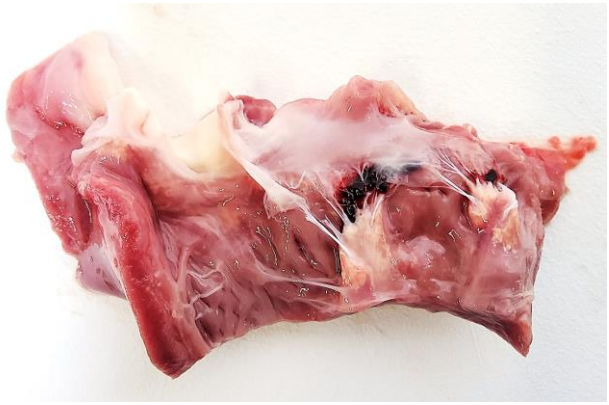

Figure 3 – Myocardial pallor due to nutritional myopathy in a boer kid

Generalised and systemic conditions
Systemic pasteurellosis due to *Bibersteinia trehalosi* was diagnosed in four north-east flocks during October. Homebred animals that had received a full course of a pasteurella containing vaccine were affected on two holdings. Bought in animals with unknown vaccination history were involved in the other two outbreaks. Postmortem examination findings indicated septicaemia and individual carcases showed necrotic lesions in the pharynx, oesophageal ulceration or miliary foci in the liver all of which can be indicative of *B trehalosi* infection. *B trehalosi* was isolated from at least one tissue in all cases confirming this diagnosis. A range of possible predisposing factors were identified including recent gathering, hyposelanosis and parasitic gastroenteritis.
A less common cause of septicaemia was diagnosed when the second lamb to die from a group of 120, four-month-old Scottish blackface lambs was submitted for postmortem examination. The lambs were unvaccinated and had been set stocked following weaning three weeks earlier. Enlarged lymph nodes, splenomegaly and oedematous lungs were the main findings and pasteurella septicaemia was suspected. However, *Erysipelothrix rhusiopathiae* was cultured from the liver and spleen. Histopathology confirmed a bacteraemia with purulent foci in the myocardium and occasional foci of neutrophils in skeletal muscle.

Alimentary tract disorders
An eight-month-old Texel tup became bloated and recumbent in the 24-hour period prior to death. It was the only animal affected from a group at grass with access to a small quantity of supplementary concentrate feed. Postmortem examination found extensive ventral subcutaneous oedema, congested lungs and a pale, autolysed liver. The abomasum was markedly dilated and filled the whole ventral and right side of the abdomen (Fig 4). It contained 14.5 kg of ingesta and a further 7 kg of material was removed from the rumen. The contents of both had a similar fibrous appearance. The findings were consistent with a diagnosis of abomasal emptying defect the aetiology of which is unknown. Necrotic neurons have been identified in the celiaco-mesenteric ganglia of six from nine affected sheep suggesting that it is a dysautonomia.[1]

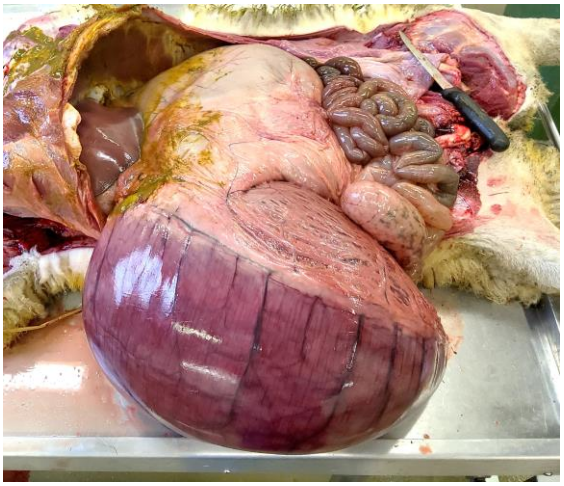

Figure 4 – Abomasal emptying defect in a Texel tup lamb

Two Texel gimmers developed signs of abdominal pain within four hours of undergoing laparoscopic artificial insemination. One died and the other was euthanased. Postmortem examination revealed a clockwise mesenteric torsion in both cases with dilation and congestion of the intestines. The ewes had been starved overnight and reduced gut fill combined with administration of a sedative was postulated to have altered gut motility. Manoeuvring on and off the cradle and the possibility of rolling or falling during recovery were suggested as additional risk factors.

PIGS
Alimentary tract disorders
A rearing unit reported poor growth rates and pallor in several batches of pigs and euthanased three typical 14-week-old growers for investigation of the problem. In both cases the pars oesophagea of the stomach was markedly thickened and fibrosed as a result of chronic ulceration (Fig 5). Ulceration of the distal oesophagus was also detected as a consequence of gastric reflux. There was scant, watery blood suggesting that significant haemorrhage had occurred, and histopathology confirmed extramedullary haematopoiesis in the spleen. Gastric ulceration in pigs can be associated with factors that cause increased fluidity of the stomach contents including very fine particle size or pelleting of the feed. Any interruption in feed uptake caused by events such as mixing of groups, bullying, restricted trough space or concurrent disease may also increase the risk. No predisposing or concurrent infectious diseases were detected and analysis of feed to determine “fineness” was advised.

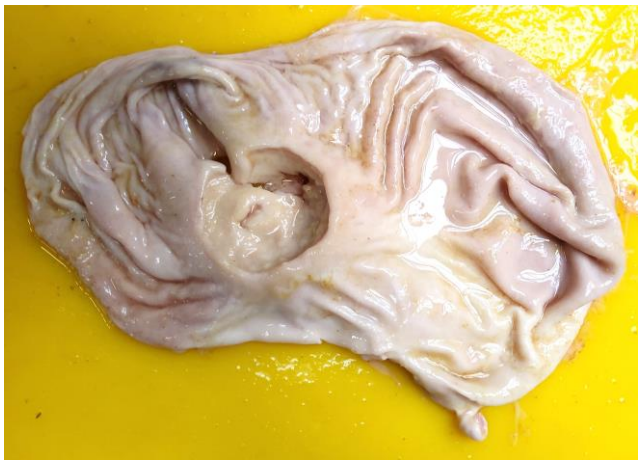

Figure 5 – Thickening and fibrosis of the pars oesophagea due to chronic gastric ulceration

BIRDS - Poultry

Two, eleven-week-old turkeys were submitted live to investigate ataxia and deaths in a group of 350 birds where seven birds had been found dead in seven days. They had been bought in five weeks previously and had access to a wooded area and a straw bedded shed. Both birds were able to stand and move around but exhibited intermittent torticollis and collapse. Postmortem examination was not diagnostic, and bacteriology produced growths of *Escherichia coli* from the lungs and brains. Histopathology revealed a subacute to chronic fibrinogranulocytic meningitis and vasculitis indicating a bacterial aetiology. *Mycoplasma gallisepticum* was suggested as a possible cause but could not be confirmed. Virulent strains can cause meningeal vasculitis and neurological signs in a small proportion of affected turkeys.

Seven, eight to nine-week-old pheasants were submitted to investigate the cause of swollen eyes (Fig 6) and nasal discharge affecting 100 birds, 30 of which had died. The poults had arrived two weeks earlier and had been turned out into release pens. The infraorbital sinuses were swollen and contained a mucoid and fibrinous exudate. Body condition was poor, and the birds were dehydrated and had failed to feed. *Mycoplasma gallisepticum* was detected by DGGE (denatured gel gradient electrophoresis)/PCR in two of three pools and histopathology confirmed a chronic lymphoplasmacytic and catarrhal conjunctivitis, rhinitis and sinusitis. Respiratory cryptosporidiosis due to infection of the nasal passages with *Cryptosporidium baileyi* was also apparent in two of the birds. Antibiotic treatment was advised to help alleviate the clinical signs of mycoplasmosis, however there is no treatment for respiratory cryptosporidiosis other than optimising hygiene to help control the spread.

Figure 6 – "Bulgy eye" due to *Mycoplasma gallisepticum* infection in a pheasant poult

MISCELLANEOUS

Seven adult alpacas from a mixed age group of 70 died over a two-week period. The owner reported that grass cover had been good throughout the summer and that hay had been available ad lib. Four carcases were examined and found to be underweight (41-55 kg) with little body fat and evidence of hepatic lipidosis which was confirmed on histopathology. Dental disease, focal areas of gastritis and moderate to high nematode burdens were each noted in two or more animals. Despite provision of a daily trace element supplement, liver analysis identified marginal copper and selenium status and low cobalt levels in all animals. Testing of cohorts revealed hypoalbuminaemia in four of six and raised GLDH and/or GGT in four. These results suggested dietary insufficiency with hepatic damage in some of the group. Forage analysis revealed that the hay was poor quality with low ME (metabolisable energy) (7.12 MJ/kg DM) and crude protein levels (34.5 g/kg DM). This was considered to have predisposed to hepatic lipidosis, particularly in those animals with dental disease or crias at foot. While the keepers were experienced, there had been a failure to recognise the decrease in body condition of affected animals. The issues resolved following anthelmintic treatment and dietary change.

References:

Pruden SJ, McAllister MM, Schultheiss PC, O'Toole D, Christensen DE. Abomasal emptying defect of sheep may be an acquired form of dysautonomia. Vet Pathol. 2004 Mar;41(2)