OVERVIEW

- Idiopathic necrotising enteritis in suckled calves.
- Mortality in goats due to haemonchosis and teladorsagiosis.
- Progressive paresis in North Country Cheviot lambs.

Dermanyssus gallinae causing anaemia and death in brahma chicks.

GENERAL INTRODUCTION

The mean temperature for August was 0.5 °C above the long-term average. Across Scotland as a whole sunshine and rainfall were both near normal, with 100 per cent of average rainfall and 96 per cent of average sunshine. Regional differences were apparent with cloudy and wet conditions in the south and central belt.

CATTLE

Parasitic diseases

A group of six-month-old Jersey cross calves were turned out onto a field previously grazed by cattle and sheep. Silage and concentrates were fed both during housing and at grass. Despite this, signs of ill thrift were observed from two weeks post turn out. Two calves were found dead after one month of grazing and submitted for postmortem examination. They had been treated with an ivermectin pour on two days prior to death. Both calves were thin and live Dictyocaulus viviparus were detected in the bronchi. Significant burdens (20,300 and 32,000) of Ostertagia ostertagi were recovered from the abomasums, confirming parasitic gastroenteritis as the cause of ill thrift and death, and suggesting high larval challenge on the field. SRUC VS advised that faecal samples should be collected from other calves in the group two weeks post treatment to check anthelmintic efficacy.

A five-year-old Montbeliarde cross cow was euthanased for investigation of respiratory disease in a group of purchased cattle. Eight animals had been coughing for one month despite treatment with a pour-on anthelmintic product approximately three weeks earlier. The cattle had been housed all year round prior to purchase but had grazed pasture that had previously been rented out for cattle grazing since late spring. Moderate numbers of live Dictyocaulus viviparus were recovered from the trachea and bronchi and histopathology confirmed a severe eosinophilic pneumonia consistent with response to lungworm. The worms measured between 0.9 and 2.9 cm, whereas adult D viviparus are 4 to 8 cm in length. SRUC VS commented that it would be advisable to check the date of anthelmintic administration, to determine whether or not there was evidence of treatment failure. Purchased cattle and a limited previous grazing history are both risk factors for outbreaks of parasitic pneumonia.

Generalised and systemic conditions

A six-week-old Holstein bull calf was euthanased for investigation of an ongoing issue with calf pneumonia. It had been housed in a pen with five heifers as a sentinel animal. Harsh lung sounds, coughing and pyrexia (41.2 °C) were reported prior to submission. Patchy consolidation was detected affecting the cranial, middle and caudal lung lobes on the right and the middle lung lobe on the left. A blood clot was found in the pericardium, which had originated from a 1 cm long defect in the right ventricle (Fig 1). Histopathology confirmed the presence of chronic lung pathology consistent with Pasteurella multocida and Mycoplasma
bovis infection, and the latter was detected in lung tissue by PCR. *Salmonella enterica* serotype Dublin was cultured from the liver and histopathology confirmed a hepatitis with necrotic foci. Overall, the findings indicated that the deterioration of the calf was due to infection with *S* Dublin and that any clinical signs associated with the earlier pneumonia had been missed. The haemorrhage from the heart had originated from an area of acute myocardial necrosis. This was considered likely to be a result of ischaemia secondary to vascular thrombosis associated with salmonella septicaemia.

**Figure 1** – Full thickness defect (arrow) in the right ventricle of a calf due to acute myocardial necrosis secondary to salmonellosis

**Alimentary tract disorders**

Two cases of idiopathic necrotising enteritis (INE) were confirmed in beef herds in north-east Scotland. The diagnosis had been made multiple times in the first herd in recent years. Only one calf was affected in 2020 and it presented with diarrhoea for six weeks prior to death. The calf from the second herd was found dead with no prior signs of ill-health noted. Despite this, ulcers were found on the tongue, hard palate and larynx, and the mucosa of the terminal jejunum, ileum and caecum was thickened and necrotic (Fig 2). Histopathological examination in both cases confirmed a diagnosis of INE. It was noted that affected calves in herd one had consistently low selenium and vitamin E results and malabsorption may have played a role in this. The aetiology of INE has yet to be elucidated.

**Figure 2** – Thickening and necrosis of the small intestinal mucosa in a case of idiopathic necrotic enteritis

**Reproductive tract conditions**

Abortion material from a second lactation Holstein cow which aborted two months before its due date was submitted for investigation after two cows aborted on the same day. The dam showed no other signs of disease, was vaccinated for IBR and BVD and fed a total mixed ration which included straw and pit silage. Culture of foetal stomach contents and placenta produced pure growths of *Listeria monocytogenes* and this was considered to be the cause of abortion. The silage was thought to be the most likely source of infection.

**Nervous system disorders**

A 15-week-old suckled calf was euthanased for investigation of recumbency and nervous signs. It was the only affected calf in the herd of 400 Highland and shorthorn cows. The only abnormalities identified on postmortem examination were myiasis of the perineum, which was considered to be secondary to recumbency and diarrhoea. Neuropathology detected severe laminar necrosis in the cerebral cortex consistent with a diagnosis of cerebrocortical necrosis (CCN) which is an unusual diagnosis in a preweaned calf. Other findings identified in this case included a strongyle count of 400 eggs per gram (epg) and typhlocolitis of unknown aetiology identified on histopathology. The possibility of gut dysbiosis was suggested but it was not clear whether this would be sufficient to predispose to CCN via changes in production of thiamine or thiaminases.
**SMALL RUMINANTS**

**Parasitic diseases**

The carcases of three, three-month-old Shetland cross lambs were submitted to investigate the cause of diarrhoea and multiple deaths in a group of 650 lambs that had been weaned two weeks previously. Uncountable numbers of *Nematodirus battus* worms were recovered in two cases with 10,600 retrieved from the third. Chronic pneumonia associated with *Mannhaemia haemolytica* was also present in all three lambs and likely to have contributed to their poor body condition. Prompt anthelmintic treatment of the rest of the group was advised. The findings suggest that these lambs had not been exposed to sufficient *N battus* challenge earlier in the summer to generate immunity.

A small goat herd reported ongoing issues with rapid weight loss plus scour in some animals. Both adults and youngstock were affected and three deaths had occurred. The carcases of a four-year-old Boer goat and an eight-year-old Angora goat were submitted for postmortem examination. Treatment with an ivermectin drench had been administered one week earlier. Both animals were very thin and appeared anaemic. *Haemonchus contortus* worms were visible in the abomasum of the Boer goat and made up the majority of the 7200 worms recovered, the remainder being *Teladorsagia* sp. The high fecundity of *H contortus* was reflected in the strongyle egg count of 31,350 epg. In contrast 24,600 *Teladorsagia* sp. worms, but no *H contortus*, were recovered from the abomasum of the Angora goat with a further 5200 *Trichostrongylus* sp. from the small intestines. The strongyle egg count in this case was 3150 epg. Further history was obtained which indicated that the goats had been grazing separate areas and this was assumed to account for the different composition of their worm burdens. While it is possible that all three worm species identified could be resistant to ivermectin, it was advised that other explanations for treatment failure, such as underdosing, should be investigated. The Angora goat also tested antibody positive in the caprine arthritis encephalitis (CAE) ELISA. It was advised that all remaining goats and sheep on the holding should be screened for CAE/maedi visna. Due to limited grazing options the decision was taken to house the goats to prevent reinfection with nematode larvae.

**Generalised and systemic conditions**

Enterotoxaemia due to *Clostridium perfringens* type D was diagnosed as the cause of death of four to five-month-old goat kids from two different herds. Contrasting clinical presentations were described with recumbency and opisthotonos reported in the first case and diarrhoea and colic in the second. One kid was unvaccinated and access to concentrate feed was considered to be a possible predisposing factor. The second kid had received a single dose of clostridial vaccine three days prior to being sold and died after two days on the new premises. The stress associated with transport was proposed as a possible risk factor.

**Respiratory tract disease**

A Cheviot mule hogg was treated for suspected pneumonia and euthanased after failing to improve. It was one of a group of 43 hoggs with lambs at foot that had been purchased four weeks earlier. The carcase was very thin and, despite the absence of fluid in the airways, significant lesions consistent with ovine pulmonary adenocarcinoma (OPA) were detected in both lungs. The suspected diagnosis confirmed on histopathology. The seller was unaware that OPA was present in his flock.

**Nervous system disorders**

Several three-month-old North Country Cheviot lambs from a hill flock were observed to develop a progressive paresis. The signs could be induced by handling or stress, and affected lambs remained bright and alert but became recumbent after a short period of handling. Affected lambs gradually lost weight as the condition progressed eventually becoming permanently recumbent. Delayed swayback was suspected, and three lambs were submitted for postmortem examination with no significant findings identified. Histopathology confirmed lesions of upper and lower motor neuronopathy involving neuronal chromatolysis, disorderly Nissl substance and perikaryonal vacuolation of small numbers of neurones mainly in red nuclei. Long fibre tract leucoencephalomyelopathy was present, involving myelinoclastic vacuole formation, reduction in myelin and gliosis particularly in dorsolateral segments of lateral funiculi and medial portions of ventral funiculi, along with involvement of cerebellar peduncles, medial longitudinal fasciculus and spinocerebellar tracts. These findings were dissimilar to delayed swayback and liver copper levels were within the reference range. The history and progressive nature of the condition raised the possibility an inherited defect predominantly affecting motor neurones. Further investigation is ongoing.

**Renal diseases**

Fifty per cent of a group of 120 herdwick lambs were described as ill thrive and 12 animals had died. Diarrhoea was reported and the group had last been wormed one month before. The carcase of a very thin lamb weighing only 9.5 kg was submitted for investigation of the problem. It smelled uraemic, the kidneys were pale and swollen, and the large intestinal contents were
liquid. No evidence of significant current parasitic infection was found and histopathology failed to detect chronic intestinal damage due to coccidiosis. However, histopathological examination found evidence of a significant parasitic abomasitis with luminal nematodes consistent with *Teladorsagia* sp, and it was considered that previous significant worm challenge had induced secondary nephrosis in this case. Liver analysis failed to detect any evidence of trace element deficiency. It was considered unlikely that large numbers of the other poor-doing lambs had also developed nephrosis, but despite that, the prospect of achieving good future daily liveweight gains seemed poor.

**BIRDS**

**Poultry**

A heavy infestation of *Dermanyssus gallinae* mites was detected on examination of six, two to four-day-old brahma chicks submitted for postmortem examination. The history was of increased chick mortality during the last three batches to hatch with almost half of 37 chicks lost. Gross findings consistent with omphalitis in one bird and failure of the chicks to feed were detected in addition to the red mite infestation. Red mites can readily cause death associated with anaemia in poultry, and the small size of these young chicks made them especially susceptible.

**Game birds**

A batch of 225 bought in pheasants experienced dramatic weight loss within a short time of being stocked in release pens. Five died and a further seven were reported to be ill. Homebred birds were unaffected. A similar problem had been seen the previous year and the same pens were in use. Two birds were submitted for examination one of which was very thin and dull with yellow, watery caecal contents. Large numbers of motile protozoa were detected in smears made from the upper and lower small intestine. Together with the history this confirmed spironucleosis as the cause of the problem. The stress of handling, transportation and introduction to a new environment were likely triggers for the outbreak.
Congenital Segmental Intestinal Anomalies In Calves

Segmental anomalies of the intestinal tract can be categorised as stenosis, involving narrowing and incomplete occlusion of the lumen, or atresia, resulting in complete occlusion. Atresia can be classified as membrane atresia (type I) (Fig A) where the obstruction is caused by a simple membrane, cord atresia (type 2) where the blind ends are joined by a cord of connective tissue, and blind-end atresia (type 3) (Fig B), in which a segment of gut and possibly the corresponding mesentery are missing leaving two blind ends.\(^1\,2\)

Figure A: Thin membrane occludes the lumen in membranous atresia (type 1)

Figure B: Blind-ending (type 3) atresia jejunii with failure of development of the corresponding mesentery.
A genetic aetiology has been reported in some breeds; for example, atresia jejuni in Jersey cattle and atresia ilei in Swedish Highland cattle are inherited as autosomal recessive traits, and there is evidence from breeding experiments that atresia coli may be inherited as an autosomal recessive trait in Holstein cattle. Colonic stenosis together with a wide range of other malformations has been associated with consumption of *Mimosa* sp in ruminants in Brazil. A genetic aetiology has been reported in some breeds; for example, atresia jejuni in Jersey cattle and atresia ilei in Swedish Highland cattle are inherited as autosomal recessive traits, and there is evidence from breeding experiments that atresia coli may be inherited as an autosomal recessive trait in Holstein cattle. Colonic stenosis together with a wide range of other malformations has been associated with consumption of *Mimosa* sp in ruminants in Brazil.3 A few observational studies have suggested that amniotic sac palpation during early pregnancy diagnosis (up to 45 days of gestation) is associated with an increased risk of atresia coli and jejuni. However, two randomised controlled trials found no association between atresia coli and allantochorion membrane or amniotic sac detection by rectal palpation (up to 45 days of gestation), suggesting that in the observational studies, rectal palpation may have been a confounding rather than a causative factor.4 5

The pathogenesis of segmental intestinal malformations remains controversial and no one theory explains the full spectrum of malformations. The two main theories currently proposed are the vascular insult theory and the non-recanalisation theory. In the former, based on experimental work in dogs, lambs and chickens, vascular compromise, and a failure of blood supply during gestation results in ischemia and a failure in intestinal development. This however does not satisfactorily explain duodenal or large intestinal atresias, or type 1 (membranous) atresias. These, and perhaps some of the others, are more plausibly explained by the non-recanalisation theory in which the gut lumen fails to recanalise after week 6 of gestation, possibly due to sporadic genetic events and aberrant regulation of molecular pathways.

Between January 2015 and August 2020, SRUC Veterinary Services diagnosed 31 cases of segmental intestinal anomalies by postmortem examination, originating from 30 herds. The majority of cases (n=18 (58 per cent) were in neonatal calves between 1 and 7 days of age, while seven (23 per cent) cases were in stillborn calves, and six (19 per cent) cases were in aborted calves (Table 1). Five of the neonatal calves presented with a dilated abdomen. Thirty (97 per cent) of the cases occurred in a wide range of beef breeds and their crosses with 19 (63 per cent) of these occurring in crossbreeds. The remaining case was atresia jejuni in an aborted Holstein foetus. There was no apparent sex predisposition. Twenty-nine (94 per cent) had atresia jejuni and two (6 per cent) had more unusual congenital intestinal malformations which included a case of double caecum, and a case of segmental, possibly multifocal jejunal abnormality that was obscured by extensive adhesions. In the 29 calves with atresia, atresia jejuni was most common (n=19, 66 per cent), followed by atresia coli (n=7, 24 per cent), atresia ilei (n=2, 7 per cent), and atresia ani (n=1, 3 per cent).

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Table 1: Location of the 29 atresia cases according to submission type

*‘Other’ refers to more unusual congenital segmental intestinal anomalies

The atresia was blind ending (type III) in 13 (45 per cent) cases, and membranous (type I) in 2 (7 per cent) cases (type of atresia not detailed in the remainder). In 4 (21 per cent) cases of atresia jejuni, the dilated loops of intestine proximal to the atresia had undergone torsion (Figure C) which was associated with mild to moderate peritonitis. Intestinal atresia was the sole malformation in 20 (69 per cent) cases. In 3 (10 per cent) cases, it was associated with umbilical hernia, and in 6 (21 per cent) cases, it was part of a more complex syndrome involving multiple congenital soft tissue and skeletal malformations.

Our short review of cases shows that calves with segmental intestinal anomalies may not be born alive as they will be aborted or stillborn, the latter sometimes due to dystocia associated with a distended abdomen. Except for atresia ani, segmental anomalies in neonatal calves are rarely suitable surgical candidates, and calves either die within the first seven days or are euthanased on welfare grounds. In most situations, segmental intestinal anomalies are considered sporadic events, however multiple incidences in a herd raises the possibility of a hereditary aetiology and warrants further investigation.
Figure C: Atresia jejuni with torsion of intestinal loops (arrow) proximal to the obstruction resulting in dilation and ischaemia of the entrapped intestinal loop (asterisk).

References