

OVERVIEW

- **Losses due to hypogammaglobulinaemia and colisepticaemia in beef calves on multiple holdings.**
- **Atresia jejunii in an Aberdeen Angus calf.**
- **Outbreak of duck viral enteritis in a flock of Muscovy ducks.**

GENERAL INTRODUCTION

The mean temperature for April was 0.9 °C above the long-term average. It was the third driest April in a series from 1862, and the sunniest April in a series from 1929 with 31 percent of average rainfall and 151 percent of average sunshine

CATTLE

Generalised and systemic conditions

Colisepticaemia associated with hypogammaglobulinaemia was confirmed as the cause of calf death in three beef herds in south-west Scotland. Predisposing factors for poor colostrum intakes were identified in all cases. A four-day-old shorthorn calf was found dead and submitted for postmortem examination. Findings of trauma and subcutaneous oedema in the hindlimbs were consistent with the history of entrapment in a slatted area where the calving took place. The liver was enlarged and yellow. A zinc sulphate turbidity (ZST) test result of 9 units (reference range ≥ 20 units) was consistent with inadequate passive transfer of maternal colostral antibodies most likely due to the trauma in this case. *Escherichia coli* was isolated from all tissues cultured.

A second herd, which had lost five of 150 calves in the week after birth, submitted a carcass for investigation of the problem. The birth of large calves was reported and the submitted calf, which weighed 54 kg, had failed to suck in the neonatal period. Postmortem examination revealed a fibrinous pericarditis, epicarditis and pleuritis, septic arthritis affecting multiple joints and a purulent meningitis. *E coli* was cultured from the brain, joints, liver and heart. The ZST result of 11 units was lower than ideal. In the final case five calves from a herd of 40 presented with neurological signs prior to death. A two-week-old calf was euthanased and *E coli* was isolated from multiple tissues including the brain. A ZST of 4 units confirmed hypogammaglobulinaemia. An assisted calving and

fostering onto another cow were identified as risk factors in this case.

A 53 kg Charolais cross calf deteriorated within an hour of an assisted birth and died despite treatment with intravenous fluid, bicarbonate and NSAIDs. Another two calves had died under similar circumstances the previous week. Postmortem examination findings were consistent with dystocia and the cause of death was considered to be hypoxia and acidosis secondary to fractured ribs and pulmonary haemorrhage (Fig 1). Foetal oversize and bradycardia were thought to be responsible for the losses. Excessive cow condition and subclinical hypocalcaemia can also contribute, and further investigations were recommended.



Figure 1 - Rib fractures and associated haemorrhage following an assisted calving.

Respiratory tract diseases

A beef cow in late gestation became dyspnoeic over the course of a week and died despite antimicrobial treatment. The lungs did not collapse when the thorax was opened and were consolidated with interstitial oedema and emphysema. Histopathological examination of the lungs identified three different pathologies. Necrotising bronchiolitis consistent with *Mycoplasma bovis* infection was superimposed on areas of fibrosing and lymphocytic/plasmacytic bronchointerstitial pneumonia, bronchiolitis obliterans and widespread chronic interstitial pneumonia. This reflected a combination of previous airway and interstitial lung injury.

Alimentary tract disorders

Atresia jejunii was detected in a 4-day-old Aberdeen Angus calf that was born with a firm distended abdomen and had failed to pass faeces. The intestinal lumen was obstructed by a thin membrane approximately 40 cm from the

ileocaecal junction, and abundant fluid had accumulated proximally (Fig 2). This type of atresia is classed as a membrane atresia (type I). The third to fourth week of gestation, when normal rotation of the intestinal tract occurs, is considered to be a period of susceptibility.¹ Genetic factors have been demonstrated in some cattle breeds including Swedish Highland cattle.²

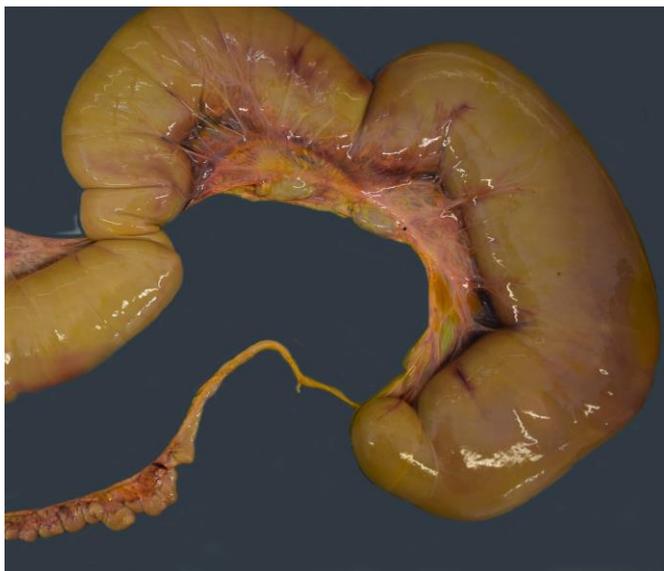


Figure 2 – Atresia jejuni in an Aberdeen angus calf

Nervous system disorders

A dairy herd north-west England reported that 13 of 18 calves born to homebred heifers were clinically abnormal. The heifers had been over-wintered at another farm, where they had contact with other cattle. All had been vaccinated with an inactivated BVD vaccine prior to service. Six calves had thus far tested positive for BVD virus, with their dams giving negative results. A typical calf was submitted alive and noted to be small with shortening of the long bones and slight arthrogyriposis. It was unable to stand unaided and tended to bear weight on its toes when supported. Continuous tremor and nystagmus were apparent. The crown rump length was 79 cm, confirming in utero growth retardation, and BVDV 1 RNA was identified by PCR from both blood and spleen. Neuropathology, including brain:cerebellar weight ratio, found no evidence of cerebellar dysgenesis and revealed a diffuse hypomyelinating leucoencephalomyelopathy typical of hypomyelination associated with persistent BVDV infection. Further typing of the virus strain is ongoing.

SMALL RUMINANTS

Generalised and systemic conditions

Six Scottish blackface hogs from a group of 300 died over a 48 hours period three weeks after being turned out to the hill. Most of the group had been purchased in autumn 2019 and a course of a clostridia/pasteurella vaccine had been given in September and October. One hogg was submitted for investigation of the problem and three ticks were found on the carcass. Postmortem examination revealed subcutaneous haemorrhages, oedematous lungs and splenomegaly. There was evidence of fibrinous peritonitis and large numbers of miliary foci throughout the liver parenchyma (Fig 3). *Bibersteinia trehalosi* was isolated from multiple tissues consistent with a diagnosis of systemic pasteurellosis. *Anaplasma phagocytophilum* DNA was detected on PCR of spleen suggesting that immunosuppression due to concurrent tick borne fever had predisposed to the outbreak.



Figure 3 – Hepatic miliary foci as a result of *Bibersteinia trehalosi* septicaemia

Ten February/March born lambs from a group of 50 at grass died after a short period of weakness and lethargy. Response to treatment with antibiotics and NSAIDs was poor. The carcass of a four-week-old texel tup lamb was submitted for investigation of the problem and postmortem examination revealed a severe fibrinous pleuritis/pericarditis and pleural effusion. There was no evidence of lung consolidation, little milk in the abomasum, and a mild fibrinous peritonitis. *Mannheimia haemolytica* was isolated from all tissues cultured. The ewes had been vaccinated with Heptavac P one month prior to lambing but any protection afforded to the lambs from colostral antibodies would have been waning by the time of the outbreak.

Alimentary tract disorders

A flock of 750 ewes submitted four, one-week-old lambs for postmortem examination after 20 were found dead or dying in various fields. Scour, abdominal swelling and salivation was reported. There had been no issues with weak lambs and ewe condition was good. ZST testing was possible in three of the lambs and there was evidence of hypogammaglobulinaemia in one. (ZST 21, 21, and 8 units; reference range ≥ 14 units). In all cases the conjunctivae were pale and the carcasses were faecal stained. The abomasa were well filled with clotted milk confirming the cases to be true sudden deaths. Emphysema was present within the wall of the proximal to mid jejunum which had red liquid contents. In one case haemorrhages and gas bubbles were also found within the mesentery (Fig 4). Histopathology confirmed a diagnosis of necrotising and emphysematous enteritis consistent with lamb dysentery despite administration of a clostridia/pasteurella booster to ewes one month pre-lambing. Discussion with the pharmaceutical company was advised.

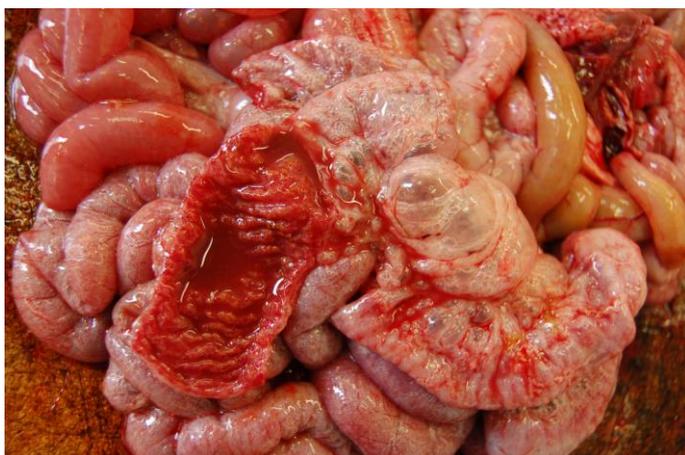


Figure 4 – Emphysema within the jejunal wall and mesentery in a case of lamb dysentery.

Reproductive tract conditions

Eleven ewes aborted from a flock of 395, resulting in the submission of foetuses and placentae from four ewes. Multiple white foci were found disseminated throughout the liver in two of the three foetuses from the first ewe (Fig 5) and *Listeria monocytogenes* was isolated in pure growth from the foetal stomach contents of one. Marked thickening of the intercotyledonary placenta was noted in the remaining three submissions and chlamydial elementary bodies were detected on microscopic examination of modified Ziehl Neelsen smears consistent with a diagnosis of enzootic abortion. When abortion storms are occurring examination of foetal material from

several ewes is advised to aid detection of multiple significant pathogens.



Figure 5 – Hepatic lesions in a foetus following abortion due to *Listeria monocytogenes*

Nervous system disorders

Seven north country Cheviot lambs from a flock of 100 ewes and lambs exhibited clinical signs of tremor which progressed to recumbency and death. The carcasses of two lambs aged two and four days were submitted for postmortem examination which proved unremarkable. Histopathology of tissues from the younger lamb confirmed a vacuolar neuronopathy with associated spheroid formation which particularly involved the lateral and medial geniculate bodies, cranial nerve nuclei and olivary nuclei. These findings were consistent with a lysosomal storage disease, likely to have a genetic basis. A range of lysosomal storage diseases have been reported in different sheep breeds.³ Similar findings were not evident on examination of the other lamb and, although autolysis hampered evaluation, no evidence was found of swayback or border disease and the possibility of hypoglycaemic encephalopathy could not be excluded.

PIGS

Generalised systemic diseases

The second pig to die from a shed of 620 weaners was submitted for postmortem examination. The five-week-old landrace cross large white was in poor body condition with evidence of enteritis, an enlarged liver and splenic infarctions. There was an excess of cerebrospinal fluid (CSF), the lateral ventricles were dilated and the cerebral cortex was thin. *Streptococcus suis* serotype 13 was isolated from the brain and liver, and rotavirus type A was

detected in faeces, which may have explained the enteric signs. Microscopic changes in the brain were consistent with a chronic sequel to bacteraemic localisation, most likely *S suis*, resulting in ventriculitis and obstruction of CSF flow. SRUC VS commented that serotype 13 is not commonly identified in primary disease outbreaks in the UK, and that the concurrent rotavirus infection in this pig may have resulted in the poor body condition and predisposed to a secondary septicaemia.

BIRDS

Poultry

Thirteen free range Muscovy ducks from a group of 25 died and a further three were unwell. Diarrhoea and coughing were reported, and wild mallards had been seen nearby. One bird, described as lethargic but in good body condition, was postmortemed at the practice. No diagnosis was reached following gross examination and bacteriology. Subsequent histopathology identified acute multifocal necrosis and fibrinous hepatitis, splenitis and oesophagitis with intranuclear inclusion bodies consistent with duck viral enteritis (DVE). DVE is caused by a herpesvirus and is endemic in wild ducks which frequently act as a source of infection for domestic ducks.

Pigeons

Three pigeons from a loft of 150 were submitted for investigation of neurological signs and diarrhoea affecting only young birds aged between two weeks and two months. The parents of these birds had been purchased three months earlier and their paramyxovirus vaccine status was unknown. Clinical signs included torticollis, ataxia and wing droop. Green gizzard and large intestinal contents were consistent findings in the three birds examined postmortem. Infection with pigeon paramyxovirus was suspected but testing proved negative. *Salmonella enterica* serovar *typhimurium* Copenhagen was isolated from all three birds and considered to be responsible for the clinical signs. This is known to be a pigeon associated strain of *S typhimurium*.

MISCELLANEOUS

Deer

The plucks from two fallow deer (*Dama dama*) that had been shot wild, but suspected to have escaped from a farm, were submitted for examination. Firm grey masses were found in the right cranial and caudal lobes of one lung, with a very firm core to one of the lesions. In the other set of lungs, the caudal half of both caudal lobes had been

almost completely replaced by firm grey masses, with large areas of hard, mineralised tissue. Histopathology on lung tissue from both animals detected some similarities to equine multinodular pulmonary fibrosis (EMPF) including the presence of intralesional intranuclear inclusion bodies. EMPF is linked to a rhadinovirus, equine herpesvirus 5 (EHV5),⁴ which can also be isolated from clinically normal horses. It was noted that there were some differences between the pathology seen in the deer and that observed in horses such as the presence of numerous multinucleated cells, mineralisation/woven bone formation and a relative lack of preservation of alveolar structures. Herpesvirus DNA was detected in the lung tissue by pan-herpesvirus PCR, and PCR product sequencing showed 100 percent sequence homology with a rhadinovirus, fallow deer lymphotropic herpesvirus. It was noted that a direct link between the presence of the virus and the lung lesions has not been proven.

References:

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Salmonella enterica serovar Dublin in Beef Herds

Although only 10 percent of SRUC VS diagnoses of *Salmonella enterica* serovar Dublin come from beef herds, it can have a significant impact on health and mortality rates during a calving season in these herds.

In dairy herds, the clinical presentation is often confined to one age group within a herd, i.e. some herds experience predominantly abortions, others calf health issues and others diarrhoea in adult cattle. SRUC VS's experience is that in beef herds S Dublin can present with a range of clinical signs across several age groups. This likely reflects that beef herds are often more naïve and there is increased contact between different ages of cattle.

There were 42 diagnoses of S Dublin in beef herds between 2014 and 2018 in Scotland. 28 percent of the diagnoses were in adult cattle, of which 40 percent were abortions. The abortion rate in affected herds ranged from 2 to 5 percent at the time of submission. The only other clinical sign noted in adult cattle was diarrhoea, with 80 percent of those affected within the two weeks after calving.

The remaining 72 percent of the diagnoses were in calves less than six months of age, with no diagnoses in animals between six months of age and pregnant heifers in their last trimester. The peak of diagnoses occurred in animals between one and four weeks of age, with 60 percent of calf diagnoses in this category.

Mortality rates were high, with herds experiencing up to 18 percent mortality of the calf crop at the time of submission. The clinical presentation in calves was more varied as shown in Table 1.

Primary clinical presentation	Percentage of calves affected
Diarrhoea	64%
Respiratory disease	20%
Neurological signs	16%

Table 1: Clinical presentation of S Dublin in calves less than six months of age

Control in dairy herds is aided by snatch calving, but this is an impractical option for beef herds making control more challenging. Hygiene of the calving pen becomes critical in beef herds to limit transmission from cow to calf and also between cows and calves.

Carrier cows are more likely to shed around calving, and cows that become infected around calving have an increased risk of becoming a carrier, and therefore maintaining infection within the herd. Infection in beef herds tends to build up in the environment towards the end of the calving period. This can be limited by grouping cows by expected calving date if known, frequently replenishing bedding and/or using a separate area for the second half of the calving period. It is important to avoid overstocking.

Minimising the age range of calves in a group will reduce spread from older, recovered calves. In the face of an outbreak, splitting the herd into smaller groups will also reduce transmission. Weather permitting, turning cattle out as quickly as possible will help to limit transmission, assuming the grazing is clean. If calving outside, avoid using the same pasture for the whole of the calving period. S Dublin should be a differential diagnosis in herds experiencing problems with calf mortality, scour, pneumonia or neurological signs. Diagnosis can be made at the time of the outbreak by culturing faeces, or postmortem samples, or retrospectively using serology.

The level of calf mortality in a beef herd will have a significant influence on the profitability of the enterprise for that year and, if carrier cows are generated, may impact the herd in future years. The period around calving is critical for control, with 60 percent of calves and 80 percent of cows affected within a month of calving.