OVERVIEW

- Idiopathic necrotising enteritis in suckler calves
- *Mannheimia haemolytica* abomasitis in a five-week-old calf
- Alopecia associated with micronutrient deficiency in lambs fed milk replacer
- Peak diagnoses of nematodirosis in lambs
- Enteric and spinal listeriosis in sheep

GENERAL INTRODUCTION

June in Scotland had a mix of wet and dry days. The mean temperature for June was 0.9°C above the long-term average. There was 107 per cent of average rainfall and 96 per cent of average sunshine. American foulbrood disease of bees was detected in South Aberdeenshire.

DISEASE ALERTS

The following conditions were reported by SAC C VS disease surveillance centres in October 2015. Given similar climatic and production conditions, they could also be important this year.

- Parasitic pneumonia in cattle
- Coccidiosis in autumn-born suckler calves
- White liver disease in lambs
- Hyposelenosis in lambs

CATTLE

**Toxic conditions**

Inverness diagnosed lead poisoning in a one-year-old Limousin heifer, one of three heifers that died suddenly following breaking into a field where a tractor had accidentally caught fire and burned out the previous year. Blood lead value was elevated at 5.75 µmol/l (greater than 1.2 µmol/l is consistent with lead poisoning). Aberdeen also diagnosed lead poisoning in a three-month-old calf, with blood lead levels of 5.89 µmol/l, that had neurological signs and was exposed to a lead battery.

**Parasitic diseases**

Babesiosis is infrequently diagnosed in Scotland (an average of three cases per year in the past five years), but Ayr diagnosed babesiosis in five- and ten-year-old suckler cows from separate farms, where severe anaemia was evident with packed cell volumes of 0.08 and 0.1 l/l (reference range 0.25 to 0.45 l/l) and typical bodies identified within the red blood cells. Aberdeen also diagnosed suspected babesiosis in a seven-year-old suckler cow that was weak and pale. Abnormal atypical bodies were seen within red blood cells and the red blood cell count was 1.4 x 10^{12}/l (reference range 5.5 to 10.0 x 10^{12}/l).

Aberdeen diagnosed type 2 ostertagiosis at postmortem examination of two eight-year-old suckler cows that presented with profuse diarrhoea and inappetance. Diarrhoea and weight loss also affected most of the adult cows in the herd. Antemortem faecal worm egg counts from the two cows were 1600 and 500 strongyle eggs per gram (epg). Plasma albumin and pepsinogen levels were 23.0 g/l and 27.0 g/l (reference range 30 to 40 g/l) and 3.8 iu/l and 4.0 iu/l (pepsinogen results above 2.5 iu/l indicate abomasal parasitism likely to be associated with clinical disease) respectively. Underlying reasons for such severe clinical disease in adult cattle were not determined.

**Generalised and systemic conditions**

Ayr diagnosed black disease in a calved two-year-old Holstein-Friesian heifer that showed extensive oedema for three days prior to death. Postmortem examination revealed oedematous ocular conjunctiva, subcutaneous ventral oedema and a large volume of straw-coloured peritoneal fluid. The liver was adhered to the diaphragm and adult *Fasciola hepatica* were present in the bile ducts. In addition there was a large haematoma, approximately 10 cm in diameter and an adjacent pale necrotic lesion with a surrounding zone of haemorrhage. Although no Gram-positive rods were detected on direct smear of the necrotic liver lesion, histopathology confirmed chronic fibrosing hepatitis, along with areas of acute necrosis, haemorrhage and surrounding supplicative inflammation. Gram staining revealed robust, predominantly Gram-positive bacterial rods, which sometimes formed chains in the areas of hepatic necrosis and haemorrhage particularly at the margins of necrotic zones. SAC C VS considered that these changes were consistent with chronic fluke exposure and secondary clostridial necrotic hepatitis. A review of flukicide treatments and clostridial vaccination protocols was advised.

**Alimentary tract disorders**

Dumfries diagnosed coccidiosis, with an oocyst count of 85,000 per gram, and concurrent *Salmonella* Mbandaka infection as the cause of death of a three-week-old Aberdeen Angus-cross calf from a dairy herd, the second to die within a week. S. Mbandaka is the third most common *Salmonella* serotype isolated from cattle. It may
be isolated from clinically normal animals, but is reported to cause diarrhoea and malaise in stressed or immunocompromised animals.

Dumfries examined a six-week-old Charolais-cross calf, the third from a group of 400 to die at grass. Postmortem examination revealed anaemia and an oedematous omasal wall with erosions along the edges of the leaves (Fig 1). Histologically, there was erosion/ulceration particularly at the tips of the laminae with associated fibroblast activation/granulation tissue. No evidence of current mycotic or viral infection was detected and despite extensive testing the aetiology was not determined.

Ayr diagnosed idiopathic necrotising enteritis in a two-month-old Charolais-cross heifer calf, the second calf to die after showing signs of abdominal pain. Postmortem examination revealed bilateral pharyngeal ulceration and ulceration of the abomasal, small intestinal, caecal and colonic mucosa. The lesions were more severe in the large intestine, where necrotic tissue adhered to the ulcerated areas and perforation resulted in localised peritonitis with adhesions. Screening for Salmonella species, coccidial oocysts and bovine viral diarrhoea virus (BVDV) was negative. Histological changes were consistent with idiopathic necrotising enteritis.

Aberdeen cultured Histophilus somni from epididymitis in a two-year-old Aberdeen Angus bull, where clots were observed in the semen sample at a routine bull breeding soundness examination. Microscopic examination revealed a high number of neutrophils and bacteria in the semen.

St Boswells also diagnosed idiopathic necrotising enteritis in a ten-week-old Aberdeen Angus calf that was unwell for one week and died despite supportive treatment. Postmortem examination revealed diphtheresis of the larynx and trachea and consolidation of the diaphragmatic and anterior lung lobes. There was ulceration of the small intestinal mucosal surface and infarct-like lesions in the kidneys. The diagnosis was confirmed histologically.

Ayr diagnosed abomasitis in a five-week-old Simmental heifer calf that was treated for suspected respiratory disease, but died the following day. Postmortem examination revealed diffuse thickening and oedema of the abomasal wall with extensive reddening and ulceration of mucosa (Fig 2). Peritonitis, perihepatitis and perisplenitis were also present. A heavy growth of Mannheimia haemolytica was recovered from abomasal mucosa. Histopathology confirmed severe, subacute abomasitis with Gram staining identifying intralesional small Gram-negative bacilli, consistent with M. haemolytica. SAC C VS commented that the pathology detected was very similar to Mannheimia abomasitis in lambs, but this diagnosis is uncommon in calves.
Thurso identified a developmental abnormality in a two-day-old calf from a herd which had several dwarf calves. At gross examination there was shortening of the long bones of limbs, but the skull and vertebral column appeared normal. Histological examination of one growth plate revealed no changes to explain the gross abnormalities. The physis was of even width, and physeal chondrocytes formed orderly rows with all zones represented and of normal morphology. There was prominent active remodelling in the metaphysis characterised by plentiful osteoclasts and osteoblasts, which lined primary trabeculae. Specifically, the histological appearance was not consistent with that described in chondrodysplastic calves associated with silage feeding of the dams. SAC C VS considered it possible that previous growth plate abnormalities had resolved, for example after restoration of a suitable diet, so that changes were not visible histologically. The metaphysis appeared quite active; however this would be expected at this age and the significance was therefore equivocal. Advice for future cases included submission of a range of growth plates the following season, as well as considering sampling cows for manganese levels after being on silage ration for at least a week. Mycotoxicosis has also been associated with skeletal developmental abnormalities, and this was considered a possible contributing factor given the poor summer weather.

Renal diseases
Ayr diagnosed nephritis in an ill-thriven two-month-old Aberdeen Angus-cross bull calf. Postmortem examination revealed small, pale, firm kidneys with the capsule adherent. Histopathology identified severe chronic fibrosing, tubulointerstitial nephritis with extensive tubular atrophy/loss and glomerular atrophy together with prominent interstitial fibrosis and variable interstitial mononuclear cell infiltrate. Scattered tubules were filled with purulent exudate. SAC C VS considered these findings consistent with severe chronic ‘white spotted kidney of calves’, which is associated with earlier bacteraemic localisation, with *Escherichia coli* considered the most likely cause. A limited mycotic abomasitis was also detected and was likely due to colonisation following injury to the abomasal mucosa associated with azotaemia. In addition sternal bone marrow was found to have sparse haematopoietic population. SAC C VS considered this was a result of a combination of suppression of erythropoiesis as a consequence of inflammation and renal disease, together with depletion of granulocytes associated with the nephritis.

**SMALL RUMINANTS**

**Nutritional and metabolic disorders**

St. Boswells diagnosed hypovitaminosis A or micronutrient deficiency as the cause of non-pruritic alopecia in two live lambs from eight pet lambs affected in a group of 26. Milk replacer was fed for two weeks and both lambs were in poor body condition with diffuse wool loss, skin thickening and crusting lesions particularly around the eyes and mouth (Fig 3). Serum and liver analysis confirmed low vitamin A levels (Table 1). Clinical signs are likely to occur when liver vitamin A content falls to 2 µg/g or below (Radostits and others, 2000).

<table>
<thead>
<tr>
<th>Serum Vitamin A (µmol/l)</th>
<th>Liver Vitamin A (µmol/kg fresh tissue)</th>
<th>Liver Vitamin A (converted to µg/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference range</td>
<td>0.87-1.75</td>
<td>≥ 60</td>
</tr>
<tr>
<td>Lamb 1</td>
<td>0.24</td>
<td>4.22</td>
</tr>
<tr>
<td>Lamb 2</td>
<td>0.59</td>
<td>19.0</td>
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</tbody>
</table>

Table 1. Vitamin A results from two lambs submitted live for examination.

The milk replacer was also analysed and found to contain levels of zinc, iron and manganese well below the declared level (Table 2). The levels of crude protein, sodium, phosphorus and calcium were as declared.
A diagnosis of multiple dietary deficiencies due to failure to add a mineral/vitamin balancer to the milk replacer was made and the feed company confirmed that this was omitted from that batch.

Parasitic diseases
Perth confirmed fourteen outbreaks of nematodirosis in lambs aged between six and twelve weeks. This contributed to an total of 54 nematodirosis diagnoses across all eight SAC C VS disease surveillance centres; this considerably higher than in the previous two years when there were 15 and 21 reports respectively. Three lambs were submitted to St. Boswells following the deaths of 22 lambs from a group of 200 over a seven day period. The lambs received an anthelmintic treatment the previous week. All three carcases were sunken eyed and dehydrated with liquid intestinal contents. In one lamb 11,700 *Nematodirus battus* worms were recovered suggesting incorrect dosing. No worms were detected in intestinal washes of the other two lambs. Vitreous humour urea was measured with results of 80.9 and 113 mmol/l (serum reference range 4 to 8 mmol/l) indicating uraemia. Histopathology confirmed nephropathy considered secondary to previous nematodirosis.

Generalised and systemic conditions
Perth, Ayr, St. Boswells and Dumfries all reported unusual presentations of *M. haemolytica* infection in lambs aged between three and eight weeks. Two cases presented with a severe abomasitis, including oedema and emphysema of the abomasal wall and folds, with localised peritonitis and splenomegaly evident in one lamb. Abomasitis due to *Clostridium sordellii* was suspected, but *M. haemolytica* was isolated and in both cases numerous intralesimal small gram negative bacilli were present on histological examination. Peritonitis was observed in a further two carcases with adhesions over the forestomach serosa in one and a severe rumenitis confirmed by histopathology in the second. In another case *M. haemolytica* was isolated from the liver, but histopathology revealed the most severe pathology to be within the diaphragm with abundant small gram negative bacilli associated with phrenic cellullitis and serositis. In contrast, in another lamb, emphysematous abomasitis was associated with histological lesions of severe histiocytic lymphangitis. Bacteriology produced only mixed growths and no evidence of significant bacterial involvement was detected with Gram and Ziehl Neelsen staining SAC C VS noted that histiocytic lymphangitis and *M. haemolytica* abomasitis can be present concurrently and postulated that the former may predispose to secondary bacterial infection. The cause of abomasal emphysema in the absence of clostridial infection is unclear but may be related to dietary and management factors.

Alimentary tract disorders
Aberdeen examined five of 18 gimmers that died in a group of 140, with lambs at foot. Postmortem examination revealed abomasitis in four animals with concurrent typhlitis in two. *Listeria monocytogenes* was isolated from four animals and histopathology confirmed lesions typical of enteric listeriosis. Deaths began seven days after whole crop silage was fed for a 24-hour-period while the sheep were housed for clipping and SAC C VS considered that this silage was a possible source of infection. *Anaplasma phagocytophilum* was also detected by PCR in one animal and a degree of immunosuppression may have increased susceptibility to listeriosis.

Nervous system disorders
Dumfries investigated hind limb ataxia in two three-month-old Suffolk tup lambs, from a 25 ewe flock. A similar case, which was not investigated, was reported the previous year. The lambs received their second clostridial/pasteurellosis vaccine dose and one week later one lamb was trailing its hind legs. It was housed and treated with antibiotics, but failed to improve and was submitted live for examination one week later. The lamb could stand, but was weak on the left hind leg which showed some muscle wasting. Postmortem examination revealed a large area of haemorrhage and necrosis within the muscles of the right hind leg. A slight reaction was found in the muscle adjacent to the left hip and sciatic nerve. A second lamb began to hop on its hind legs two weeks after vaccination. It continued to deteriorate and two weeks later was unable to support weight on its hind legs and proprioceptive reflexes were weak or absent in the right hind leg (Fig 4). The lamb nibbled when handled along its back and short, two-to-three minute convulsions were seen. Postmortem examination revealed a small area of haemorrhage and fibrosis within the muscle of the right hind leg, but this was not contiguous to the major peripheral nerves. Histopathology revealed severe lumbosacral meningomyelitis in both lambs. Gram staining revealed Gram-positive bacilli within ventral spinal nerve roots and immunohistochemistry confirmed that the lesions were due to *L. monocytogenes*. Myelitis is a relatively unusual presentation of listeriosis in sheep.

<table>
<thead>
<tr>
<th>Element</th>
<th>Analysis result (mg/kg dry matter)</th>
<th>Amount stated on bag (mg/kg dry matter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc</td>
<td>14.9</td>
<td>50</td>
</tr>
<tr>
<td>Iron</td>
<td>21.1</td>
<td>80</td>
</tr>
<tr>
<td>Manganese</td>
<td>9.15</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 2. Zinc, iron and manganese content of milk replacer
The pathogenesis most likely involves centripetal spread of infection via peripheral nerve axons and, in the present cases, intramuscular introduction of listerial bacteria at vaccination followed by centripetal spread via the motor nerves to the spinal cord is a possible explanation. However, haematogenous localisation could not be definitively excluded.

Histopathology also revealed a seromucoid and fibrinogranulocytic enteritis with "cryptitis" and occasional crypt abscesses, SAC C VS commented that, during investigation of some previous cases from other premises, enteritis was suspected but the bowel mucosa was autolysed and unsuitable for histological examination. Enteritis was clearly recognisable in the upper small intestine in the samples from the current case and seeding of bacteria or bacterial toxins to the liver following intestinal damage could provide a possible explanation for the liver lesions. The flock continued to show low mortality and egg production improved. “Spotty Liver” of layers is usually characterised by fairly low mortality, although the egg drop can often be around 15 to 20 per cent. The egg drop of over 50 per cent in this case was considered atypical. Management factors and concurrent infections were evaluated. No evidence of viral infection was found and, although there had been some work on both the water supply and the feed conveyor, there was no evidence of a causative timing or effect on the episode.

**MISCELLANEOUS**

**Exotic animals**  
*Clostridium perfringens* type D disease was diagnosed in an eleven-month-old male Huacaya alpaca (*Vicugna pacos*) that died three days after transport from another holding. Body condition was poor and there were also multiple necrotic ulcers, up to 0.5 cm in diameter, in the C3 compartment of the stomach. Two localised sections of small intestine showed congestion and blood stained content and the kidneys appeared swollen and slightly pale. A profuse pericardial effusion with a gelatinous fibrin clot was observed. Histological examination of the brain showed oedema characterised by perivascular serum leakage into the white matter, changes which supported a diagnosis of focal symmetrical encephalomalacia/*Clostridium perfringens* type D disease.
REFERENCES


Featured Article - Caseous lymphadenitis

Caseous lymphadenitis (CLA) is an infectious disease caused when the bacterium *Corynebacterium pseudotuberculosis* gains entry through skin abrasions and results in abscessation of lymph nodes and viscera in both sheep and goats. A definitive diagnosis of CLA may only be reached by culturing *C. pseudotuberculosis* from suspect lesions. Serological testing is available and is indicated to screen incoming sheep and during an eradication programme once CLA has been confirmed by bacteriology.

Rams are no more susceptible to CLA than ewes, but ram management is likely to play an important role in transmission of CLA between farms and also within farms. This is because rams tend to be kept at a higher stocking rate, are more likely to be trough-fed and are more prone to fighting.

Between January 2010 and July 2016 SAC C VS diagnosed CLA in 144 submissions from a total of 120 different sheep flocks (Table 1).

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of submissions where CLA was diagnosed</th>
<th>Number of new flocks where CLA diagnosed since 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td>2011</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td>2012</td>
<td>24</td>
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<td>2014</td>
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<td>25</td>
</tr>
<tr>
<td>2015</td>
<td>21</td>
<td>17</td>
</tr>
<tr>
<td>2016 (Jan to Jul)</td>
<td>11</td>
<td>11</td>
</tr>
</tbody>
</table>

Table 1: Number of CLA diagnoses and number of newly diagnosed flocks by year of submission

In the UK CLA is most frequently associated with the superficial lymph nodes of the head and neck region (Baird 2007) and this is mirrored in the submission material to SAC C VS. The majority of samples submitted for bacteriology were taken from abscesses on head and neck including those from parotid and submandibular lymph nodes. Additional samples came from popliteal and visceral lymph node abscesses. References: