OVERVIEW

- Closantel toxicity in a five-week-old suckled calf.
- Clostridial enterotoxaemia type D in beef calves.
- Chemical pneumonopathy due to inhalation of copper containing mineral drenches in lambs.

GENERAL INTRODUCTION

The mean temperature for June was 1.2 °C above the long-term average. Rainfall was 139 per cent of average, but well below average in the Northern Isles, especially Shetland. Sunshine was 91 per cent of average and was near or above normal in the north and far south-west, but below average across most central and eastern counties.

CATTLE

Toxic conditions

A five-year-old Limousin cross cow died four days after first showing neurological signs. It was ataxic, showed manic licking behaviour and repeatedly tried to access a loch, twice becoming stuck in the mud around the edge. It was also believed to be blind. Lead poisoning was suspected and the farmer was advised that no animals should be sent to slaughter pending the results of lead analysis. As expected kidney lead was very high at 82.2 mg/kg fresh tissue (FT) (reference range < 0.15 mg/kg FT), and a discarded car battery was found in the field. One other cow in the group of 30 was also found dead but there were no clinical signs in the remaining animals. A 16-week withdrawal period was placed upon the group, and the farmer advised to blood sample a small number at the end of this period to ensure that restrictions could be safely lifted.

A five-week-old suckled calf was euthanased and submitted to investigate multiple cases of sudden onset blindness in a group of calves. Pupillary light reflexes and menace responses were absent. The cows had been treated with a closantel pour on a few days previously and the farmer was concerned that the calves may have licked the product off the skin of their dams. Brain histopathology revealed extensive leucoencephalopathy involving intense rarefaction and vacuolation of myelin particularly perivascularly, accompanied by small axonal swellings. The history and neuropathological findings were consistent with closantel intoxication and it was advised that the incident was reported to the Veterinary Medicines Directorate as an adverse reaction.

Generalised and systemic conditions

*Clostridium perfringens* type D enterotoxaemia (pulpy kidney) was diagnosed as the cause of death of individual calves from two beef herds. In the first case a three-month-old suckled calf was found recumbent after moving to a new field and died soon after. In the second case a four-week-old calf was suspected to have died of pneumonia or heat stroke after exhibiting dyspnoea prior to death. Postmortem examination detected interlobular pulmonary oedema in both cases (Fig 1). Focal symmetrical encephalomalacia was described on brain histopathology of only the second case suggesting that this animal had survived for a longer period. Epsilon toxin selectively damages the vascular endothelium thus accounting for the gross and histopathological findings.
Reproductive tract conditions

A Simmental herd reported a higher than average barren rate, two abortions and a stillborn calf with a short jaw and limbs. The farm had purchased a new bull but, in addition, the neighbour’s Charolais bull had broken in and was suspected to have served some of the cows. The second aborted foetus was submitted for postmortem examination and displayed brachygnathia. Complete absence of the corpus callosum but no other abnormality was noted on examination of the brain making an infectious or toxic aetiology unlikely. A genetic basis to the congenital deformities was proposed suggesting that the neighbour’s bull was not responsible.

Musculo-Skeletal conditions

A single eight-week-old Charolais calf from a group of thirty was noted to be lame and was also suspected by the farmer to have pneumonia. It was treated with antibiotics but died overnight. Postmortem examination detected subcutaneous emphysema over both hind legs and the thorax. The muscles of both upper hind legs and ventral to the sternum and lumbar spine were black with a dry appearance and multiple small gas bubbles (Fig 3). A fluorescent antibody test for Clostridium chauvoei proved positive and the organism was also isolated on anaerobic culture, confirming a diagnosis of blackleg. The farm had experienced losses due to blackleg in the past but had not yet vaccinated the 2020 calf crop.

Skin diseases

A six-month-old Holstein heifer was submitted for investigation of ill-thrift and hair loss that had commenced at one month of age. The calf had steadily lost body condition but remained bright. It had been housed since...
birth and no episodes of disease such as pneumonia or diarrhoea had been noted. One other calf was reported to be similarly, but less severely, affected. Patchy hair loss was evident over the head, body, lower legs, hocks, pelvis and tail (Fig 4). The remaining hairs were easily removed and the skin was thin. Postmortem examination failed to detect any evidence of chronic disease to explain the ill thrift. The main lesion described on histopathology was of hairless telogen and the changes were distinct from both Holstein follicular dysplasia and alopecia areata. There were no findings to suggest telogen effluvium secondary to systemic disease. The main differentials were considered to be an intrinsic abnormality of hair growth or of endocrine function. T4 and cholesterol levels were checked and were within their respective reference ranges. A final diagnosis of congenital alopecia with no recognised genetic predisposition was reached.

**Figure 4 – Alopecia in a six-month-old Holstein heifer**

**SMALL RUMINANTS**

**Parasitic diseases**

Nematodirosis was diagnosed in a two-month-old North Country Cheviot lamb which had been found dead with evidence of faecal staining. The group had been treated with an anthelmintic two weeks previously but product details were not supplied. The intestinal contents were liquid and an uncountable number of *Nematodirus battus* and a smaller number of *Trichostrongylus* spp were recovered from the small intestines. This mixed burden was reflected in the faecal egg counts of 1,150 *Nematodirus* eggs per gram (epg) and 400 strongyle eggs epg. The shorter life-cycle of *Nematodirus* sp. could explain the presence of such a burden two weeks post-treatment but the finding of a patent strongyle burden suggested the possibility of treatment failure and the need for further investigation. Outbreaks of nematodirosis in Scottish flocks usually peak in June. The number of diagnoses were low this year most likely as a result of the warm weather earlier in spring allowing an early hatch.

**Respiratory tract diseases**

Losses following inhalation of copper containing mineral drenches were confirmed in lambs from three holdings during June. A group of 50, two-month-old Scottish blackface lambs were gathered into a shed and dosed with an anthelmintic and a mineral drench. Although the day was hot they were handled slowly and none was reported to be breathing heavily. Four animals died within ten minutes of dosing with a fifth death later that day. Postmortem examination found thick material in the airways and wet mottled lungs. Inhalation of the mineral drench was suspected and a lung copper result of 1590 umol/kg DM confirmed that this had occurred. Results from control lambs are usually between 114 and 140 umol/kg DM. In the second case six lambs from a group of 150 North Country Cheviots died in the 48-hour period after routine administration of clostridial vaccine, anthelmintic and mineral drench. Two lambs died in the third case and the farmer commented that he hadn’t administered mineral drench to such young lambs before. Histopathology confirmed a chemical pneumonopathy secondary to inhalation of an irritant substance in all three cases. This syndrome has been identified by SRUC VS multiple times in recent years and appears to be more likely when animals undergo multiple procedures or treatments at one time. Work is ongoing to establish the precise risk factors involved.

**Alimentary tract disorders**

A six-week-old Texel cross lamb was submitted for postmortem examination after two thriving lambs from a group of 70 were found dead in a three-day period. A 20 to 25 cm diameter area of the abomasal wall was found to be thickened, oedematous and emphysematous with a small partial thickness ulcer at the centre. Brown-green material had accumulated in the submucosa. Histopathology confirmed the presence of foreign material such as plant fragments together with mixed bacteria including degenerate tetrad structures consistent with *Sarcina* sp. Frequent colonies of *Sarcina* sp were also detected in the lumen. These findings confirmed that the bacteria were present at the time of ulceration suggesting that bacterial acid and gas production, as a consequence of fermentation of excessive luminal carbohydrate, was a contributing factor in the ulceration.
Mammary diseases

Two Romney ewes from a group of 126 were found dead in the five-day period after shearing and one was submitted for postmortem examination. Subcutaneous oedema was found over left side of the udder which was swollen and hard. Findings of lung oedema and haemorrhages elsewhere in the carcase suggested septicemia and *Mannheimia haemolytica* was isolated from the lung, liver and udder consistent with a diagnosis of *M haemolytica* mastitis and septicemia. Screening for maedi visna antibodies was carried out in case this was an underlying predisposing cause but proved negative. The ewe had received a booster dose of a clostridial/Pasteurella vaccine prior to lambing but this does not protect against *M haemolytica* mastitis.

**BIRDS**

**Game birds**
The carcase of a nine-week-old pheasant was submitted after 20 birds died since arriving on site three days previously. Diarrhoea and vent pecking were reported. The carcase was thin, the kidneys pale and mottled and the ureters were distended with urates which were also present on the pericardium and gizzard. Caecal cores were noted. Examination of a faecal sample returned a very high coccidial oocyst count of 556,000 oocysts per gram and a final diagnosis of coccidiosis with secondary renal failure was reached.

**Pigeons**
A pigeon fancier reported the deaths of 30 young birds from a group of 40 over a two-week period. A few of the remaining young birds were ill but the adult birds were unaffected. Mucofid green droppings and polydipsia were described with affected birds sitting hunched on the ground. Only the adult birds had been vaccinated against pigeon paramyxovirus and this was suspected to be the problem. A three-month-old pigeon was submitted and the main postmortem examination findings included green liquid intestinal contents and localised 1 to 2 mm lesions on one airsac. A yeast species was isolated from the airsac and *Escherichia coli* from the liver and airsac. Testing for both pigeon paramyxovirus and salmonellosis proved negative. Histopathology revealed acute focal nephrosis and enteropathy with degeneration and vacuolization of the gut associated lymphoid tissue indicative of systemic infection. Examination of the bursa of fabricius revealed lymphocyte depletion and follicle atrophy with botryoid inclusion bodies consistent with pigeon circovirus infection. Pigeon circovirus is immunosuppressive and it was suggested that secondary *E coli* and yeast infection was the cause of death in this case.
Chronic copper poisoning is due to ingestion and/or supplementation of copper over a long period of time. Once the liver lysosomal storage capacity is exceeded, copper is released into the circulation where it enters red blood cells and induces intravascular haemolysis. A haemolytic crisis can be precipitated by stressful events such as transport, and concurrent exposure to copper may not be a feature. Presenting signs can range from sudden death to lethargy and recumbency followed by death. Fragments of red blood cells block renal tubules leading to uraemia. The mucous membranes and sclera will often appear jaundiced (Fig A) and haemoglobulinuria may be reported. Typical postmortem examination findings include generalised carcase jaundice, a bronze liver and black kidneys (Fig B).

Figure A: Jaundice in a case of chronic copper toxicity

Figure B: Urine, liver and kidney from a case of chronic copper toxicity

In order to record a diagnosis of copper toxicity, liver or kidney copper must be in excess of 10,000 µmol/kg dry matter (DM) or 1000 µmol/kg DM respectively (reference ranges 314 to 7850 umol/kg DM and < 787 umol/kg DM). During the past five years SRUC VS has diagnosed copper toxicity in sheep on 62 occasions with peaks in March and September (Fig C). These may be associated with increased supplementary feeding pre-lambing and prior to autumn sheep sales.
Two thirds of diagnoses were in animal less than 2 year of age. Twelve different breeds were represented with Texel and Texel crosses accounting for 32 per cent of cases. This reflects high susceptibility to chronic copper poisoning in sheep of this breed which, due to low copper availability in their native range, are very efficient at absorbing copper. 42 per cent of cases were reported to be receiving supplementary feeding at the time of diagnosis and 9 per cent had received a copper containing drench or bolus. The administration of ewe boluses to lambs was the issue in several cases. Other risk factors included, accidental exposure to cattle cake, the application of distillery effluent to pasture, access to copper containing foot baths and grazing fields with a high red clover content.

Liver copper values in cases of toxicity ranged from 11,500 µmol/kg DM to 68,700 µmol/kg DM with an average of 25,156 µmol/kg DM. Liver copper levels in excess of 28,000 µmol/kg DM have food safety implications and Food Standards Scotland was informed of 13 of the 62 incidents. Serum/plasma copper levels do not provide any information on liver copper loading and the risk of a haemolytic crisis. Measuring the liver copper content of cull ewes may be a useful monitoring tool.

Copper poisoning should be considered as a possible differential diagnosis in all unexplained deaths in intensively managed sheep whether supplementation has been given or not. By law, hard feed for sheep must contain no more than 15 mg copper/kg DM. Despite this limit prolonged concentrate feeding can cause chronic copper toxicity in susceptible breeds. Mineral supplementation whether in feed, drenches or boluses should be based on known requirements with breed susceptibility taken into account.