SRUC Veterinary Services Monthly Report for March 2021

OVERVIEW

- **Udder cleft dermatitis as a possible source of bacterial septicaemia in a dairy cow**
- **Inadequate nutrition leading to peri-parturient losses in both cattle and sheep**
- **Congenital motor neurone degeneration in a cross bred lamb**
- **Liver cirrhosis as a result of chronic fasciolosis in an alpaca**

GENERAL INTRODUCTION

The mean temperature for the month was 1.1 °C above the long-term average, and it was warmest relative to normal towards the east. Scotland as a whole had 100 per cent of average rainfall and 98 per cent of average sunshine. The west and north-west were rather wet, while some southern and eastern parts were drier and slightly sunnier than average.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in June 2021. Given similar climatic and production conditions, they could also be important this year.

- **Clostridial enterotoxaemia type D in suckled calves.**
  Pulpy kidney is a well-recognised cause of sudden death in lambs and should be considered a differential diagnosis when calves are found dead. Interlobular pulmonary oedema may be found on postmortem examination, but histopathology of tissues including brain is often required to confirm the diagnosis.
- **Chemical pneumonopathy due to inhalation of copper containing mineral drenches in lambs.**
  This syndrome has been identified by SRUC VS numerous times in recent years and appears to be more of a risk when animals undergo multiple procedures or treatments at one time. Some lambs may die within a matter of minutes and losses can continue over the next 24 hours. Histopathology confirms a chemical pneumonopathy secondary to inhalation of an irritant substance.

CATTLE

Toxic conditions
A four-month-old Holstein heifer calf was found twitching in lateral recumbency and died rapidly. Ninety heifer calves had been moved into cubicles four days earlier and simultaneously changed from a straw and concentrate ration onto a silage-based diet. It was the only animal in the group to be affected. Postmortem examination revealed an enlarged liver with petechial haemorrhages throughout the parenchyma. Histopathology detected massive hepatic necrosis, early biliary proliferation, faint greyish pigment deposits in portal areas and frequent sinusoidal erythrophagocytosis consistent with the acute clinical phase of chronic copper toxicity. This was supported by an elevated liver copper of 20300 µmol/l (reference range 314 to 7850 umol/l). It was postulated that the calf died before a haemolytic crisis could develop and that the hepatic necrosis was triggered by stress relating to the change of housing and diet. An audit of dietary copper sources was recommended.

Generalised and systemic disease
A herd submitted the carcase of a three-day-old Charolais bull calf to investigate an ongoing problem with dull calves that failed to suck and died in the first week of life. Only pedigree Charolais calves were affected and had to be stomach tubed repeatedly. No issues were seen in Aberdeen Angus calves despite identical cow management. Postmortem examination confirmed abomasal dilation and colisepticaemia both of which were considered to be secondary findings. Histopathology revealed changes suspicious of the early stages of colloidal goitre raising the possibility of earlier iodine deficiency. Mild hydrocephalus and white matter oedema were also detected and it was suggested that these lesions may have been responsible for the clinical signs. It was advised that iodine status in early gestation should be monitored with supplementation if necessary. The possibility of a genetic component to the problem could not be excluded.

A high yielding six-year-old Holstein cow experienced milk drop and malaise 10 weeks post calving. Treatment, including oral fluids, produced some improvement, but the animal was found dead the next day. Body condition score was good but an area of ulceration with crust formation was found on the ventral skin cranial to the udder. Multiple fibrous adhesions were present between the liver capsule and the diaphragm, and pale yellow coalescing foci were detected in the parenchyma. Widespread petechial haemorrhages were found on the epi- and endocardium, jejunal serosa and within the tissues of the neck. Histopathology identified

hepatocellular necrosis associated with microvascular thrombosis, and secondary consumptive coagulopathy could therefore explain the multisystemic haemorrhages. There was no evidence of primary bacterial hepatitis but it was noted that bacterial septicaemia/toxaemia is the most common cause of disseminated intravascular coagulation in ruminants. In the absence of any other evidence of bacterial infection the udder cleft dermatitis was thought be a possible source of origin. A five-year-old Holstein cow from a second farm was euthanased in order to investigate unexplained loss of condition as the herd had previously experienced similar cases. A large area of crusting was noted on the anterior udder and histopathology confirmed severe udder cleft dermatitis with extensive involvement of underlying mammary tissue. The left anterior, ventral middle and a small part of the anterior diaphragmatic lobes of the lung were consolidated with some pus present. The right anterior lobe contained numerous 1 to 2 cm diameter abscesses. *Trueperella pyogenes* was isolated from the lung and *Bacteroides* sp from the skin. A link between udder cleft dermatitis and suppurative pneumonia has been described (see feature article for more information), although descending bacterial infection could not be ruled out as the cause of pneumonia in this case. Histopathology also confirmed amyloidosis affecting liver, kidney, lymph node and small intestine. Amyloidosis is most commonly a response to chronic inflammation and will have contributed to the weight loss in this case.

Alimentary tract disorders

A Luing heifer calf from a herd of 12 cows started to lose weight from 13 days-of-age, stopped suckling and became progressively dull. Treatment with oral fluids and antibiotics was unsuccessful and it died three days later. Another calf with a similar history had died the previous week. Postmortem examination found the abomasal mucosa to be haemorrhagic and ulcerated in places. Histopathology confirmed a focal necrotising and leucocytoclastic abomasitis and revealed this to be associated with intralesional fungal hyphae. Possible predisposing causes for mycotic abomasitis are stress ulcers, prolonged antibiotic treatment, and neutropenia.

Reproductive tract conditions

A beef herd submitted four calves to investigate an issue with stillbirths in heifers. At the time of submission 38 from a group of 60 Aberdeen Angus cross two-year-old heifers had calved with eight stillbirths reported. The group had been housed since November on a diet of wheat and barley straw, bruised barley, distillers' dark grains and minerals. Body condition score was reported to average 3.5. The rest of the herd were split into young and mature cows with the former managed as for the heifers. All four foetuses were thin, slender and very poorly muscled with body weights below 24 kg in all cases. There were no significant findings on bacteriology and serology. The farm had a history of selenium deficiency, but current levels were adequate, and both dam pooled plasma iodine and foetal thyroid iodine results were within their respective reference ranges. The diet was assessed, and it was concluded that the protein levels were inadequate. Metabolic profiles detected some low urea levels supporting this finding, and elevated NEFA results indicating additional energy deficiency. The young cows on the same ration did not experience similar issues and it was suggested they were able to cope better with the diet as they were no longer growing.

Nervous system disorders

A three-year-old Holstein cow became unilaterally blind with ipsilateral ear droop one week after calving. Listeriosis was suspected but despite treatment the cow deteriorated into recumbency over the next three days. Postmortem examination was carried out and neuropathology described a purulent encephalitis with microabscesses consistent with listerial encephalitis. A whole crop based total mixed ration (TMR) was fed during the dry period switching to a grass silage based TMR once calved. The available evidence on the incubation period associated with centripetal spread of *Listeria monocytogenes* via the cranial nerves suggested that infection had occurred during the dry period.

SMALL RUMINANTS

Nutritional and metabolic disorders

A mule ewe was euthanased for postmortem examination after 13 ewes from a group of 170 were lost in a one-week period. All cases were found recumbent and died within 24 hours with no response to treatment with calcium. Body condition was reported to be adequate at scanning but since then they had grazed in-bye hill with little grass and were now thin. A benzimidazole anthelmintic had been given two weeks prior to submission and the group were due to start lambing in 14 days. Twin foetuses were found in utero and the liver was noted to be very pale. 33,600 worms were recovered from the abomasum the majority of which were *Teladorsagia* sp.

A pre-mortem blood sample returned a BOHB result of 7.8 mmol/l indicating significant energy deficiency (reference range < 1.2 mmol/l). This result was considered to be too high to be explained by the short period of recumbency before death. Histopathology findings of subacute necrotising polioencephalopathy involving superficial laminar cerebrocortical neuronal and extensive cerebellar Purkinje neuronal necrosis supported a significant pre-existing ketosis. Examination of foetal

brain also detected patchy superficial laminar cerebrocortical neuronal necrosis consistent with changes secondary to ewe energy deficiency. In addition to improving nutrition future investigation of possible anthelmintic resistance was advised.

Alimentary tract disorders

500 homebred hoggs were gathered from the hill and around a third were found to be ill thriven. Body condition had been poorer than expected at weaning and they had grazed the hill since then with no supplementary feeding apart from high energy blocks during December. Trace element boluses and a benzimidazole drench were administered but thirty animals died over the next ten days. Four, Highlander cross hoggs were submitted for investigation of the problem and found to be very thin with no body fat. Postmortem examination revealed dosing gun injuries (Fig 1) in all cases. No worms were recovered from the small intestines but up to 12,000 *Teladorsagia* sp. were recovered from the abomasa. This was considered highly suspicious of anthelmintic resistance but due to the dosing gun injuries it could not be assumed that treatment had been administered effectively. Faecal samples were collected from cohorts and the detection of strongyle eggs confirmed treatment failure with benzimadazole resistance a possibility.

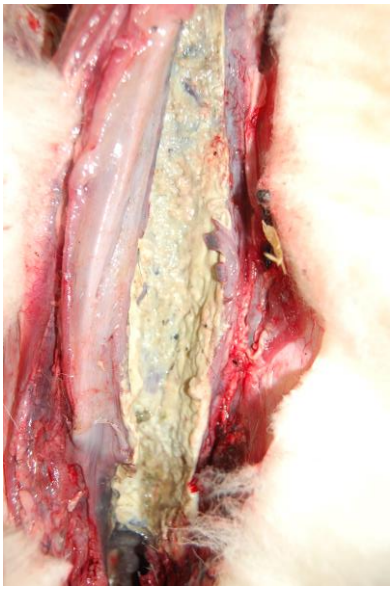

Figure 1 – Infection and necrosis in the tissues of the neck secondary to a dosing gun injury

A flock of eight Dartmoor ewes were turned out in mid-March after lambing in January. A six-year-old ewe rearing twins was noted to have lost a lot of body condition and continued to deteriorate, became anorexic and died. A large volume of ascitic fluid containing fibrin strands was found with the abdomen and an area of narrowing was detected in the mid jejunum. The proximal intestines were oedematous and red. The constriction was due to proliferation of the jejunal mucosa which formed a raised ridge around the circumference of the lumen (Fig 2). Intestinal adenocarcinoma was suspected and confirmed on histopathology. These cases are usually considered to be sporadic.

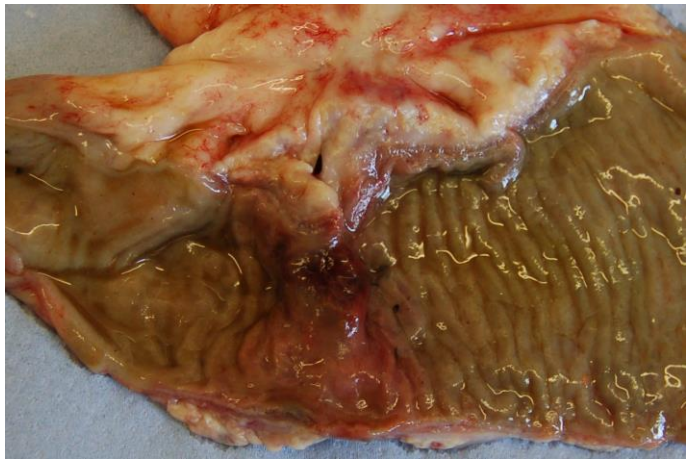

Figure 2 – Intestinal adenocarcinoma in a ewe

Nervous system disorders

A total of eight ewe lambs and six ewes from a flock of 1800 Cheviots developed clinical signs of ataxia, paresis, paralysis and recumbency two to six weeks after administration of a *Dichelobacter nodosus* vaccine. Six died or were euthanased while the others partially or completely recovered. Postmortem examination was carried out on four animals. Unilateral proliferation of grey-white tissue was found within the muscles of the cranial neck and within the epidural space at the level the atlanto-occipital joint causing compression of the spinal cord (Fig 3). Histopathology revealed this to be a granulomatous and fibrous inflammatory reaction containing well defined clear, globular spaces consistent with the presence of an oil-based compound, likely to be vaccine adjuvant. The farmer had followed the datasheet instructions on administering the vaccine 5 to 8 cm behind the ear but it appeared that some had been accidentally injected intramuscularly rather than subcutaneously. This triggered an injection site reaction that extended into the vertebral canal. This case highlights the importance of correct technique during administration of vaccines to sheep particularly in the neck.

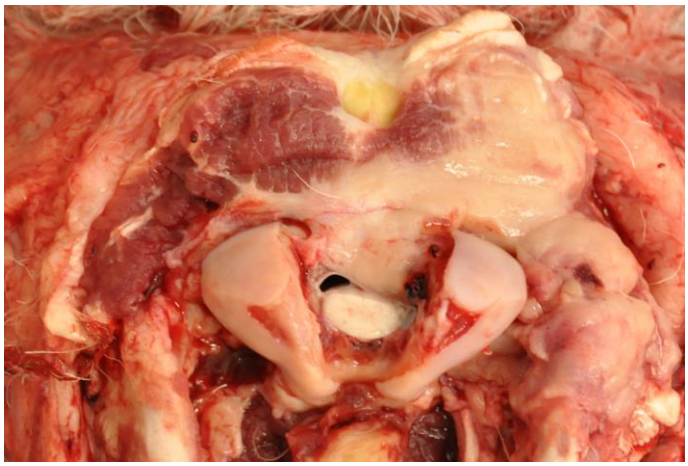

Figure 3 – Granulation tissue resulting from an injection site reaction extending into the epidural space at the atlantooccipital joint

A deformed lamb (Fig 4) born to a commercial cross ewe was euthanased for investigation of suspected Schmallenberg virus (SBV) infection. Arthrogryposis was evident in all four limbs with additional abnormal rotation of multiple joints in the right fore and both hind legs. Postmortem examination failed to detect any abnormalities in the brain, spinal cord and vertebral column and screening for both SBV and border disease virus proved negative. Foetal fluids tested positive for antibodies to toxoplasmosis, but this was considered to be an incidental finding. Histopathology failed to detect evidence of viral infection or teratogen exposure, but the distal ventral horns were described as small and underpopulated with neurones, and several chromatolytic neurones were present in the ventral horn in the cervical and lumbosacral intumescences. The dorsal nerve roots were relatively large while the ventral nerve roots were small with reduced numbers of axons and narrow myelin sheaths. Secondary changes in the skeletal muscles were consistent with denervation atrophy and a diagnosis of congenital motor neurone degeneration. No other cases were reported, and the lesions were therefore considered to most likely be the result of a sporadic genetic mutation.

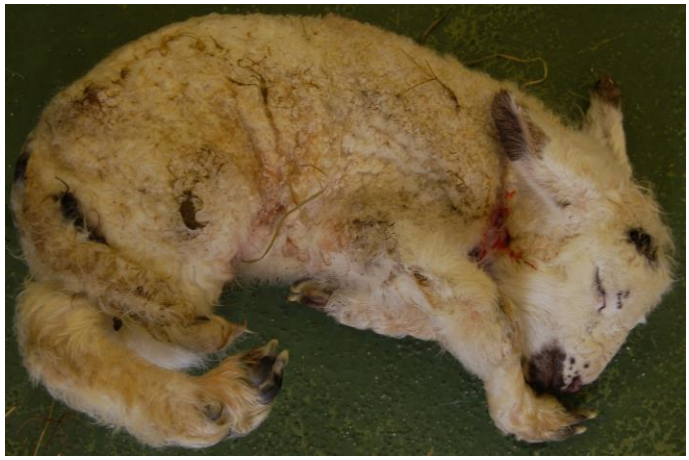

Figure 4 – Arthrogryposis secondary to congenital motor neurone degeneration

Renal diseases

An on-farm postmortem examination was carried out to investigate the birth of four lambs with severe ascites in a flock of 20 Border Leicester ewes. Affected lambs were born alongside apparently normal siblings and died soon after birth. Similar cases had been noted in spring 2020. The examined lamb was also reported to have a bifurcated scrotum. Marked congenital hydronephrosis, consistent with obstruction of the lower urinary tract, was confirmed on histopathology but the ascitic fluid was too viscous to allow urea and creatinine analysis. A genetic aetiology is possible and further investigation was recommended.

PIGS

Alimentary tract disorders

A duroc cross sow on an outdoor unit was recumbent and kicking ten days after farrowing eight piglets. It died soon after and was submitted for postmortem examination. This revealed evidence of peritonitis with a large volume of dark red ascitic fluid containing fibrin found in the abdomen. The left half of the liver was dark coloured and emphysematous as a result of a 360-degree clockwise torsion of the left lobe. Deep fissures between the lobes predispose pigs to liver lobe torsions.

MISCELLANEOUS

Camelids

Three, six-month-old alpacas were purchased in autumn 2020. One animal died in January and a second in March following a three-month history of poor appetite and weight loss. The carcase was very thin with no body fat and serous effusions in the thorax and pericardium. The liver was enlarged and fibrous (Fig 5) and moderate numbers of adult *Fasciola hepatica* were detected. One lobe contained a localised area of multiple necrotic foci plus two, 2.5 cm diameter abscesses. The carcase did not appear to be anaemic, and the liver fluke burden was not considered to be large enough to explain the death of this animal. However, histopathology confirmed severe portal to bridging fibrosis with extensive biliary replication. Fragments of curvilinear brownish yellow refractile material (consistent with remnants of fluke eggs) surrounded by macrophages were detected in the multifocal fibrosing, necrotising and pyogranulomatous hepatitis giving a final diagnosis of chronic fasciolosis and secondary liver abscessation. Hepatic fibrosis is a characteristic response to fasciolosis in alpacas.

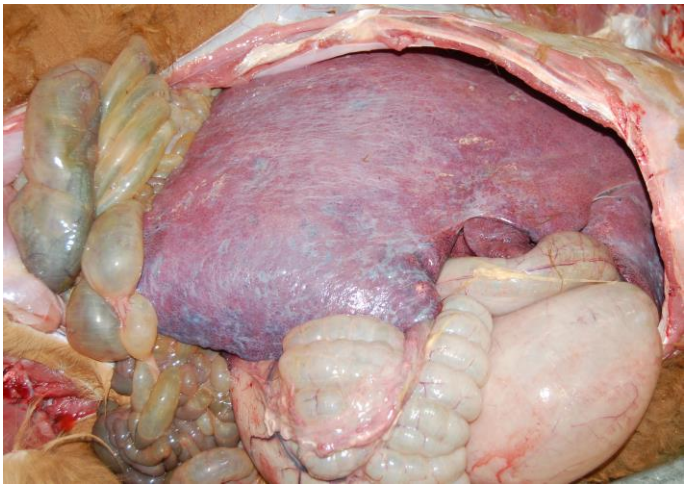

Figure 5 – Liver enlargement in an alpaca with chronic fasciolosis

Udder Cleft Dermatitis in Dairy Cows

Udder cleft dermatitis (UCD) lesions are found on the skin between the fore-quarters or at the junction of the anterior udder with the ventral abdomen (Fig A). The aetiology is not fully understood but is likely to be multifactorial involving both cow and herd level risk factors. Lesion scoring systems have been described.[1,2] Mild cases may show erythema and a moist appearance, but skin integrity is maintained. Severe lesions consist of open wounds with necrosis, haemorrhage or granulation tissue. If left untreated exudates form dry crusts which delay the healing process.[3] A primary infectious cause has not been confirmed but opportunistic secondary bacterial infection is common. A Dutch study found no evidence of infection with treponemes, fungi or mites, but anaerobic, pyogenic bacteria (*Trueperella pyogenes* and *Bacteroides* pyogenes) were detected more often in cows with severe lesions.[4] UCD can adversely affect cow welfare and may increase mastitis risk. Haemorrhage following invasion of subcutaneous veins has been reported and embolic spread of bacteria can predispose to septicaemia or suppurative pneumonia from which recovery is unlikely.[5,6]

An overall prevalence of between 5.2 and 28 per cent has been reported; with 80 to 99 per cent of herds affected; and estimates of within herd prevalence ranging from 0 to 62 per cent.[1,7] The prevalence of the condition in UK dairy herds is unknown and likely to be underestimated. A longitudinal study showed spontaneous healing in 38 per cent of affected cows however the problem subsequently recurred in 47 per cent of cases.[2] The chances of recovery were higher if the lesions were mild, of short duration and affecting heifers or second calvers.[2] Cow risk factors include large fore-quarters, increasing parity and a small angle between the udder and ventral abdominal wall.[1,7] Herd level risk factors include short cubicles and the use of mattresses.[7]

Thorough debridement and drying should be carried out before any topical treatments are applied however more research is required to establish the best approach to these cases. One study trialled the application of an enzyme alginogel based on the similarity between UCD lesions and human pressure sores.[3] Cows with severe UCD were 3.4 times more likely to show improvement than animals in the untreated control group, but less than 10 per cent of lesions healed during the 12-week treatment period.[3]

Monitoring cows in the parlour every two weeks for evidence of UCD will aid detection of lesions at an early stage, reduce the risk of lesions becoming chronic, improve treatment success and safeguard cow welfare.

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7 Ekman L, Nyman AK, Landin H, Magnusson U. Mild and severe udder cleft dermatitis – prevalence and risk factors in Swedish dairy herds. *J Dairy Sci* 2018;101:556-71

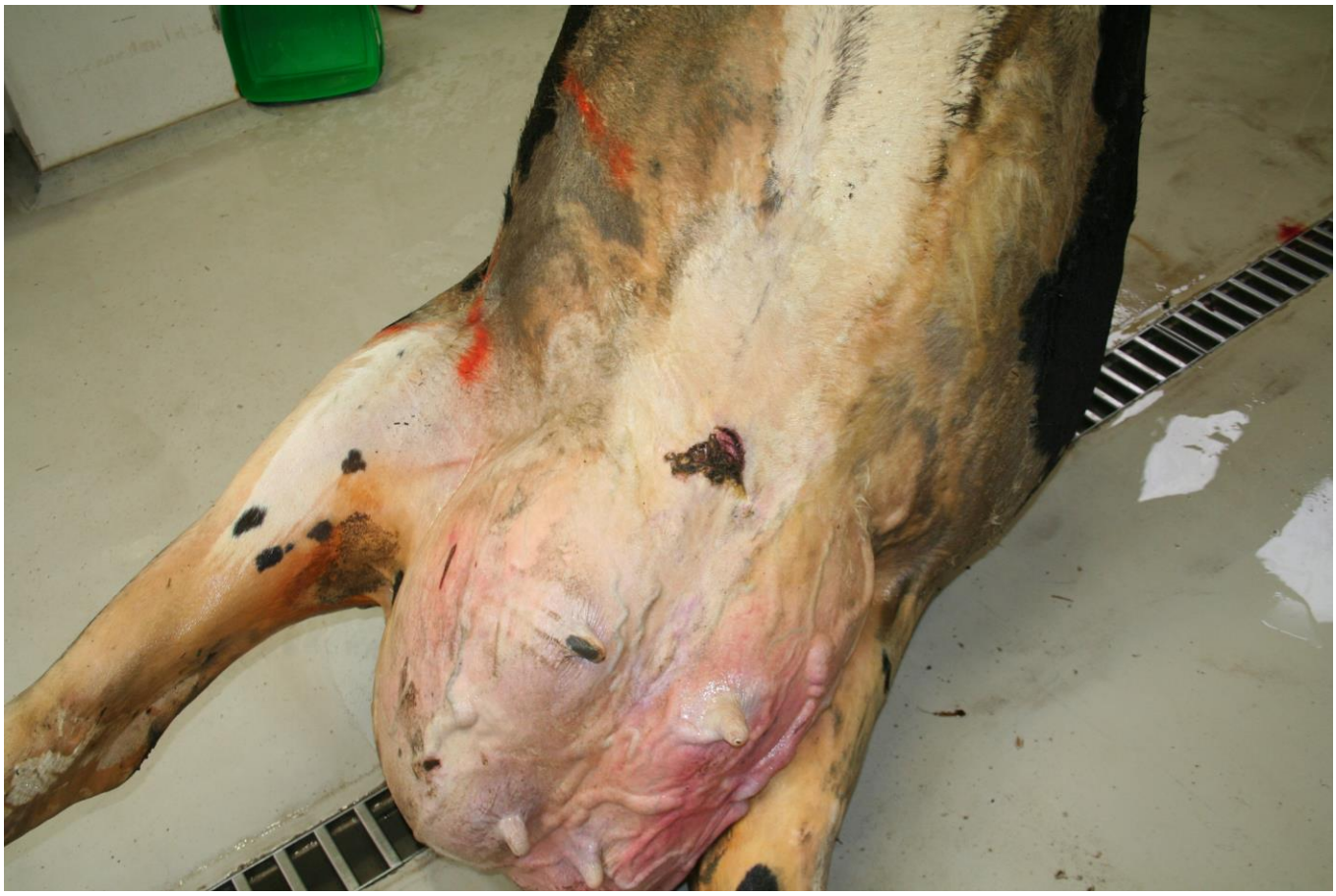

Figure A: A case of udder cleft dermatitis in a dairy cow