OVERVIEW

- **Congenital alpha-mannosidosis in belted**

Histopathology revealed clear cytoplasmic microvacuolation within neurones (Fig 2), hepatocytes, renal tubular epithelium, and thyroid follicular epithelial cells. These findings are typical of a lysosomal storage disease, and genetic testing on two of the calves confirmed that both were homozygous for the genetic mutation causing alpha-mannosidosis. This condition has an autosomal recessive mode of inheritance and has previously been well described in Galloway and Aberdeen Angus cattle.[1] It causes an inherited deficiency of the enzyme alpha mannosidase resulting in storage of water-soluble oligosaccharides within vesicles. Clinical signs can vary from stillbirth to severe congenital neurological disease. Calves that survive the neonatal period can exhibit progressive ataxia, head tremor and aggression. In this case the affected calves were stillborn or died a few hours after birth. A new young bull had been used for the first time in 2022 along with an old bull previously used in 2020 and 2021. Affected calves were sired by both bulls. All of the old bull's calves had survived but there had been two stillbirths in 2020 that were assumed to be a result of dystocia and not investigated.

Figure 2 Neuronal cytoplasmic vacuolation typical of a lysosomal storage disease

Alimentary tract disorders

A three-week-old Holstein cross calf was submitted for postmortem examination with a history of pasty diarrhoea, weakness and recumbency prior to death. Three animals had died with similar clinical signs. Calves were housed in pairs and bucket fed two litres of milk replacer twice a day. The amount of powder used had been increased from 125 to 150 g/litre during the cold weather, and it was reported that the water in the shed was freezing intermittently. The rumen and abomasum both contained unclotted milk and faeces were liquid. Small numbers of cryptosporidial oocysts were the only enteropathogen detected. Histopathology identified a purulent rumenitis consistent with rumen drinking and acidosis, abomasal and intestinal dysbiosis, and infection with attaching and effacing *Escherichia coli.* SRUC VS commented that the cold temperatures may have affected feeding behaviour and milk temperature increasing the likelihood of rumen drinking. Additionally, although the concentration of milk replacer had been increased, feeding 2 litres twice a day is insufficient. This can also predispose to rumen drinking as well as impacting on growth rates and immune function.

A six-month-old salers calf died within an hour of being found recumbent and was submitted for postmortem examination. It was the only death from a group of 50 weaned calves at grass with access to hay and a barley blend. The abomasum was diffusely reddened and moderately thickened, with petechial haemorrhages on the mucosa. The large intestinal content was very watery with reddening of the mucosa, a few small punctuate ulcers in the colon, and prominent mesocolon oedema (Fig 3). Histopathology revealed frequent intranuclear viral inclusion bodies within the endothelia of blood vessels in the mucosa and submucosa of the abomasum, small and large intestines. Damage to the endothelia explained the mesocolon oedema, and the presence of inclusion bodies suggested infection with bovine adenovirus. A confirmatory test is not readily available, but the findings were similar to those described in cases of adenovirus enteritis in New Zealand cattle.[2] Affected animals are typically pyrexic and diarrhoeic and may die peracutely. Bovine adenoviruses are widespread in the environment but given the low prevalence of disease it is likely that a combination of host and environmental factors are required to allow infection to become established. This calf had evidence of moderate parasitic gastroenteritis and mild coccidiosis, both of which may have predisposed to adenoviral infection but are not uncommon findings in young cattle.

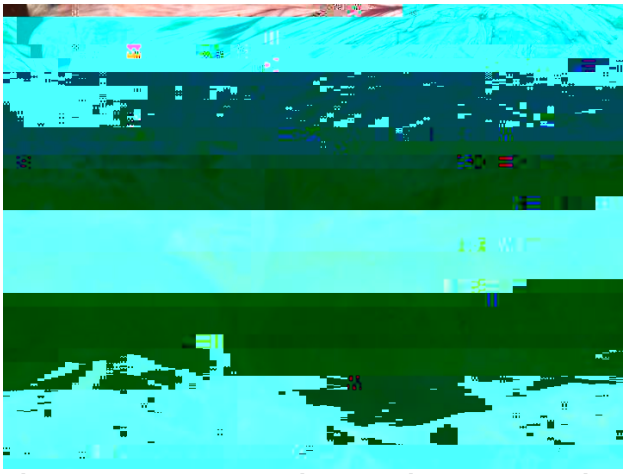

Figure 3 Mesocolon oedema secondary to suspected adenovirus infection