Necrotising encephalopathy and porencephaly in lambs

For the purposes of continuing surveillance for evidence of bluetongue virus infection in England and Wales, brains from animals submitted for diagnostic investigation are being examined for evidence of malformations, up to the age of six months in small ruminants and 12 months in cattle.

Here, we describe porencephaly and necrotising encephalopathy in fetuses of two Swaledale ewes submitted for investigation of ill thrift. The ewes were very thin (26 kg and 29 kg when fetal weights were subtracted), and each carried twin fetuses of at least 130 days' gestation, based on crown-rump length. Histological examination of the brains of the ewes found superficial laminar cerebrocortical neuronal necrosis similar to that reported by Jeffrey and Higgins (1992) in ovine pregnancy toxaemia. In both ewes, hepatocellular vacuolation was moderate and limited to mainly acinar zone 1. Significant biochemical findings at the time of presentation were ketonuria and hypoglycaemia, but not ketonaemia.

Further flock investigations confirmed suboptimal body condition, hypoproteinaemia, hypoglycaemia and subclinical ketosis in cohort animals as a result of feeding very poor quality silage. Examination of the brains of all four fetuses revealed gelatinous rarefaction of cerebral white matter progressing to porencephaly and laminar pallor (Fig 1). Histological changes were similar to those previously described in lambs with congenital necrotising encephalopathy (Scholes and Watson 2004), with additional lesions that included superficial laminar cerebrocortical neuropil mineralisation (corresponding to the linear areas of pallor on cut surfaces) and marked white matter rarefaction progressing to cavitation observed macroscopically.

The recognised differential diagnoses of porencephaly in lambs include in utero teratogenic viral infections such as Border disease virus, bluetongue virus and Akabane virus (Osburn and others 1971, Narita and others 1979, Van't tis and others 1980). The pathology in these fetuses is atypical of these viral teratogens, in particular the absence of a non-suppurative encephalitis associated with neuronal necrosis, and the presence of ongoing neuronal degeneration in the face of well-developed porencephalic lesions. The macroscopic appearance of the fetal brains, in particular the gelatinous transformation of white matter, resembled those of congenital swayback due to in utero copper deficiency. However, the histological findings, particularly those in the cerebellum, differed from those occurring in congenital swayback and the lambs' liver copper concentrations were in the upper half of the Veterinary Laboratories Agency’s (VLA’s) reference range.

White matter necrosis and rarefaction have been recorded in ovine fetuses following a range of insults including hypoxaemia, reduced placental blood flow, placental embolisation and exposure to endotoxin (Penning and others 1994, Duncan and others 2000, 2002, 2006, Loeliger and others 2003). No evidence of an infectious abortifacient, previous or current CNS haemorrhage, or significant placental pathology was detected, militating against these possible causes.

Similar histological changes in grey and white matter, although less marked, were detected in brains of fetuses from other ewes with confirmed twin lamb disease from two additional flocks (unpublished observations). These findings support an association between rarefaction of white matter (leading to porencephaly) and necrotising encephalopathy in lambs as a result of inadequate maternal energy status.

Interestingly, despite the development of hypoglycaemic encephalopathy in the dams of the porencephalic lambs and ketonuria in one of these ewes (3·46 mmol/l), the serum β-hydroxybutyrate (BHB) concentrations of 0·49 and 1·16 mmol/l were within the reference range (0 to 1·2 mmol/l). This may reflect a combination of chronic starvation and physiological adaptation to the poor diet by the ewe, as well as exhaustion of fat reserves, and reduced fetal demand due to low fetal weights. We would welcome colleagues’ comments on this observation.

We suggest that, in addition to in utero virus infections such as Border disease and bluetongue, copper deficiency and hypoxia, other possible metabolic causes of porencephaly exist in lambs, most likely related to severe prolonged maternal energy deficit. Careful examination of the health, nutritional status and management of the ewes, and, where possible, analysis of maternal blood BHB and glucose levels if obtained within 24 hours of giving birth to affected lambs, may be helpful when investigating the cause of congenital neurological disease in lambs.

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