ASVP DIAGNOSTIC EXERCISE NO. 32

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History:
A 3-year-old female alpaca had been imported as a yearling from Australia to New Zealand to an alpaca stud in Northern Taranaki [location map see http://en.wikipedia.org/wiki/Onaero]. The stud is composed of 30 females and 9 males. This animal had developed peeling skin on nose and ears each autumn for the last 2 years. Skin lesions eventually resolved but the alpaca became progressively thinner. In August 2009 the alpaca was circling and showing nervous signs; it was dead by the time the veterinarian arrived. At field necropsy the carcass was in poor condition with few fat reserves, and the liver was described as firm, pale, scarred, and shrunken.

1. Description, histopathology:
Apart from a small remnant group of atrophic hepatocytes to the right of the field, the hepatic parenchyma has been almost entirely replaced by numerous ductules in a loose fibrous tissue matrix. There is no inflammatory cell infiltration.

2. Interpretation:
The dominant disease process appears to be proliferation with degeneration, so neoplasia has to be considered, however the gross description of shrunken liver does not support primary hepatobiliary neoplasia. Fibrosis is expected in chronic inflammation, but the absence of other cellular components of the inflammatory response reduces the likelihood of this being a response to an infectious agent.

3. Pathological diagnosis:
Extreme portal fibrosis and bile-duct proliferation; hepatocellular atrophy; cholangiohepatitis.

4. Aetiological possibilities:
Camelids (the South American ones, at least) seem to have exquisitely reactive portal triads. Thus any agent that can irritate the bile ducts tends to cause this florid over-reaction by bile ductules and associated fibroblasts. Sporidesmin, the toxin responsible for facial eczema, is produced in ryegrass pastures by the fungus *Pythomyces chartarum* in New Zealand and southern Australia under certain seasonal conditions, and is a prime suspect because its irritant properties are directed mostly at the biliary epithelium during its excretion by the liver. But proliferative responses almost as spectacular as this may be seen in chronic liver fluke parasitism in camelids (although it may be difficult to eliminate the possibility that sporidesmin may be involved in these cases). One would, however, expect there to be at least some inflammatory cell infiltration in the parasitic disease.

Because of their susceptibility to sporidesmin, camelids must be carefully managed through autumn in seasons where pasture litter is abundant, overnight temperatures exceed 13^0C and humidity is high (Coulton et al., 1997). For a description of the clinical and anatomical pathology changes see (Smith and Embling, 1991).

Vacuolation was observed in sections of the brain at the interface of the cerebral grey and white matter. This suggests that hepatic encephalopathy was the cause of the clinical nervous signs.

References:
Coulton, M. A., Dart, A. J., McClintock, S. A., Hodgson, D. R., 1997. Sporidesmin toxicosis in an alpaca. Aust Vet J 75, 136-137.

Smith, B. L., Embling, P. P., 1991. Facial eczema in goats: the toxicity of sporidesmin in goats and its pathology. NZ Vet J 39, 18-22.

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