Polioencephalomalacia associated with ingested ammonium sulphate

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The clinical condition known as polioencephalomalacia (PEM) which literally translates to "grey matter softening", occurs when brain swelling is severe enough to cause death of neurones particularly in the cerebral cortex because of the increased pressure in the confined cavity of the skull. Affected animals display a range of symptoms including blindness, a dull unresponsive state, a high stepping gait, stargazing, and occasionally convulsions. Dopey animals may be ridden by other cattle.

The condition is commonly associated with thiamine (vitamin B1) inadequacy or with the presence of thiaminase in the rumen. Unfortunately the term polioencephalomalacia and this thiamine responsive condition commonly are considered synonymous, however several other neurological disorders of toxic or metabolic origin may cause the same lesions e.g. lead poisoning, water intoxication, and sulphide poisoning.

Ammonium sulphate is commonly included in feedlot rations as a urinary acidifier to prevent the formation of magnesium ammonium phosphate bladder stones. The inclusion of ammonium sulphate in the ration has been associated with the development of clinical cases of PEM. The affected animals do not respond to thiamine therapy. Other possible sources of sulphur in the ration include mollasses (high in inorganic sulphur), sulphur containing amino acids and drinking water high in sulphur. All dietary sulphur (organic or inorganic) is metabolised to sulphide in the rumen. Any unbound sulphide is belched up as hydrogen suphide gas. Much of this gas is in turn inhaled and at high enough concentrations is toxic. Several dietary factors may affect the amount of sulphide produced in the rumen and the availability of it for absorption; the amount and form of dietary sulphur, the fibre and fermentable carbohydrate content which will affect rumen microflora and the dietary concentration of metals that can affect sulphide bioavailability.

In studies in the US, experimental feeding of calves sodium sulphate (with a total sulphur content of 0.36% of the diet) resulted in PEM within 21 days. PEM has been observed in Australian feedlot cattle at 20-25 days on feed with a ration containing 1.25% ammonium sulphate (with a total sulphur content of 0.5% of the diet). Interestingly older cattle did not develop PEM after the introduction of ammonium sulphate. This could reflect differences in diet and/or rumen microflora at different stages in the feedlot.

Polioencephalomalcia can be diagnosed at a veterinary laboratory by histopathologically inspecting the brain but this will not determine the cause. No biochemical tests are available to confirm sulphide toxicity, but measurements of total sulphur levels in the diet and drinking water are useful if PEM unresponsive to thiamine is suspected.