PEER REVIEWED

A Review of Polioencephalomalacia in Cattle and Sheep

Polioencephalomalacia (PEM) is also referred to as thiamine deficiency, vitamin B1 deficiency, or cerebrocortical necrosis (CCN). It was first reported in ruminants in 1956 in North America[1]. PEM occurs sporadically in cattle and sheep in many parts of the world. Thiamine deficiency is most commonly seen as clinical affected animals with low thiamine levels upon testing. However, research also exists looking at subclinical thiamine deficiency in ruminants which may be economically significant.

History and Clinical Signs

The clinical signs of acute polioencephalomalacia (PEM) are aimless wandering, circling, hyperexcitability, incoordination, rigid stance, opisthotonos, recumbency, nystagmus, apparent blindness, coma and death[5]. The clinical signs depend upon the species and the stage of the disease. These clinical signs are not specific for polioencephalomalacia, so differential diagnoses need to be considered; lead poisoning, bacterial meningitis, encephalitic listeriosis, viral leucoencephalomyelitis, aflatoxicosis, hypomagnesaemia, enterotoxaemia and pregnancy toxaemia.

In cattle, it is mostly commonly reported in calves, both housed and at pasture. It can be an occasional finding but PEM also occurs in outbreaks which tend to last three to four weeks. Herd morbidity is usually low but if not treated in the early stages of the disease, mortality can be high[2]. Animals most commonly affected are calves of 6–18 months of age. PEM is often diagnosed under the following conditions: calves on poor quality dry summer pasture for some time, or when fed high levels of concentrates or when the sulphur content in the diet is high.

In sheep, PEM is most commonly seen in lambs, 2–7 months of age in late summer on poor quality pasture. Clinical cases in adult sheep generally occur a few weeks after movement to lush pasture. However, a rapid change from an all-forage to high-concentrate diet can also cause PEM. This is due to a shift in the bacterial populations in the rumen which can produce thiaminases which breaks down thiamine in the rumen.

Aetiology and Pathology

Pre-ruminant young animals depend on dietary thiamine. However, in adults with functioning rumens, thiamine is produced by rumen microbes. Thiamine deficiency disease can be caused by the reduced production of thiamine by rumen microbes, or by factors which affect the action of thiamine, e.g. plant thiaminases or thiamine analogues. Thiaminases can be produced by gut bacteria or ingested and can either destroy thiamine or hinder thiamine function.

Thiamine is a component of coenzymes in ruminants including transketolase, pyruvate decarboxylase, pyruvate dehydrogenase and oxoglutarate dehydrogenase. These coenzymes assist in the transfer of an activated aldehyde unit in several energy metabolism pathways. Thiamine deficiency thus inhibits carbohydrate metabolism and results in increased blood lactate, pyruvate and oxoglutarate levels. Thiamine is water-soluble, so its metabolic half-life is relatively short. The main storage organ appears to be the liver; however, half of the total thiamine in an animal is contained within its muscles. Thiamine is excreted in urine and faeces[5].

Thiaminases can be ingested via plants. Bracken fern and rock fern contain an enzyme called Thiaminase I. This enzyme breaks down thiamine. There is a complex balance between thiamine production and thiamine destruction which depends

upon the microbial population in the rumen. It appears that body reserves are sufficient for about three weeks before clinical signs of PEM occur[5].

In the early 1980s, PEM outbreaks were seen in cattle herds fed rations with high levels of sulphate, i.e. >2% sulphate DM, which led to the hypothesis that excess sulphur intake may be associated with PEM. Since then, sulphur-induced PEM has been experimentally reproduced in sheep and cattle. Numerous field cases of sulphur-induced PEM have also been reported in cattle. Although sulphur-induced PEM has been recognised for some time, the role of sulphur in PEM is uncertain[1].

Gross lesions in the brain can be variable depending on the stage of the disease. Acutely affected animals may have brain swelling and coning of the cerebellum due to herniation into the foramen magnum. The initial histologic lesions are necrosis of cerebrocortical neurons. The neurons are shrunken and have homogeneous, eosinophilic cytoplasm.

Requirements

The daily requirement for thiamine varies according to animal species, age, metabolic activity, and feed intake and dietary composition. There are few published data on minimum daily requirements for ruminants. The daily thiamine requirement for adult sheep has been estimated at 2–4 mg, which is similar to estimates of 1–3.5 mg/day for intra-ruminal synthesis of thiamine. Therefore it is possible for sheep to have subclinical thiamine deficiency if the dietary thiamine is low[5].

Adult ruminants synthesise thiamine via rumen bacteria. However, bacterial rumen synthesis of thiamine has been shown to be reduced when sheep or cattle had acute or subacute acidosis. This is often due to diets high in concentrates. Dietary supplementation with thiamine has been shown to increase the pH in the rumen during acidosis[4].

Diagnosis

Diagnosis of PEM includes the demonstration of clinical signs, a likely history of deficiency or thiaminase activity and response to treatment with thiamine.

Blood samples are useful in any suspected case of PEM. 10ml of whole (EDTA) blood should be submitted for thiamine level investigation. When appropriate, a longitudinal section of formalised brain can also be submitted for histopathology.

A normal reference range for thiamine blood levels in ruminants is 50–150 nmol/l. Low thiamine levels are considered to be <50 nmol/l, which indicates likely PEM. However, levels as low as 6–12 nmol/l have been found in suspected cases of PEM[5].

Treatment

Successful treatment of thiamine deficiency depends upon early recognition and diagnosis of the disease. Such animals can respond quickly to one dose of thiamine, often within a few hours. Reports from some who have treated the disease are parenteral doses of 100 mg of thiamine to deficient lambs and 200-500 mg to ewes, calves, and goats. Doses for adult cattle have ranged from 1000–2000 mg. These doses equate to 2–15 mg/kg bodyweight and appear more than adequate to saturate the relevant enzymes and restore their activities[5]. However, a dose rate of 5–10 mg/kg bodyweight of thiamine HCL is most likely sufficient for sheep and cattle where thiamine deficiency is diagnosed or suspected. It should be administered intramuscularly or via slow intravenous injection. Therapeutically

administered thiamine is rapidly excreted. Therefore, multiple injections in a day are recommended (TID or QID). Treatment for approximately three days may be required. If treatment is delayed the damage can be irreversible, and clinical signs persist and worsen. Euthanasia on welfare grounds is a consideration in these cases.

Another treatment option for sheep has been reported in the literature. Oral treatment with 1g of thiamine propyl disulphide was effective in treating PEM and subclinical thiamine deficiency in sheep[6].

Good nursing care is also recommended by keeping the animals calm and away from hazards like wire fences, waterways and steep hills. If recumbent, attempt to keep the animal on its sternum. Make water available or administer fluids as necessary.

Prevention

Ensure that the diet has adequate levels of fibre. Fresh or conserved green forage is also important, particularly in late summer when pasture quality can be poor. Take care with grazing bracken as this is a risk for PEM. Reduce sudden changes in diet from forage diets onto predominantly grain diets and if previously on a rationed diet. Also review the sulphur levels fed offered to animals and potentially water levels of sulphur as this can be another source of sulphur.

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