NADIS Cattle Disease Focus

Hypomagnesaemia (Grass Staggers)

The NADIS data shows that in previous years the number of cases of hypomagnesaemia (grass staggers, grass tetany) increases each spring after turn out reaching a peak incidence in May, with another peak in October.

![Hypomagnesaemia - All Cattle](chart)

What is Hypomagnesaemia?

Hypomagnesaemia occurs when the intake of magnesium is exceeded by its output. The clinical signs can occur very quickly because the cow does not store magnesium, and is thus reliant on its daily dietary intake. Hypomagnesaemia is most commonly a disease of lactating cows at grass, because grass can be very low in magnesium and the output in milk is high. When the losses in the milk exceed the dietary intake, clinical hypomagnesaemia occurs. Like most nutritional diseases, the animals showing clinical signs are just the tip of the iceberg, for every cow with obvious disease many more will be affected sub clinically.

The increase in disease in spring occurs because rapidly growing grasses, particularly rye grasses, are very low in magnesium. This low concentration of magnesium can be further reduced by the application of fertiliser, partly because of the increase in growth rate, but also because application of potassium directly inhibits uptake of magnesium by the grass. Slower growing forages, particularly clovers and other broad-leaved plants contain much higher levels of magnesium.
The significant increase in late autumn, may be partly due to the increase in forage growth that occurs at this time, particularly after a dry summer, and also to the feeding silage produced from spring grass that was low in magnesium.

**Clinical Signs**
The signs of hypomagnesaemia are vary variable ranging from mild apprehension and tremor to sudden death. Hypomagnesaemia is probably the major cause of sudden death in lactating cows at grass. The signs of hypomagesaemia are probably best visualised by detailing the changes in an untreated animal with mild hypomagnesaemia.

1) The animal appears apprehensive with the head held high and a mild tremor.
2) The gait stiffens, and the animal begins to stagger and frequently falls over
3) The animal becomes recumbent (lies on its side unable to get up), with marked convulsions
   Other signs, which may be seen at any of these stages, include watery diarrhoea, skin that is cold to the touch and a low body temperature
4) Death
Often there are signs of struggling evident on the ground around the cow

For every cow obviously affected with hypomagnesaemia, there are many more with marginal magnesium levels. Some of these animals mild show very subtle behavioural changes but the most common effect is a small reduction in milk yield.

**Diagnosis**

- Clinical signs

- Blood magnesium

  *Individual cows*
  For most animals treatment will have to begin before a diagnosis is confirmed as there are no rapid cow-side tests available, but individual measurements can be useful retrospectively in animals where treatment has been unsuccessful

  *Herd*
  As hypomagnesaemia is primarily a herd disease, in most cases it is important to assess the extent and severity of the hypomagnesaemia in the rest of the herd. This is best done by testing six cows at the same stage of lactation as those clinically affected. Care must be taken when handling such animals as stress could initiate clinical hypomagnesaemia.

- Aqueous humour magnesium concentration.
  Samples can be taken by a veterinarian in animals suspected to have died of hypomagnesaemia, however results must be interpreted with caution

**Treatment**

1) All animals that are suspected of having hypomagnesaemia should be immediately treated with 400ml of 25% magnesium sulphate given subcutaneously. A 25% solution of magnesium should never be given
intravenously as it can cause a heart attack. No further action is necessary for mild cases that respond to treatment, but if mild cases do not respond to treatment or if the animal is recumbent a veterinarian should be called.

2) Veterinary treatment usually involves further infusion of magnesium, often intravenously diluted a calcium solution. For severely affected animals a sedative may be given. As both of these treatments are unlicensed they should only be given by a veterinarian.

Following treatment it is important to prevent stimulation for 10-15 minutes to prevent further convulsions. For recumbent animals they should be put on their breastbone and left to get up.

Oral administration of magnesium bullets is recommended to prevent relapses as is moving affected groups to fields with slower-growing pastures.

**Prognosis**

Animals treated at an early stage have a very good prognosis. For recumbent animals, the prognosis is very poor if they do not rise within two hours of treatment and casualty slaughter should be considered.

**Prevention**

1) Feeding magnesium in the concentrates to the cows.

2) Giving magnesium bullets at turn out to cover the period when the animals are most at risk. For some farms, several bullets per cow are needed to prevent hypomagnesaemia.

3) Buffer feed silage with magnesium added

4) Supplement the water supply (this is only effective if the cows have no access to natural water supplies, and may depress water intake)

5) Avoid potassium fertilisers if possible.

6) Adjust grazing sward to a clover/grass mixture

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The Meat and Livestock Commission is a sponsor of NADIS (National Animal Disease Information Service), which is a network of 40 veterinary practices and 6 veterinary colleges monitoring diseases in cattle, sheep and pigs in the UK.