Poisoning in Cattle (non – plant)

Lead poisoning

Lead poisoning follows ingestion of lead-containing materials such as material from discarded car batteries; lead-based paints are now rare.

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Clinical presentation

Lead poisoning is characterised by acute brain disease. Irrespective of the rate of uptake of lead, the clinical signs of intoxication are sudden in onset and characterised by behavioural changes. Affected cattle become isolated and depressed but are over- reactive to touch and sound. They are blind but show no clinical lesions in the eyes and may press the head into corners and against walls.

Cattle with lead poisoning become isolated and depressed

As the disease progresses, cattle become frenzied, bellow, stagger and crash into obstacles. There may

be signs of abdominal pain including kicking at the abdomen and frequent teeth grinding. Death may occur suddenly or within days.

Cattle with lead poisoning may show head pressing behaviour.

In the advanced stages of lead poisoning, cattle become frenzied, bellow, stagger and crash into obstacles.

Differential diagnoses

Your veterinary surgeon will also consider:

- Hypomagnesaemic tetany (staggers)
- Polioencephalomalacia
- Rabies

Diagnosis

The diagnosis of lead poisoning is based upon clinical signs and the presence of a lead source. Confirmation depends upon increased kidney and liver lead concentrations.

Treatment

Cattle with severe neurological signs of several days' duration may have extensive brain pathology that is unlikely to respond to treatment. Slow intravenous injections of a 5% solution of sodium calcium edentate and oral solutions of magnesium sulphate are the treatments of choice but are rarely successful once severe neurological signs are present. Your veterinary surgeon will decide whether treatment or euthanasia for welfare reasons is the better course of action, and which drugs to administer.

Prevention/control measures

Prevention is a matter of good management by not allowing cattle access to sources of lead. Regulatory authorities, such as the Food Standards Agency, may recommend withdrawal periods before milk and/or meat from exposed cattle may enter the food chain.

Copper poisoning

Toxicity may result from inadvertent dietary supplementation or incorporation of a feedstuff with a high copper content, possibly as the result of contamination. Acute toxicity is rare. Copper toxicity in cattle is much less common than in sheep. Ingestion of high copper content in the ration over several weeks/months will result in a high liver copper content. Sudden release causes an acute intravascular haemolytic crisis.

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Clinical presentation

Acute copper toxicity causes severe gastro-enteritis with colic signs, diarrhoea and rapid dehydration. The affected cattle are very depressed, do not eat, and death usually ensues within three days.

In cases of chronic copper toxicity the appearance of clinical signs is associated with the destruction of red blood cells which may be precipitated by stress. Affected cattle are weak, very dull and depressed and separate from others in the group. They have a poor appetite and often foetid diarrhoea. There is jaundice (yellowing) of mucous membranes. Death is preceded by recumbency.

Differential diagnoses

Your veterinary surgeon will also consider

- Babesiosis
- Post-parturient haemoglobinuria
- Kale poisoning.

Diagnosis

The diagnosis is based upon history, a source of excess copper, and clinical findings of jaundice in chronic toxicity. The diagnosis is supported by laboratory findings of increased serum copper and liver enzyme concentrations.

Treatment

The suspected copper source must be removed immediately. There is no ammonium tetrathiomolybdate preparation licensed for use in food-producing animals and its use in suspected cases of chronic copper toxicity is poorly defined from a regulatory standpoint.

Prevention/control measures

Copper supplementation must be carefully considered after first establishing a deficiency situation.

Fluororis

Historically, fluorosis resulted from industrial pollution of grazing land where chronic disease results after ingestion over many months/years but is rare in the UK.

Clinical presentation

Growing cattle develop mottling of the enamel and premature loss of teeth. Adult cattle show insidious onset lameness affecting the hindlegs caused by periarticular exostoses of the long bones. Fracture of the pedal bone may occur but there are other more common causes of such sudden inset lameness. There is no specific treatment.

Nitrate poisoning

Brassica plants and fertilisers are potential sources of nitrates.

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Fertilisers are also potential sources of nitrates. Note storage of fertilisers adjacent to cattle feed- there is the possibility of feed contamination.

Clinical presentation

Acute poisoning with cyanosis, weak rapid pulse, and dyspneoa, is seen within hours of ingestion progressing rapidly to weakness, recumbency and death.

Differential diagnoses

Causes of sudden death your veterinary surgeon will consider include

- Hypomagnesaemia
- Lightning strike

Diagnosis

Diagnosis is based upon classical clinical signs and exposure to nitrates.

Treatment

Intravenous injection of 4 mg/kg methylene blue as a

2 per cent solution.

Organophosphate poisoning

Overdosage and accidental exposure to organophosphates lead to toxicity.

Failure to correctly dispose of concentrated dip(not an OP in this case) can lead to toxicity.

Clinical presentation

Profuse salivation, colic and diarrhoea are followed by muscle tremors, stiffness progressing to paralysis. At this stage cattle show marked depression with increasing severity of colic, sweating and breathing difficulties followed rapidly by death.

Diagnosis

Based upon clinical signs and history of exposure to/treatment with organophosphates.

Treatment

Atropine sulphate (0.1 mg/kg injected slowly intravenously followed by 0.4 mg/kg injected subcutaneously) is repeated as necessary.

Prevention/control measures

Correct storage and disposal of empty containers containing organophosphates.

Urea Poisoning

Definition/overview

Urea is used as a source of non-protein nitrogen (NPN) in feed supplements especially in beef cattle. Nitrogen from urea is released in the rumen as ammonia in the rumen. Accidental urea intoxication occurs sporadically and can cause severe losses. One incident resulted in the death of 17 of 29 suckler cows within six hours after the contamination of their drinking water with urea fertiliser.

Aetiology

Poisoning episodes typically occur after sudden access to urea, which may simply involve only a break of several days' supply then free access. Urea is highly soluble and will wash out of the diet/feed blocks following heavy rain then cattle may drink the puddles with a high urea content.

Clinical presentation

Signs of urea poisoning can appear within several hours and include muscle twitching, teeth grinding, frothy salivation, bloat, colic, frequent urination, forced rapid breathing, staggering, bellowing, and terminal seizure activity. Often, animals are found dead near the source of the urea supplement.

Differential diagnoses

Your veterinary surgeon will also consider:

- Botulism
- Hypomagnesaemia
- Anthrax
- Clostridial disease such as Blackleg

Diagnosis

Diagnosis is based upon a history of access to urea, and the clinical signs.

Treatment

50 litres of cold water then several litres of 6 per cent vinegar have been recommended.

Prevention/control measures

- Ensure thorough mixing of the ration.
- Gradual introduction to urea feeding with an uninterrupted supply

To see the full range of NADIS livestock health bulletins please visit www.nadis.org.uk