Workshop - TRACE ELEMENTS

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Three practitioner cases were presented.

Case 1: Copper poisoning in deer

History

Twelve mixed age fullmouth, Wapiti cross hinds were involved. They were animals that had previously been caught in Fiordland (12-18 months earlier), transported to Motueka and grazed on a small property in an area known to be marginal for copper. They were on a mainly grass diet and supplemented with fodder beet. No topdressing had been used on the place by the present owner.

Immediately prior to the poisoning episode they were given a routine anthelmintic drench containing selenium and a copper EDTA/KI mixture (of unknown composition).

Clinical findings

1 hind died 7 hours post drenching
2 hinds died within 48 hours
1 hind died 5 days after
All remaining hinds showed diarrhoea and inappetance for some days.

Post mortem results

Gross haemorrhagic areas throughout lungs, muscles and intestines, similar to bracken fern poisoning.

Histopathology - abomasum: extensive haemorrhagic abomasitis.
liver: necrosis and haemorrhage
kidney: degenerative changes of the tubular epithelium.
lung: alveolar and interstitial haemorrhage.

Bacteriology - no salmonellae isolated
no yersinia isolated
no campylobacter isolated

Biochemistry - serum copper a) 14.09 umol/L
b) 24.3 "
c) 14.9 "
serum selenium
a) 5900 nmol/L
b) 1700 "
c) 3300 "

liver copper a) 3242 umol/kg
b) 1326 "

kidney copper a) 837 umol/kg
b) 232 "

drench copper a) 252,770 umol/L or 16,100 mg/L

Discussion

The copper levels considered toxic are:-

Liver 200mg/kg or 3400 umol/kg
Kidney 10mg/kg or 170 umol/kg

The kidney levels in this case would be considered toxic although the liver levels may not be. Perhaps the acute nature of this outbreak would explain this.

The histological findings were consistent with, and typical of copper poisoning.

A check of the drench gun and the drenching technique indicates that a 20ml dose of the drench was administered, i.e. approx. double dose.

A quick calculation shows that 300mg of copper would have been given, i.e. approx. 2½ times the normal copper dose we recommend.

Conclusion

Although we don't know the copper status of these deer prior to the drenching it would appear from the history that it is unlikely that the copper levels were unduly high.

This could indicate that the copper EDTA used is readily absorbed and highly toxic to deer.

This case highlighted that the toxicity range of liver copper values can be wide and that kidney is the tissue of choice in investigating suspected copper toxicities. The rate of presentation of copper to the liver in the individual animal is more important than the actual amount.

Case 2: Copper deficiency in deer

Farm A

Soil type: swampy loam, known to be low in copper, no molybdenum topdressing.

Signs of poor condition and ataxia first seen in January in four yearling hinds. These animals were given copper with no obvious improvement in the condition. During the winter one adult hind appeared ataxic and was post-mortemed. Histologically, the changes in the spinal cord were typical of enzootic ataxia.

Blood samples taken in August had a mean serum copper level of 1.2 umol/litre. After copper treatment the mean serum copper levels one month later were 7.69 umol/litre.

The affected yearlings were kept until two year olds with an improvement in body condition but not gait.

Farm B

Unweaned fawns noticed in February in poor condition with rough coats and difficult movement in which both hind legs were brought forward at one time with arched backs, clinically similar to acute white muscle disease. On yarding one fawn fractured a leg without an obvious accident. Several fawns had an abnormal configuration of facial bones. On post mortem a severe degenerative arthritis was found, particularly noticeable in the hock and elbow joints. Histological examination showed poor bone formation.

The mean serum copper level was 3.3umol/litre. Two liver samples had copper levels of 1.3 mg/kg and 2.4 mg/kg respectively.

With copper injection a marked improvement was apparent in gait and condition over several months. The worst affected had slight gait problems and misshapen heads one year later. One liver sample taken four months later had a copper value of 6.2 mg/kg.

Doubts that treatment may need to be more frequent were followed up on several properties with monthly blood samples. At times levels were so low as to give no recordable value, this brought into question the persistence of copper treatment.

This case study highlighted the importance of analysing whole farm situations with reference to available pasture copper levels and animal tissue copper levels. The ultimate objective being to farm the deer with an emphasis on copper sufficency rather than deficiency.

Case 3: Copper deficiency in deer

In February 1984 a deer farmer complained of mixed age fawns being very lame. They had stunted growth rates, long haired rough coats rusty red in colour. The hock joints of these calves were swollen and the fawns walked as if on "hot bricks". There was no evidence of any infectious foot conditions.

A tentative diagnosis of copper deficiency was made and severely affected fawns were blood sampled.

Blood	Copper umol/litre	Selenium nmol/litre
1	3.4	
2	less than 3	270
3	less than 3	260
4	less than 3	
5	7.2	
6	3.9	130

Examination of the radiographs demonstrated changes consistent with disruption to the growth pattern of the growth plates.

The fawns were initially treated with an oxfendazole drench to which a proprietary mineral premix was added. When the diagnosis was confirmed the fawns were given 50mg copper by injection with variable response. One month later the fawns were again injected with 75mg copper as well as 10mg elemental selenium. This resulted in a noticeable improvement in serum copper and selenium levels. The mean copper serum level recorded in May 1984 in the group of fawns was 14.5 umol/l.

At this sampling time many of the most affected fawns were difficult to recognize from their herd mates.

The farm is on developed peat soil with no history of any mineral deficiency. The hinds were fawned in two groups on different parts of the farm. Only one group of fawns was affected. Pasture analysis results as follows:

	Copper mg/kg	Iron mg/kg	Molybdenum mg/kg	Sulphur %
1	12	110	2.0	0.37
2	8	110	4.1	0.32

The farm has since been treated with selenium prills and copper sulphate with potassic serpentine superphosphate at 300 cwt/acre.

General discussion

The best tissue sample to obtain in evaluation copper sufficiency is liver. The cattle liver biopsy technique is directly relevant to deer and is vastly superior to analysis of dead animal liver tissue. Anorexia in sick animals may rapidly deplete liver copper reserves and give a false low level. Deer are born with naturally higher liver reserves than other species of animals and this may explain the development of enzootic ataxia in older groups of animals, rather than the congenital or neonatal syndrome normally seen in sheep for example.

Copper has the potential, as has been shown, to be toxic. Starved animals have a three or four fold increase in uptake of copper by liver thereby enhancing the potential of toxicity. The optional dose rate under standard situations is 1.0mg/kg.

The workshop concluded by learning of an interesting macroelement problem seen in weaners kept indoors in Southland.

Ten members from a group of seventy weaner fawns developed signs of an osteodystrophy about one month after being housed indoors. The initial sign was an enlargement of the carpal joints, this was followed by a bowing of both front legs. As the disease progressed the hindleg joints became involved and varying degrees of lameness were present. The weaners were fed haylage and 1kg barley per head.

Successful treatment was effected by adding Ca CO_3 to the diet and dosing with a commercial multi-vitamin preparation at regular intervals. The palatability of the additives was increased by mixing with oilseed rape.

When lucerne was fed instead of haylage the problem was evident but not to the same degree.

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