A case report : Preliminary observations of severe clinical copper deficiency in farmed red deer (Cervus elaphus)

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Introduction

Copper deficiency (Cu-deficiency) is common in livestock in New Zealand, and is a widespread health problem on deer farms (Familton et al., 1985; Harrison et al., 1989; Killorn and Wilson, 1990; Wilson et al., 1993). In farmed deer, it is commonly associated with enzootic ataxia (Wilson et al., 1979; Mackintosh et al., 1986a). However, a number of other clinical or subclinical syndromes such as anaemia, light-coloured coat, poor condition, and low growth rates can be associated with Cu-deficiency. Recently, Cu-deficiency has been associated with skeletal abnormalities including osteochondrosis, weakness of the physes of long bones, and osteoporosis in farmed deer (Thompson et al., 1993).

Osteochondrosis affects cartilage in various joints, and may result in the production of a dissected flap of cartilage in the joint associated with inflammatory changes (OCD)(Fox and Walker, 1993). OCD affects young growing animals including dogs, horses, pigs, sheep, cattle and deer. In dogs, there are multiple predisposing factors (Fox and Walker, 1993), but a definitive causative factor has not been identified. However, a relationship between Cu-deficiency and OCD has been found in sheep (Pitt et al., 1980), cattle (Smith et al., 1975; Suttle and Angus, 1978), pigs (Pond et al., 1990), and horses (Bridges et al., 1984; Gunson et al., 1982; Bridges and Moffitt, 1990) and that association has been suspected in deer (Brooks, 1984; Thompson et al., 1993).

This paper reports the clinical and epidemiological observations of an outbreak of osteochondrosis, OCD, poor skeletal development and enzootic ataxia in red deer on a severely copper deficient deer farm which was monitored as part of a two-year observational health and production study (Audigé et al., 1993).

The farm characteristics

The property was located in the Northern Wairarapa area of the North Island of New Zealand. 48 mixed-aged stags, 150 mixed-aged hinds, and 64 weaner deer were farmed on a 45-Ha fenced block which is part of a 520-Ha sheep and beef unit farming 2300 ewes, 1400 hoggets and 100 steers in June 1992.

Four of 11 deer paddocks were located on steep hill pasture of browntop and other grasses with very little clover content. The remainder were flat and pastures were predominantly ryegrass and white clover, with some cocksfoot and browntop.

Animal management

Hinds were set stocked onto the hilly paddocks for calving from November 1991 to weaning in March 1992. They had spent most of the winter season on those paddocks. In March 1992, 83 calves were weaned, but 15 other calves remained in the calving paddock until April 1992.

The 150 hinds grazed these paddocks almost exclusively from May 1992 until weaning in March 1993. They were given 3 bales of clover hay per day from July 31st to August 10th. The pasture was grass dominant all winter and very short; from grazing management records, the average height of the pasture from June to November was 5.7 cm. Hinds were set stocked for calving on the same hilly paddocks on November 7th.

Clinical observations

The chronological sequence of clinical observations and investigations is presented in Table 1.

The farmer noticed swelling of the hocks in 5 weaner hinds in February 1992. They were killed on the farm without veterinary examination in April 1992. Some joints including hip joints were later examined and osteochondrosis of the hip joint diagnosed in one deer. Four of the other 15 weaner deer which were not mustered in March were noticed to have swollen hocks or carpal joints in April 1992. Overall 8.5% of calves had clinically affected joints at 3 to 4 months of age.

Results of laboratory investigations of serum and liver copper levels are presented in Table 2. Radiographs have been presented elsewhere (Thompson et al., 1993).

On November 7th 1992, two hinds became severely uncoordinated. Similar signs were shown by an adult stag on December 3rd and another hind on December 25th. The farmer's veterinarian was called in early December for further investigations. Copper deficiency was diagnosed by serum copper measurements from 6 mixed-aged hinds. Between December 1992 and January 1993, 12-month-old and mixed-age deer were given 10g and 20g, respectively, copper bullets ("Copacaps" - Rhône Mérieux]. Calves were given 5g copper needles on January 22nd, 1993.

As early as mid-December 1992, the farmer noticed lame calves with swollen hocks, but it was not until late January 1993, that these could be mustered. On January 22nd, fifteen out of 125 calves showed clinical signs of osteochondrosis or poor bone development (figure 1). Two were severely affected (Table 2, Figure 2). Clinical signs varied between calves and included poor condition, dull coat, swollen hocks and carpal joint, "cow-hocked" stance, and lameness. Blood samples were taken from ten calves (including 3 clinically affected) for copper measurement.

Figure 1 : A typical example of an affected calf showing poor condition, dull coat, swollen carpal joints and hocks

Figure 2 : Severely affected fawn with the "cow-hocked" stance, characteristic of fracture and dislocation of the hip

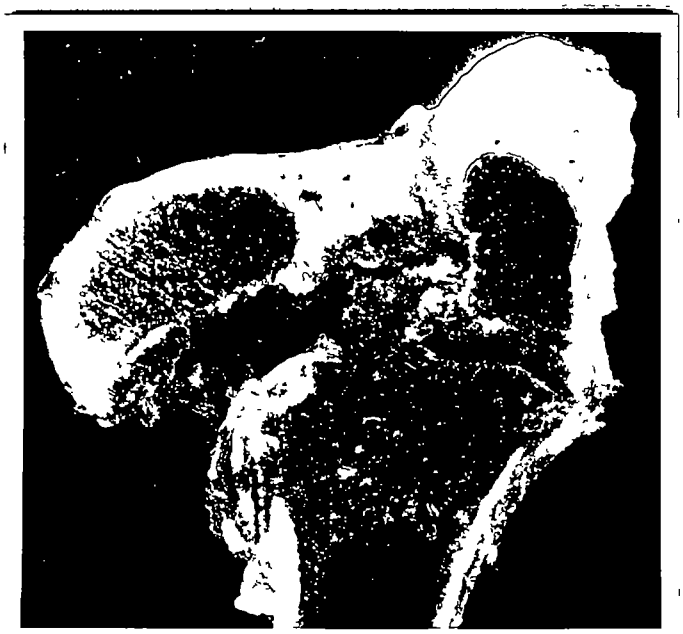

Figure 3 : Epiphyseal fracture of the femoral head in a severely affected 2-month-old deer

Table 1 Chronological sequence of clinical observations and investigations from February 1992 to June 1993

	Date	Number, type and age of affected deer	Mob size	Clinical signs	Action/Investigation
Case 1 (Farmer diagnosis)	Feb 92	5 hinds (3 months)	103	Lameness, swollen carpal joints and hocks	5 WH killed on farm in April 92, some backleg joints examined
Case 2 (Vet diagnosis)	April 92	2 hinds 3 stags (5 months)	15[1]	Lameness, swollen carpal joints and hocks	Mob separated from other weaners Serum copper, June
	Sept 92	2 stags (10 months)		Lameness, bent front knee, swollen carpal joints and hocks "Cow-hocked" posture	Taken to Massey on Sept 15, Serum copper on Sept 20 & 25 Liver copper on Sept 25 Radiographs Post-mortem examination of joints Histology joints and spinal cord
Case 3 (Vet diagnosis)	Nov 92 Dec 92	3 adult hinds 1 adult stag	150 MAH 48 MAS	Ataxia - Incoordination	Serum copper from 7 AH (including 1 affected mixed-aged hind) Histology cervical and lumbar spinal cord from 2 affected hinds sampled at DSP[2] in May 1993
Case 4 (Vet diagnosis)	Jan 93	15 calves (2 months)	125	Lameness, swollen carpal joints and hocks, "Cow-hocked" stance	Serum and liver copper Post-mortem examination Histology joints and spinal cord
	May 93	11 weaners (5 months)		Swollen carpal joints or hocks (6 deer) Fracture hind leg (1 deer) "Cow-hocked" stance (5 deer) Swollen fetlock (1 deer)	Serum copper from 7 affected and 10 unaffected weaners Post-mortem 2 WS for liver copper and histology, joints and spinal cord
	June 93	9 weaners (6-7 months)		Swollen carpal joints or hocks (3 deer) "Cow-hocked" stance (4 deer)	Serum copper from 10 unaffected weaners

WS = Weaner stag; WH = Weaner hind; MAH = Mixed-aged hind; MAS = Mixed-aged stag
1 = Weaners left behind in hilly paddocks in March at weaning
2 = DSP · Deer Slaughter Premises

Table 2 Chronological sequence of laboratory analyses from affected deer

	Class of deer	Date	Serum copper, (µmol/l)	Liver copper (µmol/kg)	Post-mortem and histology results
Case 1	Weaner	April 1992			Osteochondrosis lesion on one coxo-femoral joint
Case 2	4 Weaner clinically affected	June 1992	16.0 8 6 15.0 11 0		
	2 Weaner (arrived at Massey on the 15.9.92)	Sept 1992	Dates of sampling Sept 20 Sept 25 No61 5.9 8 9 No62 5 9 9 5	53 39	Weaner No62 Severe degenerative arthropathy of coxo-femoral joints associated with lesions of osteochondrosis in hocks and fetlock joints, Weaner No61 . Billateral osteochondrosis lesions in hocks, stifle and elbow joints, demyelination of the ventro-medial and dorso lateral tracts of the wite matter in the cervical spinal cord, increasing in severity caudally, to also involve the lateral tracts in the lumbar cord
Case 3	6 M.A hinds	Nov 1992	1 0, 1 8, 3.0, 5 7, 5.8, 7 8		
Case 4	10 Fawns (* and ** clinically affected calves)	Jan 1993	0 0*, 0 1, 0.2, 0 3, 0 4, 0 8**, 1.2, 1.6*, 2.3, 4 1 ** Necropsied calf	25	No3 . Severe chronic bilateral osteochondrosis of the coxo-femoral joints with bilateral femoral head fractures and secondary chronic osteoarthropathy No evidence of nervous lesions in the spinal cord
	17 Weaners (* seven clinically affected)	May 1993	Weaner 259 14 6 Weaner 297 18.2 2.7, 2.9*, 4 4, 5.1, 6.2*, 6.9, 7.3, 8.0, 8.0, 8.1*, 8.1*, 8.5, 10 0, 10.5, 11 7*, 14 6*, 18 2*	624[1] 348[1]	Weaner 259 : osteochondrosis with secondary degenerative osteoarthropathy (hocks and fetlocks). No evidence of demyelination in the spinal cord. Weaner 297 Fractured left femoral head, cartilage erosion on left tarsal joint No evidence of demyelination in the spinal cord
Case 3	2 Adult hinds	May 1993			Both hinds had severe demyelination of dorso-lateral and ventro-medial white matter tracts in all sections of the spinal cord and the peripheral white matter tracts of the brain stem.

1 = 2 5 months after treatment with 5g copper oxide wire particles

During sampling, one calf died and was post-mortemed (Tables 2 and 3, Figure 3). Gross and histological lesions are described in the previous paper in this proceedings (Thompson, 1993).
Figure 1 : A typical example of an affected calf showing poor condition, dull coat, swollen carpal joints and hocks

The weaner deer were subsequently monitored for serum copper levels and clinical signs. On the 7th of May, two severely affected deer were humanely killed and post-mortemed (table 2).

Pasture analyses

Pasture samples were taken in March and September 1992 from both flat and hilly paddocks; samples were washed and oven dried before being analyzed for micro-nutrients levels. Data are presented in Table 3. Because of the lack of reference values of pasture mineral requirements for farmed red deer, interpretation can only be made on the basis of cattle and sheep data (Grace, 1983). All micronutrients appeared adequate in March 1992. By September 1992 pasture sulphur, molybdenum and iron content had increased significantly, while pasture copper levels remained constant at about 8 ppm.

Table 3 : Pasture micronutrient profiles

Paddock	Date	Sward height (cm)	S %	Mn ppm	Zn ppm	Cu ppm	Fe ppm	Co ppm	Mo ppm
Hill No3	March	13	0.18	396	28	6	234	0.19	0.61
Flat No5	1992	22	0.29	113	35	10	130	0.07	0.88
Hill No9		9	0.23	374	31	8	186	0.11	1.12
Hill No1	Sept	5	0.32	195	41	7	394	0.19	3.56
Hill No3	1992	3	0.26	325	81	9	2886	0.80	1.42
Flat No6		6	0.34	248	38	8	1035	0.35	1.34
Ref[a]	Sheep		0.15	25	25	5	30	0.08	0.05
	Cattle		0.18	25	25	7	40	0.04	0.05
	toxic		0.40[b]	400	900	20	500	35	2[b]

[a] Mineral requirements for cattle and sheep (Grace, 1983)
[b] Value given by MAF, Ruakura AHL, Hamilton

Soil analyses

Four paddocks were soil sampled in June and analyzed by the Massey University Soil Fertility Service for pH, phosphate, sulphur, potassium and magnesium (Table 4).

Table 4 : Soil analysis results

Paddock	Soil pH	Olsen P	Sulphur	Exch.K	Mg
Hill No 2	5.7	23	11.0	0.60	2.21
Hill No 3	5.9	24	15.0	0.89	1.09
Flat No 6	5.7	36	15.3	1.19	1.83
Flat No 8	5.6	25	15.0	1.26	2.98
"Normal" range[1]	5.6-6.4	>20	>7	>0.4	>0.35

1 Fertilizer & Lime Research Centre, Massey University

Further analyses

As part of a whole herd health and production profile research project, randomly selected weaner deer, hinds and stags were blood sampled to investigate a wide range of haematological and blood biochemical characteristics as detailed elsewhere (Audigé et al., 1993). Serum copper was determined by atomic absorption analysis from frozen sera. Results are presented in Table 5. As there were no sex differences, data were pooled for further analysis and presentation.
Highest serum copper values were observed in June and November with values below 8 µmol/l in less than half of the deer; lowest serum copper values were encountered in March and in September with almost all values below 8 µmol/l. Further data when available on haematology, and weight data will be presented elsewhere.

Table 5 : Serum copper (µmol/l) of stags, hinds and weaner deer sampled from March 1992 to June 1993

	MARCH 1992		JUNE 1992		SEPT 1992	
	Hinds n=5	Weaners n=10	Stags n=10	Weaners n=11	Hinds n=7	Weaners n=10
Minimum	7.9	2.2	8.4	5.6	1.0	2.1
Maximum	14.0	13.0	16.0	14.0	2.4	7.4
Mean	12.0	5.3	12.5	8.9	1.5	4.2
% < 8 µmol/l	20	90	0	45	100	100
	NOV 1992		MARCH 1993		JUNE 1993	
	Stags n=5	Weaners n=10	Hinds n=10	Weaners n=10	Stags n=10	Weaners n=10
Minimum	3.2	3.9	8.2	1.4	3.1	2.6
Maximum	19.0	17.0	15.0	7.7	13.5	7.1
Mean	8.9	10.1	12.2	4.4	9.4	4.7
% < 8 µmol/l	60	30	0	100	40	100

Discussion

Osteochondrosis or development of bone abnormalities associated with primary or secondary copper deficiency has been described in horses (Bridges and Harris, 1988; Bridges and Moffitt, 1990), in sheep (Pitt et al., 1980), in cattle (Smith et al., 1975; Suttle and Angus, 1978), in pigs (Pond et al., 1990) and in deer (Brooks, 1984; Thompson et al., 1993).

The diagnosis of osteochondrosis in January 1993 confirmed the recurrence of the syndrome on the property with a higher clinical prevalence (12% cf 8% in 1992]. Clinical and pathological observations were consistent with poor condition, lameness,

swollen joints due to osteochondrosis, and postural abnormalities associated with femoral epiphyseal fractures. Although no radiographs or post-mortems could be performed early on apparently healthy calves, a proportion of calves may have been affected by subclinical lesions of osteochondrosis. Such lesions were observed on the 3-week-old progeny of swine that were Cu-deficient during pregnancy (Pond et al., 1990). The observations of affected calves in December and severe lesions in January suggest that bone and cartilage growth abnormalities may have developed during pregnancy, and after birth, associated trauma may have triggered the splitting of weight-bearing cartilage in articulations or epiphyses. Brooks (1984) described the syndrome in 3-to-4-month-old calves, but lesions may have developed earlier.

The diagnosis of 4 cases of enzootic ataxia in November 92 was made on the basis of previous descriptions in red deer (Wilson et al., 1979; Mackintosh et al. 1986)(Fig 1). Confirmation was by histological lesions in the spinal cord and the known low copper status of the farm. Surprisingly, no clinical cases of enzootic ataxia appeared in the mob of apparently healthy weaner deer, despite low serum copper levels recorded in September. However, one weaner stag, which had severe lesions in the coxo-femoral joints in September (Thompson et al., 1993), was also affected by extensive spinal cord demyelination characteristic of enzootic ataxia (Table 2; case 2]. This observations supports the suggestion that Cu-deficiency may be only one of many factors that predispose to the occurrence of enzootic ataxia in deer (Wilson et al., 1979; McTaggart et al., 1981). It is interesting to note that severely Cu-deficient calves had no lesions in the spinal cord in January.

It was confirmed early by monitoring programme that the property was Cu-deficient (Table 5], as the normal range for serum copper level in farmed deer has been estimated to be 8 to 22 µmol/l (Clark and Hepburn, 1986). Serum copper levels on mixed-aged deer were considered marginally low. The decrease in serum copper levels in deer of all age group during winter, and a progressive increase later in spring, is consistent with results of previous serum copper monitoring (Harrison and Familton, 1992) and similar investigations on 14 other red deer farms (Audigé et al., unpublished data). Although liver copper reserves were not investigated in mature hinds, they were likely to be very low in September (Mackintosh et al., 1986b).

No clinical signs of copper deficiency were observed on mature deer between March and November despite the low copper status of the farm. In cattle and in sheep, plasma copper levels below 9 µmol/l have been reported as marginal, but values drop to less than 3 µmol/l before dysfunction occurs (Suttle, 1986).

Although anorexia may have depleted liver copper reserves, the serum and liver copper concentrations that were measured on the calves in January 1993 indicate a very low copper liver storage and supply. In farmed deer, calves at birth usually have higher liver copper (up to 5589 µmol/kgWM] concentrations than adult deer (165 µmol/kgWM] which should cover their requirements for copper for the first months of life (Reid et al., 1980). The

observation of clinical disease reported here conflicts with the previous belief that calves are well protected against low copper status at birth despite the low adult liver Cu levels (Reid et al., 1980). In this case, hinds were unable to adequately supply their offspring with copper during the last months of pregnancy.

Copper levels in pastures (6-10 ppm) are considered adequate. Therefore, this deficiency is secondary to impairment of the absorption or utilisation of Cu by other micro-nutrients such as Mo, S, Mn, and Fe. High dietary Mo or S inhibits copper metabolism in deer (Mason et al., 1984; Freudenberger et al., 1987; Osman and Sykes, 1989) by forming insoluble complex salts (thiomolybdates) in the rumen. On this farm, it is suggested that the high S and Mo levels in the pasture may be the principal causative factor inducing copper deficiency.
High dietary iron levels may have also reduced the absorption of copper (Grace, 1983). The apparent high iron content of the pasture may be partly from soil contamination, as sward heights in September were very short (Table 3), but deer were likely to ingest large amount of soil while grazing. Manganese levels were marginally high, thus may have impaired further absorption of copper. As soil pH was marginally low (table 4), manganese absorption by plants was increased.

So far, the causative role of copper in the development of skeletal abnormalities in growing deer, and enzootic ataxia in mature deer has not been fully explained. The chronological sequence of observations in this report, however, suggests a cause-effect relationships. Experimentally induced Cu-deficiency in foals (Bridges and Moffitt, 1990) resulted in a similar syndrome to that encountered on this property in deer. Copper is an essential cofactor for lysyl oxidase, an enzyme involved in the formation of collagen cross-links (Seigel et al., 1970). Its deficiency will result in weak connective tissue, thus increasing the risk of cartilage split and bone fractures.
Affected deer become lame, thus unable to meet their nutritive requirements. Thus, liver copper concentrations reported in case 2 (Table 2), although very low, should be interpreted cautiously since the low levels may have been exacerbated by reduced copper intake. In this case, serum copper levels may have increased 5 and 10 days after arrival at Massey University because of a change in diet.

Because affected calves may remain undetected until handled, prevention strategies and treatment would generally be implemented post-weaning. This is too late in many cases. Copper supplementation of affected animals may lead to improvement of the general condition (Eamens et al., 1984), but will not cure existing lesions (Smith et al., 1975).

Copper supplementation of mares during late pregnancy appears to reduce the prevalence and severity of osteochondrosis and other cartilage lesions in foals (Knight et al., 1990). This suggests that hinds should be supplied with adequate copper levels during the last months pregnancy.

In this farm, 5g copper oxide wire particles were given to each calf in January. Despite the normal liver copper values recorded

from two affected deer in May, the serum copper levels of apparently healthy deer in March 1993 and in June 1993 suggest that this copper supplementation was not sufficient. Calves should have been given higher or more frequent doses of copper. Adult hinds appear to have reached adequate copper levels by March 93, but a proportion of adult stags had low levels in June 93. The farmer supplemented all deer with copper needles again in June 1993. It is expected that copper levels will be satisfactory during winter, thus preventing the recurrence of the syndrome in the calves next year.

At the time of writing, no more clinical case of enzootic ataxia had developed, and copper supplementation should prevent it providing copper levels will be adequate in mature deer. This report shows that lesions of enzootic ataxia can develop in deer between 2 and 9 months of age. The reason why enzootic ataxia is not observed in weaner deer is unknown.

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