OSTEOCHONDROSIS ASSOCIATED WITH COPPER DEFICIENCY IN DEER

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Introduction

Osteochondrosis is a generalised disturbance in endochondral ossification affecting rapidly growing young animals of several species, including pigs[1][2], dogs[3][4], horses[5] and cattle[6]. Affected animals usually present with lameness associated with focal or multifocal defects in the articular cartilage of major weight-bearing joints[1][4][6][7]. Joint lesions are often bilateral and vary from foci of thickened, wrinkled articular cartilage to loose flaps of cartilage which have become separated from subchondral bone, and may even detach to form loose fragments or "joint mice"[1][6][7][8]. Because of the dissecting nature of these lesions and an associated inflammatory response in the synovium, the syndrome is often referred to as osteochondritis dissecans. Irregular thickenings may also be present in the physes of long bones, especially in pigs and horses[6][8].

The pathogenesis of osteochondrosis is poorly understood but probably involves the trauma of weight-bearing superimposed on rapidly growing cartilage, which may or may not possess an underlying matrix abnormality[9]. Although deficiency of a specific nutrient is not generally considered to cause osteochondrosis, primary and secondary copper deficiency has been implicated as a predisposing factor in suckling foals[10][11][12][13]. During the last eight years a syndrome characterised by lameness, swollen joints and abnormal gait in young red deer and wapiti/red deer hybrids has been recognised on several deer farms throughout New Zealand. Consistent findings in affected animals have included joint lesions typical of osteochondrosis and very low serum and/or liver copper concentrations. The purpose of this paper is to describe the clinical signs and pathology of osteochondrosis in farmed deer and suggest an association with copper deficiency in this species.

History and Clinical Findings

The association between copper deficiency and osteochondrosis in young deer has been established on at least eight farms throughout New Zealand. In most cases the farm or area was either known or suspected by the veterinary practitioner to be copper deficient. One farm was adjacent to a fertiliser works and the farmer commented that his pasture was sometimes covered with yellow dust, presumably sulphur, which is know to cause secondary copper deficiency[14].

Most affected animals were red deer but on one farm the syndrome occurred in red deer/wapiti hybrids. Signs of lameness were noticed in calves as young as one month of age, but more commonly, the problem was not evident until the calves were weaned at around four months of age.

The prevalence varied considerably between farms but was consistently greater than 7% and on two farms more than 30% of calves were affected to some degree. Some of the farmers had had similar problems in previous seasons but in most cases had not requested veterinary investigation. One farmer, knowing that his farm was copper deficient, had treated his pregnant hinds each year with copper oxide needles and had not observed any affected calves. The syndrome occurred on his farm for the first time in the season that he had elected not to supplement with copper.

The most consistent clinical sign in young deer with osteochondrosis was lameness. Affected animals usually lagged behind the rest of the group and on one occasion a very lame individual could be caught in the paddock after a short chase. Bilateral swelling of joints, particularly the carpal and hock joints, was commonly observed and sometimes led to a clinical diagnosis of polyarthritis by the referring veterinarian. Some animals were unable to fully extend their legs because of the severity of joint lesions.

A characteristic feature, which was observed on three of the eight farms, was an abnormal "bunny-hopping" gait where both hind legs were advanced together. Several animals also had a pronounced "cow-hocked" stance with hocks touching and hind feet widely spaced. This persisted in deer that had recovered from the lameness. Both of these clinical features appeared to be related to the presence of hip lesions in affected deer.

In most cases affected deer and others in the group were in poor physical condition. The only exceptions were the red deer/wapiti hybrids, which the attending veterinarian described as being well-grown.

Pathology

Some of the postmortem examinations on affected deer were performed by veterinary practitioners and although specimens were submitted to a Ministry of Agriculture and Fisheries (MAF) Animal Health Laboratory for further examination, full details of the nature and distribution of lesions were not available in each case. Several consistent findings did however emerge.

Typically, there was multiple joint involvement with the most severe lesions usually in the carpal, tarsal, stifle and hip joints. Milder lesions were often present in the shoulder, elbow and fetlock joints.

Affected joints were usually swollen and contained excess synovial fluid, which was sometimes blood-stained. The earliest gross lesions were found in calves of around 3-4 months of age and consisted of sharply defined, irregular-shaped defects in the articular cartilage on weight-bearing surfaces. Flaps of under-run cartilage were sometimes attached to one edge of these defects but more often, there was complete separation of the cartilage flap creating deep ulcers in the articular surface. In such cases loose fragments of cartilage ("joint mice") were usually found within the joint space. More advanced lesions in older calves and weaners had features of chronic degenerative arthropathy. The capsules of affected joints were thickened and the margins of articular defects were worn smooth. Exposed subchondral bone was often eburnated and sometimes contained grooves aligned in the direction of joint movement. Flakes of cartilage were often present either free within such joints or attached to the synovial membrane.

The lesions were usually bilateral and roughly symmetrical and tended to have predilection sites within some joints. In the stifle, the caudal aspect of the medial femoral condyle was often affected and in the hock there was usually extensive loss of cartilage on the opposing articular surfaces of the tibiotarsal bone and calcaneus.

Deer that had shown a "bunny hopping" gait or "cow-hocked" stance clinically were consistently found to have hip lesions at necropsy. Although separation and loss of cartilage from articular surfaces was sometimes observed, more commonly there was a fracture through the growth plate (epiphyseolysis) of the femoral head with dislocation of the hip joint. Usually, both hip joints were affected. In early cases the detached femoral head was still present within the joint but in one 10-month-old weaner that had been lame for approximately 6 months it was no longer evident. In this animal the growth plate had become markedly thickened and had formed a false joint, articulating with a fibrocartilaginous thickening of the joint capsule dorsal to the acetabulum. The joint space contained cloudy, red/brown fluid and many small solid fragments which presumably were remnants of the femoral head.

Osteoporosis was evident grossly in some of the deer with osteochondrosis, especially those with chronic lesions. The cortices of long bones were thin and there was a reduction in the volume and density of trabecular bone in metaphyseal and epiphyseal regions. This was usually accompanied by mild serous atrophy of medullary adipose tissue. In spite of the presence of osteoporosis none of the affected deer had fractures through the shafts of long bones. The only fractures detected were those involving the growth plate of the femoral head and, in one case, the growth plate of the calcaneus.

Histological changes were typical of osteochondrosis as reported in other species. Horizontal clefts in the deep layers of articular cartilage were the earliest lesions detected. In some cases the clefts communicated with the articular surface forming flaps of underrun cartilage or had detached completely leaving deep ulcers. Advanced lesions included fibrillation and loss of articular cartilage, eburnation of exposed subchondral bone and thickening of joint capsules.

The spinal cord was only examined histologically in three of the deer with osteochondrosis. Wallerian degeneration in dorsolateral and ventromedial white matter tracts, consistent with enzootic ataxia, was present in a 10-month-old deer with joint lesions but not in the other two younger deer where the cord was examined. On two farms adult deer had shown clinical signs of enzootic ataxia.

Chemistry

Serum and/or liver copper concentrations in deer with osteochondrosis from each of the eight farms are presented in Table I. Liver copper estimations were performed on one or two affected deer from each farm and concentrations were well below the level considered to be adequate[15]. In one animal the liver copper concentration was too low to be detected by atomic absorption spectrophotometry. Results of serum copper estimations were available from groups of deer on four farms and mean concentrations were low in each case.

Table I: Copper concentrations in the serum and/or liver of red deer and wapiti/red deer hybrid calves with osteochondrosis on eight farms.

	Farm								
	A	B	C	D	E	F	G	H	Reference Range ***
Mean serum copper concentration * (µmol/l)	2 2 (4)	4 4 (9)	0 8 (8)	-	-	5.1 (8)	-	-	8-22
Liver copper concentration ** (µmol/kg)	27	39 53	41	39	0	44	40 49	19 29	>100

* Number of animals tested shown in brackets.
** When more than one animal tested, each value is presented
*** Based on data of MAF Animal Health Laboratories and Mackintosh *et al.*[15]

Pasture mineral analyses were available from two farms, one of which is presented as a case report in the following paper of these Proceedings. On the other property pasture copper concentrations from three sites varied from 4 to 5 ppm DM, suggesting that the deer on this property had primary copper deficiency. Circumstantial evidence pointed more towards secondary copper deficiency on some of the other properties where osteochondrosis was diagnosed in young deer. Elevated pasture concentrations of molybdenum, sulphur or iron are capable of inducing copper deficiency even when pasture copper concentrations are adequate[16].

Discussion

The occurrence of osteochondrosis in association with low serum and/or liver copper concentrations in young deer on several farms throughout New Zealand suggests that copper deficiency may be an important cause of the disease in this species. The causal role of copper is supported by follow-up information on five of the eight farms. No further cases of osteochondrosis have been recognised on these farms since copper supplementation of pregnant hinds was introduced or copper sulphate was added to annual fertiliser applications. These observations are consistent with the results of recent studies demonstrating that primary or secondary copper deficiency can cause osteochondrosis in foals[10][11][12], and that copper supplementation of both mares and foals reduces its prevalence[13].

The manifestations of osteochondrosis vary between species but articular surfaces of several joints are often involved. According to our observations, osteochondrosis in deer typically affects multiple joints, the most severe and consistent lesions involving the hip, hock, stifle and carpal joints. Milder lesions may also be present in the shoulders, elbows and fetlocks. An interesting feature was the frequent occurrence of bilateral epiphyseolysis of the femoral head causing severe lameness, a "bunny-hopping gait" and "cow-hocked" stance. Epiphyseolysis has also been described as a manifestation of osteochondrosis in pigs[1] but appears to be present in a greater proportion of deer with the disease.

Copper deficiency is well recognised as a cause of skeletal abnormalities in grazing ruminants. Osteoporosis and spontaneous bone fractures have been described in sheep and cattle grazing copper deficient pastures[17][18][19]. In calves, growth plate abnormalities characterised by irregular thickening of the hypertrophic zone, similar to the lesion of rickets, have also been described[20]. Thickening and fissuring of articular cartilage consistent with osteochondrosis has previously been reported only in copper deficient foals[10][11][12], but our observations suggest that this can also be a manifestation of osteochondrosis in red deer and red deer/wapiti hybrids. Interestingly, epiphyseolysis with separation of the greater trochanter of the femur has been described in lambs with molybdenum-induced copper deficiency[21] but articular lesions were not present in these lambs.

It is not clear from the cases reported here whether or not copper deficiency causes osteoporosis as well as osteochondrosis in deer. Some affected deer clearly had osteoporosis at the time of necropsy but this could have been secondary to reduced food intake in animals with severe joint lesions rather than a direct effect of copper deficiency. Spontaneous fractures through the shafts of long bones were not reported in young deer on any of the farms where osteochondrosis was diagnosed.

Based on the history and soil types of the farms involved, together with pasture analyses in some cases, it appears that osteochondrosis in deer can be associated with either primary or secondary copper deficiency. This is consistent with the situation in foals were osteochondrosis has been reproduced by feeding low-copper diets[11] and has occurred both naturally and experimentally in association with dietary zinc excess[10][22][23]. Zinc is an antagonist of copper and, is capable of inducing secondary copper deficiency[19] although in New Zealand, secondary copper deficiency is more likely to be induced in grazing animals by high pasture levels of molybdenum and/or sulphur.

Copper catalyses the activity of lysyl oxidase, an enzyme required for the formation of cross-linkages between collagen molecules[24][25]. In the absence of copper, there is impaired formation of cross-linkages and a reduction in the strength of collagen fibres[19]. Since collagen is a key component of cartilage matrix the cartilage formed during a period of copper deficiency would be expected to show increased fragility and be more likely to suffer traumatic damage under the stress of weight-bearing. An increase in the solubility of collagen in articular cartilage, reflecting reduced cross-linkage, has been demonstrated in copper-deficient foals with osteochondrosis[11].

Assuming a causal relationship between copper deficiency and osteochondrosis in deer, prevention should be aimed at increasing the liver copper stores of newborn calves and the copper concentration of hinds' milk. This could be achieved either by copper supplementation of pregnant hinds or inclusion of copper in fertiliser applications. Treatment of affected calves with copper is recommended but may not result in complete recovery. In cases where there has been extensive loss
of articular cartilage or epiphyseolysis of the femoral head, chronic degenerative arthropathy is inevitable.

Enzootic ataxia has also been associated with copper deficiency in deer[26]. It is not surprising therefore that spinal cord lesions typical of this syndrome were detected histologically in a 10-month-old deer calf with osteochondrosis, or that on two of the eight farms adult deer had shown clinical signs of enzootic ataxia. The syndrome was not seen clinically in young calves on any of the copper deficient farms, thereby supporting previous observations that enzootic ataxia in deer does not occur in animals less than 9 months of age[26].

In conclusion, a syndrome resembling osteochondrosis in other species has been recognised in young red deer and wapiti/red deer hybrids and appears to be caused by copper deficiency. The syndrome has occurred throughout New Zealand and should be suspected when deer calves show signs of lameness and swollen joints, especially when accompanied by an abnormal "bunny-hopping" gait or "cow-hocked" stance. The effect of copper deficiency on the skeleton of young deer appears to differ from that in lambs and calves but more closely resembles that seen in foals. Further studies are required in order to determine whether or not osteoporosis is a feature of copper deficiency in deer and to establish the mechanism of the lesions in articular cartilage.

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