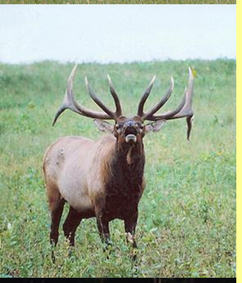

ABOMASAL PARASITE SYNDROME IN NORTH AMERICAN ELK (*CERVUS ELAPHUS*)

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Abstract

521 elk of mixed ages and both sexes were translocated in Maine, USA between 2 elk farms with widely divergent management systems and without regard to physiological, seasonal, social, nutritional and environmental factors known to affect the outcome of such translocations. A two year clinical course of chronic ill-thrift, weight loss and emaciation, and eventual death occurred in the translocated animals, and mortality was approximately 84% with 436 of 521 animals dying, despite supportive nutritional and medical treatment. General clinical and postmortem examinations indicated only selenium and copper imbalance and nutritional inadequacy. Additional purposeful postmortem examination and histological evaluation of tissue sections from 4 of the affected adult elk demonstrated proliferative abomasal lesions as the most significant finding, consistent with type II ostertagiasis, with intralesional nematodes seen in the abomasum of 2 of the 4 elk. Necropsied animals had elevated abomasal pH levels, and copper and selenium deficiency.

Introduction

A condition of chronic ill-thrift with progressive weight loss, and skeletal muscle wasting, has been described mostly in New Zealand, in North American wapiti (elk) as well as in wapiti-type red deer hybrids. Reports of this disease are uncommon but it is referred to as "fading elk syndrome" and has been associated with increased susceptibility of farmed elk-red deer crosses to gastrointestinal parasitism and copper deficiency, along with differences in nutrition between farmed elk in New Zealand and their native North America. Typically no overt cause for disease is recognized in affected animals, although associated clinical features have included hypoalbuminemia, hypocuprosis, elevated abomasal pH and plasma pepsinogen, as well as diarrhea. The primary purpose of this case series is to describe the occurrence of fading elk syndrome in a large North American herd of translocated elk with direct histological evidence of intramural abomasal parasitism by *Ostertagia sp.* larvae. A secondary purpose is to provide an example of the consequences of large scale translocations of cervids without regard to seasonal, social, nutritional and environmental factors that may affect the outcome.

Case History

In May 2005, a herd of 521 elk of mixed ages and sex was transferred over several weeks between 2 elk farms in Maine. Elk began dying within 2 weeks of arrival but veterinary assistance (from a veterinarian in private practice, a diagnostician from an animal health laboratory, and the State Veterinarian) was not sought for 3 months. Only translocated animals became sick. Elk resident on the farm when new animals arrived were in good condition without signs of illness, but within 5 months, 150 of the purchased animals had died. Necropsies on five of these elk were inconclusive, except for gross findings associated with ill thrift (poor body condition and depletion of intra-abdominal fat). Antemortem serum selenium (Se) levels from these elk indicated profound selenium deficiency (1 elk at 1.05µmol/L; others below the 0.32µmol/L detection limit; reference range 1.27-2.28µmol/L). Farm water samples showed a mild elevation in iron (37.6 µmol/L; 5.4µmol/L was set as the upper desirable limit) but were otherwise unremarkable and feed analysis indicated low levels of crude protein (mean 6.3% DM; range 4.6 – 9.0%), total digestible nutrients (mean 58.3%, range 52.8 – 63.08%), calcium (mean .44%, range .26- .69%) and selenium (all samples < 0.30 mg/kg) with high acid detergent fiber (mean 42.5%, range 39.5 – 50.4%). Few sarcocysts were identified in skeletal muscle sections from the animals. At this stage, a diagnosis of selenium deficiency was offered. Veterinary recommendations included parenteral selenium and thiamine treatment, *ad libidum* supplemental feeding of a Se based mineral mix, and addition of oral amprolium crumbles to the diet in the unlikely event that the presence of sarcocysts was clinically significant.

In October 2005, cheek muscle, heart, lung and tongue samples from 2 more animals were sent to the Maine Animal Disease Diagnostic Laboratory for histopathology, and liver was submitted to the Animal Health Diagnostic Center at the College of Veterinary Medicine, Cornell University (AHDC-CU) for trace mineral analysis. Mineral imbalances were found with deficiencies in copper (5 and 6 mg/kg DM; reference range 20-120 mg/kg DM) and molybdenum (<2 mg/kg DM; range 2-4 mg/kg DM) and mildly elevated zinc levels (95 and 136 mg/kg; reference range 23- 80 mg/kg DM). Liver selenium levels were low (0.42 and 0.25 mg/kg) but within normal values (0.25 – 1.4 mg/kg DM). Thus copper deficiency was added to the list of diagnoses made in the herd. Whole blood, blood smears and serum from 9 live elk were also submitted for a CBC, routine ruminant serum chemistry testing and serum Se analysis. All values were within reference ranges. Feces were submitted along with tissues and blood but fecal floatations failed to reveal significant intestinal parasitism.

Clinical Findings

Animals continued to die throughout 2006 despite nutritional supplementation with a commercial cervid diet formulated with trace minerals, coccidiostats, parenteral treatment with selenium/vitamin E, and administration of anthelmintics. At this stage, there was no shortage of opinion, professional or otherwise, about the causes of the losses numbering into the hundreds of animals of both sexes and all ages, and appropriate remedial measures.

Various diet supplements listed above, designed to supplement protein, selenium, copper and other trace minerals were provided to the elk one after another. In November 2006, whole blood, blood smears and serum from two yearling male elk were submitted for a CBC, routine ruminant serum chemistry testing, serum lead (Pb) analysis and an AGID assay for Johne's disease. All values were within reference ranges and the AGID's were negative. Interestingly, beyond the initial postmortem examination (PME) in August 2005, necropsy records contain no reference to testing for CWD.

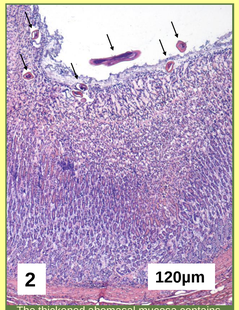
In January 2007, an opinion was sought from one author (MRW) who suggested a renewal of diagnostic efforts. In January and February 2007, 4 elk with signs of chronic wasting consistent with those of other affected animals in the herd were euthanized and their carcasses submitted for PME to another author (NMAP).

Additionally protease-resistant prion protein (PrPres) was not detected by immunohistochemistry on formalin-fixed obex, spinal cord and retropharyngeal lymph nodes (NVSL; Ames, IA). A diagnosis of prion related CWD was thus precluded.

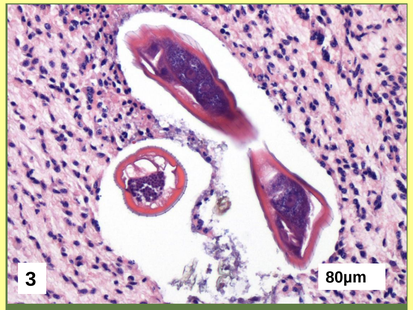
In-house parasitological analysis by centrifugal fecal floatation was performed on samples of fecal material from animal 251 and revealed moderate numbers (5-10 per slide) of trichostrongyle eggs, as well as very small numbers (1-5 per slide) of operculated eggs, suggestive of trematode infection. Analyses of fecal samples from the remaining 3 animals produced variable results. In feces from animal 143 approximately 1,200 trichostrongyle eggs were seen per gram of feces and approximately 900 trichostrongyle eggs were seen per gram of abomasal contents. In animal 157 approximately 10,650 trichostrongyle eggs were seen per gram of feces and; approximately 2,100 trichostrongyle eggs were seen per gram of abomasal contents. Paradoxically, in samples forwarded to the AHDC-CU, no parasites were detected in either of these animals. In-house parasitology was not performed on samples from animal 213, but fecal material collected from this animal was forwarded to the AHDC-CU for quantitative analysis and revealed 30 *Eimeria sp.* oocysts per gram; 9 *Nematodirus spp.* eggs per gram; 89 *Trichuris spp.* eggs per gram; 1 Capillarid-type egg per gram.

Hepatic trace element analyses (Table 1) revealed that all animals were severely copper and selenium deficient, with marginal zinc levels. Additionally, in animal 157 iron levels were in the low reference range but were elevated in animal 213, although the significance of this is unknown.

Abomasum (animal 251): Mucosal thickening & "cobblestone" appearance



The thickened abomasal mucosa contains nematodes (arrows)



Nematodes embedded within the mucosa

All were intact females (animal numbers 143, 157, 213 and 251) estimated at 3-4 years of age. At gross PME they were emaciated with prominent vertebral column dorsal spinous processes and depleted adipose tissue (subcutis, epicardium, perirenal region and bone marrow). The most consistent gross findings were in the abomasum which was thickened and hyperplastic with nodule formation, producing a cobblestone appearance (Fig. 1). Samples of abomasal fluid were collected from all animals at PME for rapid and crude analysis of pH using urine dipsticks. pH levels were elevated at approximately pH5 (reference range 1.5-4.3). No significant lesions were found in other tissues, and molar dentition was normal upon sagittal sectioning of the skull.

Tissue samples were collected for histopathological evaluation, fixed in 10% neutral buffered formalin and routinely embedded in paraffin. Five-micron sections were prepared from tissue blocks and stained with hematoxylin and eosin (HE) for microscopic examination.

Histologically the most significant finding in all animals was abomasal mucosal epithelial proliferation with mucosal gland hyperplasia (Fig. 2; Table 1). Nematodes were also seen (Figs. 2, 3; Table 1), along with eosinophilic cellular infiltrates. Microscopic features of the nematode, including external cuticular longitudinal ridges, were most consistent with a trichostrongyle species such as *Ostertagia* spp. Scattered sarcocysts were incidentally present in the heart and tongue.

There was no histological evidence of transmissible spongiform encephalopathy in the brain or brainstem.

Table 1

Elk #	Abomasal pH (ref. range: 1.5-4.3)	Histopathologic features			Hepatic trace element levels (mg/kg DM)*			
		Mucosal thickening and mucus gland hyperplasia	Nematode presence in the mucosa (+/-)†	Eosinophils (+/-)‡	Copper (ref. range: 80-600)	Zinc (ref. range: 92-400)	Selenium (ref. range 1-7)	Iron (ref. range: 400-1000)
143	5	Moderate	+	+	11	57.5	0.07	661.5
157	5	Moderate	-	+	9	75.6	0.17	405.2
213	5	Mild	+	+	7.7	530.3	0.83	1831.5
251	5	Marked	-	-	9	76.7	0.44	1003.5

* mg/kg DM = milligrams per kilogram dry matter
† nematodes present (+) or absent (-)
‡ eosinophils present (+) or absent (-)

Conclusions

The low number of postmortem examinations carried out in the face of such large losses is perplexing. Additionally the veterinary advice offered, albeit based on evidence from insufficient animals, implicated relatively common ruminant nutritional and trace mineral problems in that geographical area and placed too much emphasis on the significance of sarcocysts which, unless found in very large numbers are considered an incidental finding in elk.

The most significant finding in the necropsied elk from early 2007 was the proliferative abomasal change. This, along with the history and clinical signs, is consistent with well documented lesions and physiological after-effects of type II ostertagiasis in ruminants. As no evidence of significant underlying systemic disease was found to otherwise explain the chronic wasting in these animals, the possibility of original parasitism inciting the clinical problems in these animals was therefore considered. *Ostertagia* spp. are arguably the most important gastrointestinal nematodes of cattle and other ruminants. They localize and affect the abomasum, and represent a problem that appears undiminished with increasing host age and immune competence. Type II disease occurs in the autumn and winter, especially in females calving for the first or second time, and coincides with the stress of calving and emergence of thousands of hypobiotic fourth stage (L4) larvae from the abomasum. Severe scouring, loss of weight and even death may result.

The elevated abomasal pH and pronounced trace mineral imbalances in these animals are consistent with the secondary effects that such *Ostertagia*-induced mucosal damage initiates. Ostertagiasis has been reported to cause elevated abomasal pH in farmed red deer and cattle, negatively affecting the animal's digestive efficiency, by reducing abomasal pepsin activity and restricting bioavailability and uptake of dietary copper levels. The elevated pH may also interfere with oral anthelmintic uptake, further adding to the problem.

Findings in the 4 animals submitted for PME were alike, and their clinical histories are similar to those of other herd members that also died. Since the whole herd was subjected to the same conditions, treatments and diet, these postmortem findings are considered likely representative of what occurred across the herd. Overall, the abomasal lesions, elevated abomasal pH levels and trace mineral abnormalities are consistent with those described in a condition of chronic ill-thrift and often extreme cachexia that has been documented in elk, red deer, and their hybrids and associated with *Ostertagia*-like nematodes. As in other ruminants, this syndrome in cervids leads to increased abomasal pH with associated protein losing enteropathy and copper deficiency. Because of its progressive nature and poor response to therapy, this poorly understood condition has been termed "fading elk syndrome". Care should be taken, however, to differentiate it from chronic wasting disease of cervids and it is suggested that it be renamed "abomasal parasite syndrome of elk and red deer."

The authors are not suggesting that the 436 deaths in this translocated herd were all due to abomasal parasite syndrome. Indeed, the ultimate demise of the herd was certainly multifactorial and the inherently stressful movement of the elk to a new environment, subsequent mixing of age and established social groups, and a sudden change of feed type and quality would be sufficient to permit expression of any latent diseases in the herd; however, the few animals given postmortem examinations early in the course of morbidity yielded only nonspecific information and did not show overt signs of infectious disease such as pneumonia or enteritis. Trace mineral imbalance suggestive of dietary deficiency or abomasal parasitism appeared to be the most consistent finding and affected the animals necropsied in 2005 and 2007. It is possible that this herd was suffering from Type II Ostertagiasis at the time of translocation and that the nutritional, psychological and social stress placed on them after the move exacerbated the problem. It is also possible, however, that the stressed and immunologically depressed elk acquired the parasite after being translocated. The timing of the infection is perhaps not as important as the ultimate consequences of the establishment of a Type II cycle in this elk herd. Certainly, however, the ultimate results of the infection are consistent with other descriptions of the clinical course of this debilitating condition, except on a much larger scale.

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