FADING ELK SYNDROME: CASE STUDY

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General Background and History

Nelson has one of those ideal climates for both man and beast alike. However, we still manage our share of animal health problems.

We have in our practice area 3 main Wapiti/Elk breeders and this case report involves what could be termed 'outbreak conditions' of Fading Elk Syndrome (FES). Predominantly one of these breeders was involved. The other breeders had problems too, but being of smaller herd size, more time was inevitably spent with the one farmer who had the larger herd.

This farmer is a well known Wapiti breeder who farms in the coastal area between Nelson and Motueka. He has 48ha of very easy and productive land. The farm was once a dairy farm and the pasture content is mainly red and white clover and perennial ryegrasses. On this area he farms 320 deer only; made up of equal numbers of adults and weaners. Of the adults, 8 are Wapiti bulls and 80 are Wapiti cows. Up until 7 years ago the Wapiti herd were all of South Westland origin. Since then he has gradually bought in purebred stock, mainly from Canada. The rest of the herd are Red/hybrid cross hinds. To date the problem has only affected the Wapiti/Elk animals, and in particular a line of animals that originated from a property near Christchurch. The owner felt that the problem had only been apparent since these Canterbury animals had been bought in 2 years ago. He had noticed that it was only the purebred animals that were affected. He mentioned that he had been farming deer on this property for 12 years and that he had only had 4 deaths in that time from causes other than old age and misadventure.

In years prior to 1989 we had investigated the occasional thin Wapiti, and carried out basic lab work. The lab results indicated low serum protein, low albumin, low serum copper and anaemia. Initially, we concluded that we were dealing with a copper deficiency. However, on treatment with copper, no response was noted. This was at a time when we were finding a reasonable number of properties with copper deficiency and when the copper deficient syndrome was not completely understood. We were also only dealing with the odd animal in a herd, and the time and effort was not spent on these individuals as perhaps it should have been. In retrospect we now conclude that we were dealing primarily with Fading Elk Syndrome, and that the copper deficiency was only an incidental finding or secondary to the Fading Elk Syndrome. With this in mind, further cases were treated with B vitamins, Terramycin, Ivomec drench and copper orally, and the affected animal was confined to a lucerne hay diet. The rest of the herd were treated with copper needles and copper sulphate was administered to the drinking water. We then hoped that the problem would not involve any number of animals, as the owners were aware that we could not offer much more in terms of treatment, or prevention.

The 1989 Situation

A similar situation arose this year. The farmer himself recognised the signs and he began treatment as in previous years. However, this year the situation was of a much greater scale. By the time I became involved, there had been 11 deaths and a further 12 were looking very sick. The owner was looking very stressed too. A further 12

became affected during the first week of my involvement.

Although the winter was hard for Nelson, at no time were the paddocks devoid of grass. 2500 bales of lucerne or Red clover hay were fed out, boosted by concentrates such as barley. This farmer is generally known for his good stock and it would be fair to say that a thin animal is a rare sight.

The Clinical Picture

The initial clinical sign was excessive salivation and ventral jaw oedema. Some animals showed laboured respiration with 'belching' and some animals were scouring. There was an immediate and rapid loss of body weight. From the onset of clinical signs, the duration till death was 3 weeks to 3 months. Affected animals continued to eat.

Of the clinical pathology results, the most predominant and consistent blood changes were low plasma protein, low albumin, and an elevated pepsinogen level. More often than not, other changes included anaemia, an elevated GLDH and GGT. In contrast to clinical pathology reports recorded elsewhere, eosinophil levels were relatively normal.

Two animals in an advanced disease state, being barely able to stand, were euthanased and postmortemed. The gross pathology revealed little apart from obvious body wasting. The rumenal contents weighed about 6kg. One animal was a 10 month old bull and the other a mixed age hind.

The most notable histopathology involved the small intestine and the abomasum. There was a moderate, chronic inflammatory reaction with numerous parasite sections to be seen. The liver showed collapsed sinuses with a diffuse scattering of inflammatory cells. Brain and heart showed no specific pathology and the kidney showed some oedema. From this a diagnosis of parasitic enteritis was made. At the same time dung samples were collected from two other animals and both showed moderate to high strongylate egg counts. This was surprising after the regular oral Ivomec treatment that all animals had received. The two animals postmortemed had been drenched 10 days previously. Up until this period of our involvement, oral cattle "Ivomec" was used almost exclusively on the property at a dose rate of 20ml to 120kg animal i.e. at 3x the cattle dose rate. At one stage the drench was changed to "Synanthic" and "Chelmin" minerals. The pastures were generally well spelled, and there was no history of liver fluke or facial eczema on this property, although I do know of close neighbours that have had facial eczema problems in sheep. Possibly the browsing feeding characteristic of deer is a reason why in Nelson, we had had no reports of facial eczema. All animals are drenched with selenium and cobalt drench, on a regular basis, and copper needles are administered.

Treatment Programme:

It seemed apparent that the worm problem was significant in being responsible for the onset of this Fading Elk Syndrome (FES) situation. With this in mind my immediate concern was to stop further cases occurring by gaining control over the parasite problem. It appeared that oral anthelmintics were not effective, possible due to the damaged mucosa affecting drug absorbtion. I did not consider there to be a resistance problem against Ivomec, and so opted for continued use of "Ivomec" but in the form of "Ivomec Pour-on". We favoured the use of the 'pour-on' over the injectable form because of it's ease of administration when dealing with 88 adult Elk animals. Because the farmer was a little sceptical about the results, he only used the "Ivomec Pour-on" on his adults and continued to use his already purchased large supply of "Ivomec" cattle

oral on his weaners. New cases stopped developing abruptly in the adults, but they continued to appear in the weaners. Subsequently, the weaners were treated with "Ivomec Pour-on" and the problem stopped here too. A double dose rate based on the cattle dose was used. A single dose rate may well have been as successful, but this level was not tried.

It was a considerable relief to see a halt to the development of new cases. However, the problem now was how best to treat cases that had already developed clinical signs. The biochemistry and autopsy results pointed to a protein losing gastro-enteropathy. The resulting condition loss seemed very hard to stop, despite the use of "Ivomec Pour-on", and animals usually continued to deteriorate at varying rates.

The initial treatment attempts involved the confinement of animals to a sole roughage diet (lucerne and clover hay) and the individuals were treated with "Ivomec Pour-on", B-vitamins, anabolic steroids, "Terramycin" and sometimes anti-scour preparations. We tried intravenous amino-acid solutions, such as 'Aminoplus', but the large volumes required if we were to anticipate any significant effect, coupled with the practical deficiencies of these procedures, made the whole exercise rather discouraging. We considered, in discussion with Dr Colin Mackintosh of Invermay, the administration of blood. Although the effect of these procedures may only be short lived, the theory was that the effect may replace the protein deficiency long enough to perhaps initiate the animal into a 'self recovery' phase. However, after our previous IV infusion experiences, we did not proceed. These animals are not easy to handle and the stress of continual treatment attempts were perhaps making the situation worse. The isolation from the rest of the herd onto a sole roughage diet seemed in itself an added stress to the affected animals.

Affected animals were treated by the various injections and infusions, as mentioned, over weeks 2 and 3. It became obvious that we were making no progress, so a completely different approach seemed warranted. Certainly it appeared we would have little more to loss by trying!

I had heard that a similar condition in America had been treated with some success with products that contained a mixture of natural micro-organisms. We located such a product called "ALL_LAC" (soluble source of micro-organisms; Lactobacillus acidophilus and Streptococcus feacium), and followed the recommended dose rate for cattle at 40mls orally every 2nd day for 3 doses. We stopped all other treatment apart from a dose of "Ivomec Pour-on". This combination of "All-lac" and "Ivomec" was given to all of the Elk, although I think that it was probably only a requirement to give the "All-lac" to the affected animals.

A new block of land where the vegetation was more un-improved (Manuka, pine trees, bull rushes and native grasses) was fenced on a neighbour's property and all of the Wapiti/Elk herd (including the 12 affected animals) were moved to this area.

<u>Six Months Later</u>:

A parasite involvement in the Fading Elk Syndrome was recognised towards the end of the first week of my involvement. "Ivomec Pour-on" treatment was promptly carried out on the adults, and about one week later on the weaners; from this treatment no further new cases developed. Of the remaining 12 affected animals put out on the 'rough vegetation block' with the rest of the herd, 3 wasted away and being in an advanced, debilitated state, were shot by the owner. The remaining 9 survived and regained a good body weight, although none calved.

Discussion:

It is possible that this disease problem may have involved more animals in previous years than we knew about. Having animals that look like little more than walking skeletons, particularly when there is little that can be done for them, is not something that, as breeders, you would want publicised. Also, the Wapiti/Elk breeders have been working to establish their breed type firmly in the deer industry and this condition is not one that would further their cause. As a result, it may be that these cases are not brought to our attention as often as could be.

The economic loss of F.E.S. in this herd alone was very considerable, and when considering the breed type on a national scale it could be of a proportion more significant than other disease entities. The economic effect has prompted this farmer to not use his pure-bred stags this Autumn in favour of using hybrid stags to produce a breed type less affected by F.E.S.

As a result of last year's experience, I consider that in our situation the initial parasite problem was of major, underlying importance. However, both myself and two of the farmers involved suspect it to not necessarily be the primary cause, but perhaps acting more like a precipitating factor. I have nothing quantitative to support this, part from unanswered questions as to why, for example, liver dysfunction is evident, and why response to "Ivomec Pour-on" was not evident in clinical cases.

In addition to this, I consider that there are two other main contributing elements to the disease picture:

1. Some unknown pasture factor that possibly precedes any other factor, such as parasites.

2. A genetic element. None of the Red Deer on the property were affected, and the worse affected were the more purebred Wapiti/Elk animals.

The effect of trace elements may be important, but I consider any diagnosed deficiencies identified at the time of disease were only secondary to the symptoms already evident; such as liver damage and diarrhoea.

Currently, some formulation for the treatment of affected animals still eludes me, and will probably do so until the causes and pathology are better understood. From a retrospective viewpoint, I consider that the successful treatment will lie in the delicate restoration of normal bowel function, rather than the 'shot-gun' type of treatment (anti-biotics, Anabolics and steroids etc.,) most used.

A sense of proportion has to be maintained on apparent successful drug usage. For example, although it may appear that some success could be attributed to the use of "All-lac", any importance placed on its use must still be questionable. Three of the twelve remaining clinical cases still died, and the remaining nine may well have recovered anyway. Some more quantitative investigative work would have to be made to justify it's possible recommendation as a treatment for Fading Elk Syndrome.

With the current void existing in the understanding of this disease, my efforts are currently directed at prevention. This involves the introduction of roughage feeding in early autumn and the regular (5 weekly) usage of "Ivomec Pour-on" as an anthelmintic at an elevated dose rate. The Wapiti/Elk herd are rotated to the cleanest pasture, with the Red Deer following.