



“BONSAI” ELK

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Background

A 58 ha deer farm at Tirau, South Waikato, carrying 240 Wapiti-cross cows, 150 Wapiti velvet bulls and 180 mixed sex weaners, experienced premature births in a mob of 2-year-old, 1st calving elk-cross cows

The property was intensively farmed with high quality rye/clover pastures (Olsen phosphate levels > 40), paddock sizes varied between 1-2 ha, and there were no natural water supplies or weeds of any known significance

Approximately 15 animals out of a mob of 70 were affected. These cows carried between 40%-70% elk genes and weighed 140-170 kg. Pre-calving body condition was excellent and they were rotationally grazed on pastures of 2,600 kg Dry Matter (DM)/ha, leaving residual grazing levels of 1300 kg DM/ha approximately

On 29 October the animals were Tb tested and treated with Vetdectin (Ancare) pour-on at 1 ml/10 kg liveweight (500µg/kg Moxidectin)

The Problem

Calving commenced on November 15 with several normal live births until November 21 when 2 dead calves were found. These premature calves were a little over half the expected size, weighing between 4-5 kg. It was initially assumed that these were twins that a 1st calving cow failed to carry to full term.

On November 24 three more premature calves were found and these were immediately taken to Ruakura Animal Health Laboratory for intensive postmortem investigations.

Premature births continued on an irregular basis for approximately two weeks with normal calves also being born during the same period. Several deaths occurred outside the two-week period which I considered were indirectly connected, but not part of the initial problem, and two stunted survivors struggled through to weaning (March).

Clinical and postmortem findings

Out of the 8-9 premature calves weighed, 8 were between 4668 gms and 5537 gms. One weighed 8337 gms and it is possible that this was a concurrent, unrelated perinatal mortality. Expected birth weights from these young cows would be 8-12 kg.

Most calves had inflated or partially inflated lungs, and many had extensive bruising over the lumbar and cranial regions with occasional fractured ribs. Excess straw-coloured fluid was consistently found in the thoracic cavity.

Extensive searches for leptospira using dark ground microscopy were negative, and culture of stomach contents grew only mixed coliforms, mainly *E. coli*.

Extensive histology involving brain, kidney, lung, liver, heart, spleen and skeletal muscle revealed in general no abnormalities, but in one case there was mild locally extensive non-suppurative inflammation between myofibre bundles of the heart. Infiltrating cells were mainly monocytes with occasional eosinophils.

Liver analysis by Wallaceville for Moxidectin was negative.

The size of adrenal glands weighed were 596 and 573 gms. These were little different from full-term cohorts (606 gms) that died of dystocia.

General observations

Close observation of the affected mob revealed the peri-parturient events. Premature calves were born rapidly with the dam exhibiting normal calving behaviour. The premature calf would shake its head and demonstrate the expected responses to maternal licking. The mother would stay recumbent beside the young calf and if body positions were suitable she would encourage it to suckle while still on the ground. Those that suckled in this position and that were strong enough would then stand and be encouraged to follow the mother. Other dams would stand shortly after birth, lick the calf and encourage it to stand and feed. Calves that were too weak to stand were persuaded by the dam to do so by tapping with the forelegs. After a period this slightly unusual behaviour would attract other cows whose pedal stimulation was definitely less subtle, often resulting in death, and explaining the bruising observed at necropsy.

Of the calves that had managed to stand, most died within 2-3 days. They were not tall enough to suckle once the dam was standing and death by starvation resulted. Sometimes they were kicked to death while attempting to feed from the wrong cow.

Another more complicated scenario emerged as the problem continued. Dominant cows whose calves died or were too weak to feed then stole a calf from a more subservient herd mate, often a full-term calf (cross-mothering or multiple suckling seems more frequent in Wapiti types). Unfortunately the "new" mother had a poorly developed udder and very low milk supply, so this calf would have a slow, protracted death by starvation over 1-2 weeks.

Finally, two calves had either long legs, or short-legged dams and they managed a stunted existence until weaning, hence the use of the term "*Bonsai Elk*".

To me the calves looked like the result of "induced parturition" as practised with cattle. No evidence of disease was detected bacteriologically or histologically in the calves, and they definitely had a will.

to live. It is my experience that the rate of abortion or premature births in New Zealand deer herds is low (< 2%), and although reproductive rates are considerably lower in yearlings than older hinds, this is a result of lower conception rates and higher perinatal mortality, rather than high rates of foetal loss in late gestational. The causative agent seemed to remove an entire segment of this season's calves, all of approximately the same gestation age.

Early pregnancies reached full term normally and so did late ones. This seems more like the pattern expected of a teratogen rather than the more random picture expected of an infective agent, especially as the calves and dams appeared healthy.

Differential diagnoses

- 1 Bacteriological causes, eg leptospirosis
- 2 Viral agents which?
- 3 Fungal or protozoal causes
- 4 Plant based teratogens
- 5 Chemical teratogens
- 6 Unknown abortifacient agent

This is an area of deer medicine where little conclusive evidence exists and, as is often my experience in both bovine and cervine medicine, another abortion investigation ended without a positive diagnosis.

Discussion

I would like to collect together and outline some of the evidence I believe to be important in this case. Many of the comments are definitely open to debate and perhaps beyond our current level of veterinary medical knowledge (certainly beyond the author's).

Was Moxidectin involved?

- For*
- * only one mob out of 4 on property was treated with Moxidectin and this group had the problem
 - * A second, totally independent property, had 5 rising 2-year-old elk-X cows treated with moxidectin pour-on at the same gestation stage. Two out of 5 aborted premature calves 3-4 weeks post treatment. Only three of the 5 were pregnant. Red 2-year hinds treated at the same time had no problems.
 - * An unexplained late term abortion storm in cattle, in which only the moxidectin injection-treated mob were affected, also demonstrated similar unusual fibrosis and degenerative changes in cardiac muscle especially around the papillary muscle.

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- * Research scientists suggest that if moxidectin acts by binding its GABA receptors, then prolonged high blood levels may result in some suppression of pituitary-ovarian support because GABA does inhibit hypothalamic secretion of LHRH at high doses.

Could this trigger the birth process???

- * The highest concentration of GABA receptors is actually in the female reproductive tract, not the CNS. The function of these receptors is poorly understood

[points marked by * provided in personal communication with Pfizer Research Division, Australia]

Could the pour-on formulation in young elk with very good fat reserves provide basal levels of moxidectin over a prolonged period sufficient to interfere with pregnancy support mechanisms?

- * Elk are extremely sensitive to the effects of endotoxin found in ryegrass. Some other tremorgens have an effect mediated via GABA receptors. Could there be an additive effect? Is this why elk are affected and not red deer?

- * Incidences of abortion or unexplained low lambing percentages were reported by vets dealing with moxidectin-related deaths in sheep

- * Why should calves born half normal size have full-sized adrenals and reasonably advanced lung development? Does this indicate something stimulated adrenal development, triggering the normal birth process?

- * Moxidectin seems clinically more effective at controlling intestinal parasites in elk. Is this linked to enhanced biological activity in other areas? Are safety levels as large in elk (or goats?) as is currently believed? Elk are not just big red deer

NB: *We did not expect to recover moxidectin from the aborted calves as the time from treatment to testing allowed adequate intervals for levels to subside*

Against

- * It seems that only first calving hinds are affected. Epidemiologically, this group would be the most susceptible to any infective agent

- * No-one else has reported cases of this nature. Presumably similar type animals have been treated with this product at the same stage of gestation

- * My investigation was incomplete, I can't eliminate all other possibilities

- * Most abortion investigations end with no diagnosis - why should this be any different?

- * Perhaps because I owned the deer my approach has been less than objective

I have put this case to print in the hope that better minds will help solve the riddle Increased observation gives the only hope of resolution

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