Facial Eczema in Red Deer & New Zealand Wapiti

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Background

Considerable scientific debate continues over the reality and/or magnitude of "Global Warming" but it is certainly fact that some of the summers experienced over the past 3 years (1999-2001) in the upper North Island have been the warmest on record. These periods of high temperature and high humidity have occurred during both El Nino and La Nina weather patterns and has created environmental conditions ideal for the growth of the facial eczema (FE) fungus Pithomyces chartarum.

The spores of this fungus contain a toxic metabolite known as sporidesmin, a very powerful hepatotoxin, the effects of which are known in deer and well reported in both cattle and sheep. The economic impact of FE rates the disease as one of the most significant production limiting obstacles faced by farmers in susceptible areas and FE prevention is now one of the largest costs in the animal health budget of North Island farmers.

Despite the depth of knowledge on FE in other species, virtually no new information has been produced on its effects and prevention in Deer since the excellent initial investigations by P.H. Mortimer at Ruakura in 1980/81.

Introduction

Prior to the autumn of 1999 I had observed only a limited number of clinical FE cases in red deer; perhaps some 20 cases, all in weaners, with several small outbreaks involving 4 – 6 animals. Observed clinical signs involved:

- Poor growth or weight loss
- Photosensitivity, leading to skin swelling around the eye areas
- Skin ulceration of the muzzle and ocular margins & tongue tip
- Occasional sudden blindness

Because of the low incidence of disease and the high cost/difficulty of most preventative measures little was attempted to limit future disease.

During autumn of 1999, spore counts in the Waikato increased to levels not usually seen. Counts of 3 – 400,000 were common and reports of individual counts exceeding 750,000 spores occurred. (>40,000 are considered to be potential danger). Not only were counts very high but elevated levels continued over several months resulting in high cumulative total the intakes of sporidesmin toxin. This pattern repeated in the 2000 & 2001 seasons.

Clinical Observations

During these years I observed a number of clinical cases in deer which I could not relate to my pre-existing knowledge of FE.

Deaths of breeding bulls (wapiti) following 4 – 5 days' depression, elevated temperatures and some mild shade seeking. My initial diagnosis of FE was confounded by Lab reports indicating mild liver damage only.

Widespread chronic scouring in Wapiti weaners. This was non-responsive to all treatments tried and samples tested negative for parasites, yersinia, salmonella and anything else I could think of. Gamma gutamyl transferase (GGT) levels demonstrated only modest increases (200 – 400 i.u./l.) in 30 – 40% of the cases tested. None of the weaners demonstrated any clear evidence of photosensitization. However, some had small patches of erythema and ulceration deep inside the nose just beyond where the black pigmented mucosa became pink again. (reflected light ???). Weaners dying at this stage were typical of a protein losing enteropathy and not the acute liver damage that I would have expected.

Some weaners that survived the chronic enteritis & weight loss then became blind in early winter and died.

In 2000 one pure elk bull demonstrated classical symptoms of photosensitization with constant nose licking, tongue ulceration, rubbing of the eyes on the flank, yellow pigmentation of the aqueous humour and total blindness. Despite being locked inside away from sunlight for ten weeks this animal was still photosensitive when released outside midwinter and subsequently died.

The 2001 season had extremely hot humid weather in late January/ February when breeding bulls were still feeding actively to gain weight. Spore counts became extremely high much earlier than usual.

a) A pure Elk bull in excellent pre-rut condition, showing no signs of illness, was collected for semen storage. At the end of an excellent 200 straw ejaculate some blood was found in the post-ejaculate urine. Six days later this bull was dead and post mortem revealed a ruptured duodenal ulcer with associated peritonitis. Liver changes were also noted:

- neutrophils in the sinusoid and perivenular zones
- bile pigment in the cancliculi and Kupffer cells
- moderately dissociated and rounded up hepatocytes
- cytosegrosomes apparent (not seen in cattle)
- lobular pattern disrupted
- moderate hyperplasia of cholangioles

The lab would not categorically state that the hepatopathy was due to sporidesmin!!.

Two other Elk X bulls were then seen urinating blood. One showed clear evidence of difficulty and pain on urination – lordosis of the back and very frequent attempts to urinate. The other had haematuria, but also had an enteritis and diarrhoea with malaenas. This animal also demonstrated clear evidence of photosensitivity – swollen ears and eyes and constant tail flicking related to photosensitivity of the perineum. This animal died approx. six weeks after initial signs appeared. The bull presenting with predominantly urinary symptoms survived despite major weight loss. The following spring he produced a distorted velvet head reduced from 10 kg to 7 kg but by autumn he had fully recovered body weight indicating little long-term liver damage.

Conclusions

I now believe that sporidesmin toxicity in deer presents with a much larger range of symptoms than we usually expect from our experience in sheep and cattle. Photosensitivity is not always the major presenting symptom and acute liver damage may or may not be present. GGT levels increase only modestly and this leads us to believe the animals are mildly affected only.

I believe that the sporidesmin toxin has a direct cytotoxic affect on the mucosal cells of both the proximal intestine and the urinary system, where the toxin may be concentrated prior to excretion.

This direct cytotoxic effect explains the chronic diarrhoea seen in Wapiti weaners and the duodenal ulceration/malaena observed in Wapiti bulls. Damage to the urinary system includes submucosal haemorrhage and ulcerative cystitis with direct haemorrhage into the urine. Ureters can also become oedematous and possibly occluded resulting in hydronephrosis and sudden death. This is particularly relevant to rutting bulls as they are in a permanent state of diuresis.

Deer have an extremely effective hair coat to protect them from photosensitivity damage. I believe the pink mucosa inside the nose which becomes affected by reflected light is one of the few places to detect mild cases, and it is often missed on clinical examination. Animals presenting with acute onset blindness or progressing from other symptoms to blindness have a very poor prognosis and they probably indicate more severe liver involvement.
The chronic weight loss/ill thrift syndrome may also be linked to liver fibrosis but the differential diagnostic possibilities are far larger.

Another clinical syndrome possibly connected to FE is acute laminitis in weaner deer. This usually affects the front feet leading to permanent malformation of hoof growth patterns ("snow shoe" toes). 1–2 % of weaners have been detected with this problem during high challenge FE seasons (Toxicity induced).

Susceptibility

The clinical observations I have made fit very closely with the observations of Mortimer and Smith during their experimental dosing of Red and Fallow deer. Their trial work involved red spikers and this class of stock is probably one of the least affected groups during natural outbreaks (c.f. weaners).

I believe that elk and their hybrid crosses with red deer are much more sensitive to the effects of sporidesmin. The higher the percentage of elk-genes, the more sensitive they become to the extent that I have found it very difficult to keep pure elk weaners, especially females, alive through Autumn in severe FE seasons. Overall elk have a susceptibility pattern more similar to fallow deer with red and lower hybrids being slightly more resistant.

Within species, age groups vary in their susceptibility and this is greatly influenced by farm management patterns:

With early March weaning, young 5 month calves are forced to greatly increase pasture intake following the loss of milk as a dietary component. This procedure makes this group the most at risk.

Post-rut weaning reduces grass intake for weaners but increases the risk faced by lactating hinds.

Mature stags rapidly increasing weight prior to the rut are at high risk should spore counts increase early in the season, i.e. late Jan/Feb. They can also be subjected to further high challenge should spore counts remain high in the late April/early May period as they attempt to recover body condition post rut. Reduced feed intake during the rut may protect them to some extent at this time.

18 month growing replacement stock are intermediate in susceptibility similar to 20 month dairy replacements.

Dry hinds are moderately resistant but sporidesmin effects are cumulative so some hinds may end up with insufficient liver function to regain weight over winter or lactate effectively in future seasons.

Prevention

With death rates reaching 10% in Elk X weaners, the loss of expensive breeding bulls, and poor growth rates in sub-clinically affected weaners, whole herd preventative measures have become essential. Initial attempts at partial diet substitution during at risk periods proved insufficient to limit damage.

In the 2000 & 2001 season Wapiti weaners were treated with "Time Capsules" (sheep) (Agri-feeds) – both at weaning and six weeks later. These proved difficult to administer requiring hydraulic restraint of all animals and a high percentage (5 – 8%) appeared capable of regurgitating the capsule despite oesophageal engagement prior to delivery. Cases of chronic scouring and weight loss in the weaner group still occurred at levels beyond that acceptable for a satisfactory cure.

For the 2002 season I have adopted a multifaceted control approach involving:

1. Widespread diet substitution
   - Summer turnip crops
   - Maize silage
   - Alternative pastures – chicory, plantain & clover
2. Post or late rut weaning.

   Calves left on cows until spore counts dropped to save levels (<50,000 spores)
3. Whole farm fungicide spraying

All pastures were boom sprayed with X-SPORE/Bell Booth (carbendazim) 150gms/ha and repeated as per manufacturers recommendations.

4. An in-line dispensing system was added to the farm water supply and zinc monohydrate (Agri-feeds) added at manufacturers recommended rate.

To date (May 2002) no symptoms associated with FE have been detected in any deer but this season has been a moderate to low challenge year only.

Discussion

FE in deer presents with a range of symptoms, many being the result of direct cytotoxic effects on the intestinal and urinary system and the acute liver/photosensitivity pattern reflected by high GGT. levels less common, will zinc supplementation alone be effective as a preventative? The use of GGT levels also may not provide the full picture on protection provided/damage incurred. The answers to these questions are beyond my skills as a clinician and will require further work.

The appropriate control program for individual properties will involve many risk management and topographical considerations and simple solutions are most unlikely. If climate changes and global warming continue this problem will affect most upper North Island deer farmers to some degree, and increasingly, those further south. The personal cost to my operation over three bad years has exceeded $100,000 and ranks clearly as the No. 1 health issue for my operation, and would be likewise on similar farms. Yet no industry/commercial funding has targeted the problem.

The findings and conclusions I draw are my personal interpretation of clinical observations combined with the limited scientific information on hand currently. I cannot conclusively exclude the possibility of other fungal or toxic agents unknown being partially or largely responsible for the clinical patterns described.

References

1 Mortimer PH. (1984) Facial Eczema in Red and Fallow Deer. Proc. Deer Course for Veterinarians, Deer Branch NZVA 1: 59-63

2 Mortimer PH, Smith BL. (1981) Facial Eczema in Deer. Proc. Ruakura Farmers Conference, pp 109 – 112.