Facial Eczema in Red and Fallow Deer

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ABSTRACT

Paucity of information on the susceptibility of farmed deer to sporidesmin, the facial eczema mycotoxin, prompted trials in which red and fallow deer were experimentally challenged with sporidesmin.

Red deer, initially refractory to a first challenge, developed mainly asymptomatic liver and urinary tract lesions found at autopsy after a second challenge.

Fallow deer, given the same initial challenge dose, rapidly developed severe disease with symptoms of respiratory distress, icterus, photosensitisation, tongue and lip ulcerations and a high mortality rate. Autopsies revealed severe hepatobiliary lesions with profound generalised icterus, haemorrhagic ulcerative urinary-tract lesions and in some cases intestinal ulcerations.

Natural outbreaks of facial eczema occurred in deer shortly after the trials. Investigation of these revealed a sequence of clinical signs and pathological changes which complemented the findings in the sporidesmin-dosage trials.

I Introduction

The mycotoxic disease of facial eczema (FE) occurs mainly in the warmer North Island districts in some summers when specific climatic events encourage the rapid proliferation and buildup of toxic spores of Pithomyces chartarum on dead pasture litter. Close grazing of short pasture swards favours the ingestion of large numbers of spores containing the hepatotoxic secondary fungal metabolite known as sporidesmin. Susceptible sheep and cattle may develop an inflammatory cholangitis and pericholangitis which impairs mainly liver-excretory functions. If this injury is widespread throughout the liver, products of liver metabolism normally excreted in the bile are retained and regurgitated into the peripheral circulation. Symptoms of such an event are jaundice and the onset of a severe photodermatitis caused by photodynamic activation within the skin of the exogenous chlorophyll-derived porphyrin, phylloerythrin, by sunlight (see recent review by Mortimer and Ronaldson, 1983).

Over the last decade the farming of several species of deer, mainly of recent feral origin, has emerged as an important diversification of pastoral farming for animal products largely destined for overseas markets. This relatively new industry has rapidly established a fast growing population of farmed deer grazing at remarkably high rates of stocking mainly on pastures previously developed by sheep and cattle enterprises.

In 1980, there was little information on the susceptibility of deer to sporidesmin nor of the likely risks for the occurrence of facial eczema outbreaks on short-grazed toxic pastures in eczema-prone districts of the North Island.

For this reason two trials were initiated when small numbers of red and fallow deer were first made available to test their susceptibility to poisoning by sporidesmin obtained from laboratory cultures of P. chartarum.

In the autumn of 1981, and less than one month after these trials were terminated, one of the worst seasons was experienced in more than 10 years for the occurrence of facial eczema outbreaks. This was probably the first major challenge for natural occurrence of facial eczema in the recently enlarged populations of farmed deer in the Waikato. When the disease occurred, symptoms and lesions of facial eczema were examined from the Ruakura Agricultural Research Centre.

This is a brief account of the results of the susceptibility trials and of the clinical signs and lesions found in both the experimental and the naturally-occurring disease briefly reported elsewhere (Mortimer and Smith, 1981).

II Sporidesmin Susceptibility Trials

Lacking even basic information on susceptibility of deer to sporidesmin, three dose levels were selected; 0.3, 0.6 and 1.2 mg (total dose) per kg liveweight. The lowest dose rate was chosen because it had proved an effective challenge in routine sheep experiments at Ruakura.

A Red Deer

One year old male red deer spikers were used in four groups of four animals (3 dosed groups, 1 control) and drenched with water solutions of sporidesmin over three consecutive days.

These challenges produced no recognised clinical changes and no significant elevation of γ-glutamyl-transferase (GGT) levels in serum samples taken at weekly intervals. The group which received the highest sporidesmin challenge was autopsied 5 weeks after dosing. Livers were found to be little affected; only one liver showed a small area of fibrosis.

The remaining deer were regrouped and immediately rechallenged, one group at 0.6 mg, the other at 1.8 mg sporidesmin per kg liveweight. Again no clinical signs were produced.

Lesions Liver injury was indicated in two of the eight redosed deer when serum GGT levels increased from 12 and 18 to 522 and 602 iu/ℓ. Both these deer lost some weight and at autopsy their livers were small, showed a deep purplish plum colour and the lobular structure was exaggerated when compared with livers of controls. Mild fibrotic changes were also present. Livers of the other dosed deer were virtually normal in appearance and consistency.

A more constant finding was that of inflammation and oedema of the urinary tract which in some cases extended from the renal calyx, along the ureters and into the urinary bladder, where submucosal haemorrhages, oedema and ulcerations ranged from very localised to very extensive. Old standing severe lesions were usually deeply-coloured red, purple to almost black often appearing as rough, raised wart-like areas on the mucosal surface of the excised, collapsed bladder.

B Fallow Deer

Three groups of five 1 year old male fallow deer (spikers) were dosed with 0.3, 0.6 and 1.2 mg sporidesmin per kg liveweight respectively, given as an aqueous drench over three alternate days. These were the same amounts given to the red deer on their first challenge. A fourth group of five controls was not dosed.

Clinical signs Within 6 days of first dosage clinical signs were apparent and they became progressively more severely affected on each successive day. Affected deer stood or lay down away from the mob. They were extremely restless, showed frequent shaking of head and ears and frequently licked the lips and nostrils with their tongue. There was repeated rubbing of the lower jaw, the muzzle, the eyelids and ears either on the ground or on fixed objects. Some showed a phase in which their sight was apparently impaired for they frequently walked into obstructions. Membranes of the eyes and mouth became deeply icteric, and the frequently-voided urine became deep yellow in colour. Skin lesions of photodermatitis developed and they were particularly obvious around the eyes, muzzle and lips, and occasionally at other sites. Severe excoriation of the skin under the jaw, on the muzzle and around the eyelids followed as a result of rubbing. Closer examination revealed severe ulceration of the tip of the tongue with the necrotic glossitis extending deep into the underlying muscle tissues.

All dosed deer either died or were killed to terminate their distress. In the high dose group (1.2 mg/kg) deaths occurred between 7 and 10 days after the first dose, between 10 and 14 days at the median dose (0.6 mg/kg) and between 14 and 24 days at the low dose (0.3 mg/kg). Serum GGT levels ranged up to 460 iu/ℓ 2 weeks after dosing at which time only four of the 15 dosed animals remained.

Lesions In all deer, liver injury was marked. Most livers were enlarged with rounded edges and were severely icteric showing a deep orange-brown discolouration throughout. The lobular pattern was most pronounced in some. Many bile ducts were plugged with inspissated bile and detritus and there was oedema and fibrous thickening of peribiliary tissues. Some generalised diffuse fibrosis could be detected in livers from deer surviving for more than 10 days. In the urinary tract severe submucosal haemorrhages and ulcerative cystitis were constant findings. Oedematous thickening of the ureters was common and occasionally it had caused some obstruction to urine flow with a resultant dilatation of the renal calyces and hydronephrosis.

Intestinal ulceration, mainly in the region between the pylorus and the point of emergence of the common bile duct, was found in several deer. Blood clots from haemorrhages into the lumen were present in two cases.

Some ulcers had led to bowel perforation which had resulted either in a localised peritonitis restricted by adhesions usually to the liver or, occasionally, had produced a severe generalised peritonitis which had contributed to the cause of death.

In the most acute and severely affected animals, which had died within 7 to 10 days, marked pulmonary oedema and extensive interstitial emphysema was a constant finding. These animals had previously shown profound respiratory distress prior to death and this lesion was considered to be the major factor in the early death of the high dose group.

III Field Outbreaks of Facial Eczema in Deer

In March 1981, the occurrence of field outbreaks presented the opportunity to examine naturally occurring cases of FE, especially in fallow deer. This was particularly timely for the comparison of findings from these cases with those from the sporidesmin dosage trials recently completed.

A severe outbreak in one particular herd resulted in the death or slaughter of 26 markedly affected fallow deer which included over half the breeding does (2 to 3 year old) in the herd.

From the observations and clinical examinations made on the herd at the height of the outbreak, all the severe behavoural reactions to photosensitisation, the photodermatitis of the ears, eyelids, lips and perineum, as well as the occurrence of blindness in some, were precisely as found in the experimental disease.

Autopsy examinations on 13 of these deer were carried out and recorded. They showed the same range of severe lesions as those previously described. However, the more protracted course of the natural field disease probably accounted for the more severe and extensive skin and tongue lesions of photosensitisation. As well, livers generally showed less acute changes, were not generally enlarged but were more fibrotic in consistency and atrophy was present in some lobes. Later in the outbreak there were early signs of nodular regeneration in the livers. Lesions of urinary cystitis were present in all animals and were generally very severe. Although lesions of pulmonary oedema and interstitial emphysema were present in some animals, they were less severe and not nearly so striking as in the experimental disease.

Turning to red deer, field cases of FE were much less numerous than for fallow deer. Access was kindly given to specimen submission reports of the Ruakura Animal Health Laboratory involving both red and fallow deer over the period of the outbreak. From this information it was clear that the same range of clinical signs and the same pattern of pathological changes occurred in the smaller sample of case histories and submissions involving red deer.

During and subsequent to the 1981 outbreak an attempt was also made to determine the extent of subclinical FE liver injury in the region by assaying for elevated GGT enzyme levels in serum from herds of deer on 20 cooperating properties. Liver injury was indicated in deer of both species but with several times greater frequency in fallow than in red deer herds. Two properties grazed both red and fallow deer and on both properties the indicated liver injury was almost wholly restricted to fallow deer.

IV Conclusions

Clinical and pathological findings in these dosage trials and in the 1981 FE outbreaks showed that both red and fallow deer were susceptible to sporidesmin poisoning. FE disease apppeared to be closely similar in the two species of deer, and both species showed basic similarities to sheep with FE. This was especially true of the liver lesions.

The clinical effects of photosensitisation appeared to be much more severe and stressful in deer than in sheep or cattle and death soon intervened in a high proportion of fallow deer if there had been photosensitisation. How effective the prompt provision of full shade would prove to be in reducing

losses is not known but, as with sheep and cattle, it could be very effective. In deer, an added complication to the usual FE photodermatitis as seen in sheep and cattle, is the severe ulceration of the tongue in deer and it develops into a localised but severe glossitis which to a grazing animal is likely to be very painful and restrictive in feeding. Another distressing symptom is the apparent loss of sight. The association of the readily observed deep-yellow pigmentation in the aqueous humour of the eye with impaired vision is not clear. This effect has not been reported in sheep or cattle with icterus of FE origin.

The marked haemorrhagic inflammatory lesions and ulcerations in the urinary system, found in both species of deer, are also sometimes featured in sheep and cattle, but not with the severity commonly found in deer. Since the lesion is held to be a direct cytotoxic effect of sporidesmin excreted in the urine it may indicate that in deer more of this mycotoxin finds a renal pathway for excretion. The severe and protracted cystitis produced probably causes further stress.

It is likely that, as deer farming operations multiply and intensify, the potential for FE occurrence among deer herds in certain North Island regions will also escalate. Seasonal prophylactic control will be required to safeguard the high and growing investment in the industry. Certain control measures devised for sheep and cattle should prove equally as effective for farmed deer. These measures are described in full detail in the current series of AgLinks on FE.

AgLink FPP 493 - "Facial Eczema; Causes, Effects and Treatment",
AgLink FPP 494 - "Facial Eczema; Predicting Danger Periods"
AgLink FPP 495 - "Facial Eczema; Fungicide Control"

The use of zinc salts for FE prevention, either by drenching or by addition to drinking water or spraying onto pasture, has not been investigated for use with deer. Therefore no recommendations can currently be made, either in regard to its effectiveness in FE prophylaxis or in practical methods for its use.

V References

Mortimer, P.H., and Ronaldson, J.W. (1983). Fungal Toxin-Induced Photosensitization. In "Handbook of Natural Toxins, Vol 1, Plant and Fungal Toxins" Eds, R.F. Keeler and A.T. TU. Ch. 11, pp. 361-419, Marcel Dekker Inc., New York.

Mortimer, P.H., and Smith, B.L. (1981). Facial Eczema in Deer. Proc. Ruakura Farmers' Conf., pp. 109-112.