

OUTBREAK OF MALIGNANT CATARRHAL FEVER IN PÈRE DAVID'S DEER

Marjorie Orr, Invermay Animal Health Laboratory, Mosgiel
and Colin Mackintosh, Invermay Agricultural Centre, Mosgiel

Invermay Farm imported 24 Père David's deer in April 1986 from England. In just over six months, 11 (46%) had died of Malignant Catarrhal Fever (MCF), followed by two more a few months later, bringing the total dead to 13 (54%). The other four farms which also imported Père David's deer at the same time had a similar experience - all lost about half of their Père David's deer to MCF.

We conclude that Père David's deer are particularly susceptible to MCF. Although we acknowledge that stress can predispose deer to MCF, wapiti and red deer have been imported under the same conditions and exposed to the same stresses without post-importation outbreaks of MCF.

The outbreak at Invermay was expensive and depressing but it did provide plenty of material for the study of MCF. This paper will present an account of the clinical signs and the gross and histological lesions of MCF in Père David's deer.

Inevitably, the 24 Père David's deer imported to Invermay were severely stressed - they had been captured in December and January 1984/85 in Woburn Park where they had enjoyed a hitherto untroubled life, with little handling. The deer, two 2-year old stags, 4 yearling stags and 18 yearling hinds spent four weeks in quarantine before being transported by air to New Zealand and spending a further four weeks in quarantine at Silverstream. After a 20 hour truck journey they arrived at Invermay on the 2nd of April to a strange new environment and another winter.

Nevertheless the first cases of MCF did not occur until the end of May, nearly two months after importation. The two 2-year old stags had begun to show rutting behaviour and the group had been subdivided into male and female groups. Two days later, the two 2-year old stags died of acute MCF within 24 hours of each other. The third case, a hind, became depressed on the 11th of June, developed diarrhoea and died three days later. In the eight weeks that followed seven more deer died, and further single deaths occurred in mid-October and in January and February the following year. The clinical duration of the disease in eight of the deer was three days or less. One deer was ill for five days before death and four deer survived for two to five weeks. Generally the cases which occurred early in the outbreak were of short duration, whereas later in the outbreak affected deer survived for longer. However, two deer which died several months after the main cluster of cases showed illness of only 1-3 days duration (Table 1.).

Few detailed clinical examinations were carried out because observations were generally made at long range to minimise stress. Consequently the apparent duration of illness may have been underestimated in some cases.

Depression and a hunched stance were the most common clinical signs occurring in all affected deer except two. One of these showed hyperaesthesia and one was found dead. Dysentery occurred in three early cases and one of the last cases, and diarrhoea developed in all but one of the remainder. In the four deer which survived for 16 days or more, weight loss occurred. Three of these became blind as a result of haemorrhage into the anterior chambers of both eyes; in two there was a crusty exudate

around the muzzle. Five of the six males died, including both 2-year-olds. A summary of clinical signs is shown in Table 1.

Treatment with long acting tetracycline did not appear to alter the course of the disease.

There were no consistent gross abnormalities at necropsy, although in twelve of the thirteen deer there were various recent localised haemorrhages. The first seven and the last two deer to die all showed haemorrhage into the large intestine. In three of the four cases which survived for 16 days or more there was haemorrhage into the anterior chamber of both eyes. One deer which had been recumbant for several days had numerous pale streaks in the main muscle masses of the hind limbs. Table 2 presents a summary of necropsy findings.

A diagnosis of MCF was made in all deer, based on the histological demonstration of a vasculitis. The vasculitis was most dramatic in the brain, where the media and perivascular space of many medium sized arterioles were infiltrated by mononuclear cells, mostly lymphoid cells. The lesion was most frequent in the white matter of the cerebellum. A similar vascular lesion was often present in the lung, and in a few deer in the liver, kidney and mesenteric lymph nodes. In the three cases with intraocular haemorrhage, there was arteriolitis in the ciliary body of the eyes. Two of these deer also had vasculitis and thrombosis of arcuate arteries in the kidneys. In the skeletal muscle of the deer with gross muscle lesions, there was massive myonecrosis - perhaps the result of crushing during recumbency. In most deer there were light interstitial infiltrates of mononuclear cells in the lungs, and in some these were also evident in the liver and kidneys. In the meninges of five of the more chronic cases there was marked proliferation of endothelial cells.

Père David's deer appear to be particularly susceptible to MCF. There are already several reports of heavy losses in Père David's deer in zoos as a result of MCF. The concept that there are species differences in susceptibility to MCF is not new; it has already been suggested that Sika deer in New Zealand are more susceptible than red deer with herd losses of up to 29% (Anon, 1980). The reasons for this increased susceptibility are not clear. It is possible that the relative geographical isolation of Père David's deer has resulted in their greater susceptibility or that the considerable inbreeding in this species has impaired their immunological competence.

The gross and histological changes in MCF in Père David's deer did not differ significantly from those of MCF in other breeds of deer commonly diagnosed at Invermay Animal Health Laboratory. However, in the Père David's deer the histological lesions tended to be more severe, and the haemorrhage in the anterior chambers of the eyes was a noteworthy feature. In our experience intraocular haemorrhage is rare in red deer.

The histological vasculitis which is diagnostic for MCF in New Zealand, is like that described by Jubb et al., (1985) for MCF in cattle, deer and other ungulates and by Clark (1975) for MCF in deer in the USA. However, workers in the UK (Reid et al., 1986) reported that necrosis of the epithelia of the digestive and respiratory tracts, hyperplasia of lymph nodes and interstitial accumulations of lymphoid cells in many tissues were the characteristic lesions of MCF, and vasculitis was not a prominent feature. The apparent difference in histological lesions between MCF in deer in the UK and in deer in New Zealand and the USA may perhaps result from virus strain differences.

It is likely that sheep can excrete a form of MCF virus which is pathogenic for deer (Reid et al., 1986). Although at Invermay direct contact with sheep was scrupulously avoided, indirect contact by workers, other vectors or effluent could have been sufficient to transmit the disease. On most deer farms in N.Z. it is probably impractical to prevent indirect as well as direct contact between deer and sheep. We may simply have to accept that as long as there are sheep in N.Z., deer and in particular some breeds like Père David's will always be disadvantaged because of their vulnerability to MCF.

REFERENCES

- Anon (1980): Malignant catarrhal fever outbreaks in sika deer. *Surveillance* 7 (4): 13.
- Clark, K. A. (1975): A study of vascular lesions of malignant catarrhal fever in deer by histopathology, electron microscopy and immunofluorescence. Texas A & M University, Ph.D.
- Jubb, K.V.F.; Kennedy, P.C.; Palmer, N. (1985): Pathology of Domestic Animals, Third Edition, Academic Press Inc. 2: 102-8.
- Reid, H. W.; Buxton, D.; Pow, I.; Finlayson, J. (1986): Malignant catarrhal fever: Experimental transmission of the 'sheep associated' form of the disease from cattle and deer to cattle, deer, rabbits and hamsters. *Res. vet. Sci.* 41: 76-81

Table 1: Summary of clinical signs of MCF in an outbreak in Père David's deer

Deer Number	27	26	18	20	6	14	9+	17	Y171	1	Y163	19	8
Age (years)	2	2	1	1	1	1	1	1	1	1	1	2	2
Gender	M	M	F	F	M	F	F	F	M	M	F	F	F
Depression	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	
Hyperaesthesia		✓											
Diarrhoea			✓		✓	✓	✓		✓	✓	✓		✓
Dysentery	✓	✓		✓								✓	
Weight loss							✓		✓	✓	✓		
Haemorrhage in eyes									✓	✓	✓		
Muzzle crustiness										✓	✓		
Duration of illness (days)	2-3	1-2	2-3	2	5	3	29	1	16	33	21	2	1
Date of death	30.5.85	30.5.85	14.6.85	17.6.85*	1.7.85	11.7.85	17.7.85	18.7.85	22.7.85	10.8.85	14.10.85	24.1.86*	14.2.86

* Euthanasia
+ Treated
M Male
F Female

Table 2: Summary of post mortem examination findings in an outbreak of MCF in Pere David's deer

Deer Number	27	26	18	20	6	14	9	17	Y171	1	Y163	19	8
Oral ulcers	✓												
Pulmonary oedema	✓	✓											
Abomasal ulcers	✓	✓					✓ch						
Haemorrhage in intestine	✓	✓	✓	✓	✓S1	✓	✓					✓	✓
Mesenteric lymph node enlargement				✓					✓			✓	
Kidney infarcts									✓				
Haemorrhage in anterior chambers of both eyes									✓	✓	✓	✓	
Emaciation							✓		✓	✓	✓	✓	
Blood from nose									✓	✓	✓		
Mediastinal blood clots										✓			
Pale streaks in muscles of hind limbs									✓				

S1 - slight
Ch - chronic