PASTEURELLOSIS - A CASE HISTORY

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INTRODUCTION

During the winter of 1989, septicaemic Pasteurellosis was diagnosed as the cause of death of 13 out of a herd of 100 Fallow deer over a one month period.

Prior to this incident there had been no reported cases of Cervine Pasteurellosis in Australia.

THE PROPERTY

The property where the deaths occurred is situated in the central tablelands of New South Wales, 300 kilometres west of Sydney at an elevation of 900 metres above sea level. It consists of 50 hectares of undulating improved pastures with shelter belts of Pinus radiata and stands of native eucalypts. Sheds are provided for additional shelter. The paddocks are watered by dams.

The pasture in the winter of 1989 was a dense sward of clover, rye grass, paspalum and cocksfoot.

THE DEER HERD

Established in 1975, the deer herd totalled 300 head and was comprised of 150 Fallow, 60 Red, 50 Rusa and 40 Chital.

All deer were in good condition and quiet, due to daily contact with the owner when they were fed a supplement of lupins, oats and stale bread.

There had been no recent introductions to the herd, and prior to the deaths, management was routine. The deer had not been yarded for 7 weeks.

OTHER STOCK

Two horses, several sheep, a collection of semi-tame kangaroos and a large number of wild waterfowl, mainly ducks. The ducks grazed with the deer and competed with them for spilt grain.

THE DISEASE OUTBREAK (15th June to 18th July)

Deaths occurred in two outbreaks - the first 7 deer died over a five day period from June 15th, three weeks later a further six deer died.

Twelve does of mixed ages and one castrated buck died.

All deaths were in one mob of Fallow in a single paddock. Fallow and other species in adjoining paddocks were unaffected.

Both outbreaks were preceded by several days of cold wet and very windy weather, with sleet and wind-blown snow. A weather station 10 km from the property recorded wind runs of greater than 300 km on the two days in June immediately before the first deaths, and on the three days in July before the second mortalities (a wind run exceeding 300 km indicates a very windy day).

These high winds were accompanied by rain or sleet -15 mm on each of the June days and 18, 13 and 2 mm on the three July days, and low temperatures.

These conditions, combined with water-logged pastures, resulted in the deer being wet and chilled for several days.

The weather between the outbreaks was milder and sunny with less wind.

In June, six of the seven deer were found dead without premonitory signs. Deer 7 was noticed away from the herd and died four hours later.

This outbreak lasted five days, and veterinary advice was not sought until day 3. By the time an accurate diagnosis had been made, deaths had ceased. Medication was not attempted.

In July five deer died without premonitory signs. One deer was found with severe respiratory distress and was given long-acting oxytetracycline* but died shortly after treatment.

No deer exhibited any signs of disease and subsequently recovered.

When deaths recommenced in July in-feed antibiotic medication was instigated using oxytetracycline powder**. Sensitivity tests showed the bacteria to be sensitive to this antibiotic. This was refused by all deer and was replaced by Trimethoprim/ Sulphadimidine powder*** at the rate of 30 mg per kilogram daily for five days. The amount was roughly calculated by estimating the total weight of the herd, calculating the amount of powder, mixing with a known quantity of feed, then distributing in a number of troughs enabling each deer to eat without competition. The medicated feed was readily eaten by all remaining deer and no further deaths occurred.

There was, however, at this time a great improvement in the weather.

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* Terramycin LA. Pfizer Agricare Pty Ltd
** Terramycin 200 Feed Supplement. Pfizer Agricare Pty Ltd
*** Trimidine Powder. Parnell Laboratories.

LABORATORY FINDINGS

Post mortem appearance of carcases was unremarkable however, several showed torticolis and most had a bloody nasal discharge.

Eight deer were necropsied at the Regional Veterinary Laboratory, Orange - four from each outbreak (Carrigan *et al* 1991). All animals showed moderate to advanced decomposition and all had a frothy, blood-stained nasal discharge.

Gross Pathology was divisible into three categories :

Deer 1, 4 and 6 -

* Generalised congestion of carcase
* Petechial and ecchymotic haemorrhages sub-pleurally and over rib cage and diaphragm
* Sub-serosal haemorrhages on abdominal viscera
* Lungs congested and oedematous
* Airways froth-filled
* Small intestines thin-walled and with fluid, blood-tinged contents.

Deer 2, 3 and 5 -

* Numerous sub-pleural petechial haemorrhages over rib cage
* Severe fibrinous pleurisy and pneumonia
* Thick layers of fibrin in overlying parts of lung with underlying consolidation and severe interlobular oedema giving a marbled appearance on cut surface
* Small intestine, fluid, blood-tinged contents.

Deer 7 and 8 -

* Extensive petechial and ecchymotic subcutaneous haemorrhages over the shoulder, rump, thorax and back.
* Skeletal muscles, especially longissimus dorsi and muscles of abdominal wall, were very congested and oedematous and oozed blood-tinged fluid from inter-muscular fascia.
* Large ecchymotic splash haemorrhages sub-pleurally over the rib cage and in the muscle of the diaphragm.

* Severe fibrinous pleurisy with adhesions between lung and ribs was present in deer 8.

* Small intestinal contents fluid and blood-tinged.

HISTOPATHOLOGY

Histopathology was performed on six deer.

Deer 2 and 3 : most significant pathology was a severe pneumonia and pleurisy.

Deer 1, 4, 6 and 7 : moderate pulmonary congestion and oedema but no inflammatory cell response.

Deer 7 : myocardial congestion, sub-pleural haemorrhage, mural haemorrhage and oedema in pulmonary artery and mild interstitial oedema of skeletal muscles.

Other tissues examined, including brain, liver and kidney, were moderately congested but otherwise unremarkable.

BACTERIOLOGY

P. multocida was isolated in pure growth, or was the predominant organism in mixed growth, from tissues of 7 of the 8 deer examined. Deer 8 had been treated with oxytetracycline. The sites from which *P. multocida* was recovered in each deer are given in table 1.

TABLE 1

Sites of *P. multocida* isolation in 8 deer

Deer No.	Lung	Kidney	Liver	CSF	Small Intestine	Thoracic fluid
1	+*	+				
2	+	-	-			
3	+	+	+	+	+	+
4	+	+	+	+	(+)#	
5	+	+	+	+		
6	+	+	+	+	(+)	
7	+	+	+	+		
8	-	-	-	-		

\+ *P. multocida* isolated in pure growth or as the predominant organism in mixed growth.

A blank space means the site was not cultured

\- site culturally negative for *P. multocida*

(+) heavy mixed growth predominated by coliform.

From Carrigan *et al* (1991).

Four isolates were submitted to the Regional Veterinary Laboratory, Benalla, Victoria. They were all found to be Carter Group A (Capsule type), Heddleston type 3,4 (somatic antigen type).

OTHER LABORATORY TESTS

Serum from deer 7 was tested for Epizootic haemorrhagic disease, bluetongue, pestivirus and arbovirus with negative results.

Selective cultures for *Yersinia* spp. and *Salmonella* spp. were negative.

The intestinal contents from three deer were negative for the Epsilon toxin test for *Clostridium perfringens* type D.

DISCUSSION

Pasteurellosis, due to *P. multocida*, has been infrequently reported as a cause of deer mortalities. The disease has not previously been reported in Australia.

The nature of the outbreak - sudden death, the necropsy findings and isolation of *P. multocida*, was suggestive of haemorrhagic septicaemia which does not occur in Australia and is caused by organisms of serogroup B or E and somatic serotype 2 (Heddleston *et al* 1972).

There were similarities to the outbreak described by Jones and Hussaini in the United Kingdom in 1982 and 22 of 60 mature fallow deer died over a two month period. The few deer seen ill were described as showing an obvious swelling of the head and neck, dilation of the nostrils and to be panting. The factors predisposing to the septicaemia were not defined. The *P. multocida* strains in this outbreak all belonged to capsule serogroup B somatic serotype 3,4 (Rimler *et al* 1987).

P. multocida serogroup A is ubiquitous and is responsible for sporadic infections in many species. The source of the organisms in this outbreak was not established and they may represent part of the normal flora in the deer. The deer had close contact with wild ducks and it is unfortunate that it was not possible to examine them for the presence of the bacteria.

Beveridge (1983) states that *Pasteurella* spp. organisms are common commensals of the nasopharynx and cause disease when

predisposing factors weaken the defence mechanisms of the respiratory tract. When this occurs, apparently virulent clones are selected and these strains with enhanced pathogenicity may spread to other animals of the same species. Predisposing factors are viral, mycoplasma and chlamydia infections, transportation, exposure to cold, wet conditions, crowding, malnutrition, dipping, trauma, hunger and dehydration.

Both outbreaks were preceded by wet, cold and very windy days and, as no other stressing factors can be identified, it seems likely that these weather conditions chilled the deer sufficiently to precipitate the disease.

The issue is confused by the fact that similar weather in the winter of 1990 did not result in deaths.

There is no obvious reason why deaths were confined to one paddock other than that very close contact is required for spread between animals. All pastures were equally waterlooged and equally exposed to the weather. Rusa and Chital deer, usually regarded as being more sensitive to environmental stress, in close proximity were unaffected. The popula tion of wild waterfowl, a possible source of infection, seemed to be evenly distributed over the farm.

In the UK outbreak, deaths were confined to one group of 60 mature bachelor bucks out of a herd of 264 deer on 84 hectares. The bucks had a defined territory within this estate.

It is possible carrier animals contaminated the pasture and that this infective discharge was ingested by susceptible animals. The organism of haemorrhagic septicaemia of cattle has a short survival time on pasture of less than 24 hours in Asian conditions (Bain 1963). This may help explain why the disease was confined by a fence.

The value of the antibiotic medication is hard to assess for, although deaths stopped with treatment, there was at the same time, an improvement in the weather.

The manufacture of a customised vaccine from the isolates was considered as an option had the deaths persisted.

Stressful weather was most likely the eliciting cause of this outbreak. The source of infection remains a mystery.

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