Johne's Disease : The Current Situation in New Zealand Deer

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Abstract

Johne's disease (JD) is emerging as a serious problem on deer farms in New Zealand. The numbers of newly infected herds were 4, 7, 22, 27, 46 and 40 for the years 1992 to 1997 respectively. The majority of cases were discovered at meat inspection but there has been an increasing number of multiple cases occurring on deer farms, especially in rising yearling animals. Clinically affected deer typically are in poor condition, have rough coats, scour and have green faecal soiling around the tail. For confirmation of clinical cases the gel diffusion (GD) test and faecal smears are most useful. Both sheep and cattle strains of *Mycobacterium paratuberculosis* can infect deer. Currently the success rate of culture is good for the cattle strain, but poor for the sheep strain. On necropsy the carcass is typically emaciated and there is likely to be enlargement and abscessation of the mesenteric lymph node chain. There may not be gross thickening of the terminal ileum. Lesions due to *M.paratuberculosis*, *M avium* and *M bovis* can appear grossly and histologically identical and therefore identification relies on culture and/or the polymerase chain reaction (PCR) test. Control is difficult and relies on early culling of affected animals, together with serological testing and slaughter of positives. It is important to prevent the introduction of JD onto uninfected deer farms. The development of a deer industry programme is essential to control the spread of JD.

Introduction

The first confirmed case of Johne's disease (JD) in a red deer in New Zealand was in 1986. It occurred in a 3 year old hind which was born on the property in question and it lost weight and had diarrhoea over a 2 month period. At necropsy it was emaciated and showed thickening of the ileum, lesions in the mesenteric lymph nodes, had microscopic lesions typical of JD in the intestines and draining lymph nodes and *Mycobacterium paratuberculosis (M ptb)* was isolated from fresh material. (Gumbrell, 1986).

Five previous cases of JD were suspected between 1979 and 1985 but none were confirmed by culture. Since then the numbers of individual cases and affected farms have risen exponentially. In the last 6 years the numbers of newly infected herds (defined as a herd in which *M.ptb* was identified by culture or PCR from deer for the first time) were 4, 7, 22, 27, 46 and 40 from 1992 to '97, respectively. The overwhelming majority of the infected animals were discovered at meat inspection as having lesions resembling those caused by *Mycobacterium bovis* but recently there has been an increase in clinically affected animals. Another alarming development has been the increasing number of multiple cases of JD on deer farms.

Previously clinical JD tended to be single sporadic cases in young adults. However, in the last year there have been over 7 outbreaks and most of these cases have involved rising yearlings. Despite the increased reporting of cases, we believe this is the tip of the iceberg because the examination of viscera in the Deer Slaughter Premises (DSPs) is not sensitive enough to detect early cases and it appears that many farmers and veterinarians are either not recognising or not reporting clinical cases of JD on farms. The number of cases in the DSPs suggests that many clinical cases on farms are going undiagnosed or unreported. It is important for the deer industry to determine the true prevalence of JD in New Zealand farmed deer so that control strategies can be put in place.

The following is a summary of the disease in red deer, a discussion of diagnostic tests, potential control measures and prospects for the future.

Clinical signs of JD in deer

Sporadic cases of JD have occurred in all ages and classes of deer. However, recently outbreaks in young deer have become more common. Initially the farmer is likely to notice that there are 5-10 % of a mob of deer which "fail to thrive", have low growth rates or are in poor condition. In spring they may fail to lose their winter coats or have a patchy or "moth-eaten" appearance. They start to scour and develop obvious soiling with green faecal material around the tail, hind quarters and hocks, and they start to lose weight. The disease course can be from days to months but generally it appears that the younger the animal, the quicker the progression to emaciation and death. In outbreaks, the most common age of onset is 8 to 20 months, compared with 2-4 years in cattle and sheep. Recent outbreaks have had mortality rates of up to 12% in rising yearlings. Sporadic cases of JD may also occur in older deer. The differential diagnosis includes yersiniosis (in weaners in winter), abomasal parasites, fading elk/wapiti syndrome, bovine tuberculosis (Tb), avian Tb and chronic MCF.

Post mortem signs

Typically in deer which have clinical JD the carcase is emaciated and there are likely to be enlarged, abscessed mesenteric and ileo-caecal lymph nodes. There may be oedema of the mesenteries and there may or may not be gross thickening of the mid or terminal ileum and/or caecum. In clinically normal animals the most common lesion which is detected at a slaughter is an abscess in a mesenteric lymph node.

Diagnostic tests

A range of tests and their potential value are shown in Table 1. Many of these tests were developed to diagnose JD in sheep and cattle and have not been optimised for use in deer or fully evaluated in this species. From our experience an ante mortem serum sample from a deer with clinical JD will show low serum albumin and total protein levels indicating a protein-losing enteropathy, but this is not specific for this disease. The most sensitive serological test for red deer appears to be the agar gel diffusion (GD) test. In a small study conducted recently, on a farm which experienced a serious outbreak of JD in rising yearling deer, serum

samples were taken from 9 clinically affected animals which were subsequently killed, necropsied and confirmed as JD cases. The GD was positive in all 9 animals compared with 4/9 ELISA and 2/9 CFT (see Table 2). Paired serum samples and fixed samples of ileo-caecal valve, ileo-caecal lymph nodes and suspicious lesions in mesenteric lymph nodes were also taken from 103 deer, from this same farm, which were killed through a DSP. Approximately half these animals were in a mob of deer which had been brought onto the farm in autumn, kept separately until spring and which had no cases of JD. Results of various serological tests and histopathological findings are summarised in Table 2. The farmer identified 6 deer from the affected mob which were in slightly poorer condition or had mild faecal staining around the tail. Three of these animals were found to have positive serology and lesions of JD. None of the remaining 97 deer showed clinical signs of JD.

The results suggest that the GD test is useful for identifying JD (or avian Tb, see later) in clinically affected deer and is moderately effective at detecting animals with subclinical infections. It is not possible on this data to make categorical statements on the specificity of these three tests because they were only compared with histopathology which is not sensitive or specific enough for this purpose. Furthermore, false positive reactions have been observed in deer infected with *M. avium*.

M. ptb/M. avium

The major problem with serological, skin and lymphocyte transformation tests as well as histopathological examination of tissues is that they cannot distinguish between JD and avian Tb. *M ptb* is considered by many to be a subspecies of *M. avium* and there is a very close homology between them. As reported last year (Mackintosh *et al.*, 1997), an investigation of an outbreak of avian Tb in deer showed that clinically affected animals had clinical signs and gut lesions indistinguishable from JD. All these clinically affected deer, as well as a proportion of clinically normal deer that had mesenteric LN abscesses at slaughter, showed some positivity to the GD, CFT and ELISA tests. The GD test appeared to be the most sensitive test at detecting animals with lesions.

At DSPs the discovery of lesions in the mesenteric or ileocaecal (IC) lymph nodes (LNs) causes considerable problems because of the similarity between lesions due to *M bovis*, *M. avium* and *M. ptb* when examined grossly and histopathologically. Venison from deer infected with *M bovis* cannot be exported, whereas meat from animals with *M. avium* and *M. ptb* can. The return for venison for export is twice that for local consumption. The differentiation of these three diseases is technically challenging and is made even more difficult by the requirement for a quick, definitive diagnosis. The development and use of the new PCR test has markedly sped up this process (de Lisle et al., 1996). In addition, the PCR test can equally well detect the bovine strains and the difficult to culture, ovine strains. However, culture remains the "gold standard" for differentiating *M. bovis*, *M. avium* and *M ptb* because it is generally the most sensitive, and isolates can be characterised and typed to show strain differences in order to provide epidemiological information. Apart from distinguishing them from bovine Tb, it is extremely important to differentiate avian Tb and JD because of the epidemiological implications and the different control and prevention measures required.

Table 1. Diagnostic tests and their Properties

Sample	Test	Properties
Blood - serum	- biochemistry, total protein, serum albumin	- low TP, SA indicative protein losing enteropathy - not specific for Johne's Disease
- serum	- agar gel immunodiffusion (GD)	- most sensitive serological test in deer
	- complement fixation test (CFT)	- not very sensitive
	- absorbed ELISA	- moderate sensitivity
- heparinised blood	- lymphocyte transformation (Deer Research Lab) (plus ELISA =BTB)	- sensitive but cannot differentiate JD/avian Tb
	- gamma interferon	- not available for deer
Skin test	- avian/johnin PPD	- unlikely to be useful because they cannot differentiate JD/avian Tb
	-	-
Faeces	- smear for clumped AFOs	- not very sensitive, useful when applied to clinically affected deer
	- culture	- success rate good for cattle strain, poor for sheep strain, positive cultivars can be typed
	- polymerase chain reaction (PCR)	- highly specific for *M ptb* but moderate to poor sensitivity when used on faeces due to the presence of inhibitors
Necropsy	- gross appearance	- useful in latter stages of JD, but cannot differentiate from avian or bovine Tb
	- histopathology - AFOs	- moderately sensitive, but cannot differentiate from avian or bovine Tb
	- typical lesions	- moderately sensitive, but cannot differentiate from avian or bovine Tb
	- culture of fresh tissue	- definitive, high sensitivity for cases caused by cattle strain, allow typing of isolates
	- PCR of fresh tissue	- very specific, moderate to high sensitivity in detecting both cattle and ovine strains

Table 2. Serological and post mortem results of a deer herd with Johne's Disease

	No	Positive		
	n.	GD	ELISA	CFT
Clinically affected deer confirmed as JD	9	9	4	2 (2 sus)
Deer killed through DSP				
Histo positive	14	5 (1 sus)	5	0
Histo negative	89	1 (1 sus)	6	0 (1 sus)

Epidemiology

There is a dearth of specific research into the epidemiology of JD in deer. However, there is evidence that deer are susceptible to both "cattle" and "sheep" strains of *M ptb* (de Lisle and Collins, 1993). Circumstantial evidence suggests that *M ptb* has been introduced into deer herds from cattle and sheep sources. Once introduced it appears to be maintained in the deer herd in the absence of other infected ruminants. In some recent serious outbreaks, JD has occurred in weaner deer as young as 8 months old. It is likely that infection was present in the breeding hinds, with deer calves becoming infected at an early age, probably prior to weaning in autumn. However, there is some evidence that adult deer are susceptible to *M ptb* infection, although they appear less likely to develop clinical disease than young animals (Mackintosh, unpub).

Over the last year there have been at least 7 outbreaks of clinical disease in rising yearling red or hybrid deer with mortalities of up to 10%. On one affected farm there had been a small number of sporadic cases of suspected JD in adult deer in previous years. This deer farm was established 10 years ago and over the last 5 years was expanded by fencing in adjacent areas of the sheep farm. JD has been confirmed in the sheep on this farm. On another farm, 6 clinically affected red x wapiti yearlings (12 month old), from a mob of 300, were GD positive and *M ptb* was confirmed by culture from one of them. The deer herd has been closed for 6 years, apart from the purchase of adult sire stags, and there have been no sheep grazed on the farm since 1991. However beef cattle are used periodically to graze the deer farm to assist with pasture management. The JD status of the beef cattle herd is not known, but one old cow scoured and died in 1995 without diagnosis.

With the high prevalence of JD in sheep throughout this country, the high prevalence of JD in dairy herds in some areas and the increasing prevalence of JD in beef herds (especially in areas where dairy heifers are grazed off-station on farms running beef cattle) there is a high risk that JD will be introduced onto deer farms if sheep and cattle are brought onto the deer farm to control pasture or at times when deer numbers are down or feed shortages occur.

Control

Currently the Chief Veterinary Officer has brought together a Steering Committee for a series of meetings primarily to assess the JD situation in livestock in New Zealand and to develop

recommendations for control. The following are not official recommendations, but include some of the possible options :

1. Depopulation : if a deer herd has endemic *M.ptb* infection then this is the option with the greatest chance of complete success, but is also likely to be the most expensive. Current recommendations in Australia are to de-stock for 2 years to allow the organism to die out in the environment. Alternative farming options include grazing horses, planting crops and making hay. A major problem in using depopulation as a control measure is the difficulties of obtaining deer for re-stocking which can be guaranteed free of infection with *M ptb*

2. Test and slaughter : this option would require repeated testing with a combination of tests which are both sensitive and specific. Further assessment of the currently available tests is required before any combination can be recommended for use in a Test and Slaughter control program. In addition to serological tests, skin tests and/or LT may be considered, but these are likely to be confounded by cross-reactivity with *M. avium*. Prompt isolation and killing of all clinically affected deer and culling of all poorly performing animals should be done in conjunction with test-and-slaughter.

3. Vaccination : Currently "Neoparasec", the JD vaccine used in sheep and cattle, is not licensed for use in deer in New Zealand. Experience in the United Kingdom with the Weybridge vaccine (which is a live attenuated *M. ptb* plus oil adjuvant vaccine similar to "Neoparasec") suggests that vaccination of all calves during the first 48 hours of life significantly reduces the incidence of clinical JD but does not prevent infection (Fawcett et al., 1995). It is believed (H. Reid, pers. comm.) that the level of infection is gradually declining after 10 years of vaccination and that, with time, *M ptb* may be eliminated from a herd. In New Zealand, vaccination will interfere with skin testing for Tb, especially if only the single mid cervical test (MCT) is used. It may be necessary to use the comparative cervical test (CCT), although this would be contra-indicated in Movement Control herds or in Vector Risk (Tb endemic) areas, where the lower sensitivity of the CCT may reduce the likelihood of detecting bovine Tb. There is also the issue of vaccination site lesions which contain AFOs. This may preclude venison from all vaccinated deer from export, greatly reducing venison returns.

All these options should be subjected to a rigorous cost/benefit analysis to determine the most economic and practical alternative.

Prevention

Currently there are no proven procedures for the cost beneficial eradication of JD from deer herds where the infection is well established. The following measures should be followed to prevent herds becoming infected;

1. Deer farmers should avoid grazing sheep and cattle on the deer farm. Pasture management can be achieved with correct stocking rates, with feed conservation into hay or silage and with topping, and weed control with spraying.

2. Maintain a closed herd. Test all sire stags brought on and only buy from sources demonstrably free from JD. The deer industry should investigate the desirability of a establishing a market assurance programme where farms could be certified as having no evidence of JD.
3. If buying in weaners for raising to slaughter weight, then keep them in quarantine for the duration.

The Future

In 1988, Gilbert van Reenan stated in an article in *The Deer Farmer* that "I am betting that JD will emerge and become identified as a disease of considerable economic importance to the deer industry over the next few years". This prediction is being fulfilled. We believe that the number of JD cases and infected herds will continue to increase unless effective, nation-wide control measures are introduced promptly. With increasing awareness of JD amongst farmers it is likely that live sales will be affected. Purchasers may refuse to buy replacement stock or weaners from any property on which JD has been diagnosed in deer and possibly from farms which have the disease in cattle and sheep.

An additional concern is the possible link between *M. ptb* and Crohn's disease in humans. If this link is proven, it could have serious consequences for the deer industry, as well as cattle and sheep industries. Consumers are becoming increasing concerned about food safety as was demonstrated in the United Kingdom where beef sales dropped after it was revealed that BSE may cause disease in humans.

Essential components from JD control

1. Increase awareness of potential seriousness of situation. The problem is small at the moment but the rate of increase is exponential. It will never be cheaper to resolve the problem than at the present.
2. Integrated deer industry control programme based on "user pays".
3. Increased research and development effort to improve existing tests and develop new ones, to develop better vaccines, and to investigate other means of controlling infection and disease.

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References

De Lisle G W, Collins D M (1993). Johne's disease in farmed deer. *Surveillance 20*(1): 18-19

De Lisle G W, Wilson C A, Yates G F, Wards B J, Collins D M (1996). The post-mortem diagnosis of paratuberculosis in farmed deer. *Proc Fifth Int Colloq On Paratuberculosis, Madison, USA*. pp175-179

Fawcett A R, Goddard P J, McKelvey W A C, Buxton D, Reid H W, Greig A, Macdonald A J (1995) Johne's disease in a herd of farmed red deer. *Vet Rec 136* 165-169

Gumbrell R C (1986). Johne's disease in deer *Surveillance 13*(4). 15-16

Mackintosh C G, Webster J, Corson I, Masters B, Pearse A J, Littlejohn R (1997). An outbreak of avian tuberculosis in red deer. *Proc Deer Branch, NZVA, No 14* 243-250