Johne's disease and avian tuberculosis in deer in New Zealand

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Summary

Johne's Disease (JD) and to a lesser extent avian tuberculosis, are emerging as potentially serious problems on deer farms around the world. In addition to the loss of clinically affected animals, lesions due to *Mycobacterium paratuberculosis and M. avium* in mesenteric lymph nodes found at slaughter are a nuisance value to the venison industry due to their gross and histological similarity to lesions caused by *M. bovis*. Control of JD is difficult and relies on culling animals which are positive to serological testing and/or culture. However, none of the tests are sensitive enough to detect all subclinically infected animals. Vaccination has been used successfully in some countries to reduce the incidence of clinical disease. Avian tuberculosis is controlled by minimising contamination of feed and water by infected bird faeces.

Key words: Avian tuberculosis, deer, Johne's disease, *Mycobacterium avium, Mycobacterium paratuberculosis*

Introduction

JD is a chronic infectious granulomatous enteritis, which is usually characterised by diarrhoea, weight loss, emaciation and death. It is caused by *Mycobacterium paratuberculosis* and commonly affects sheep, cattle and goats world-wide. It has also been reported sporadically in a wide range of wild, park and captive ruminants including white-tailed deer, roe deer, red deer, fallow deer, sika deer, Tule elk, moose, aodad, mouflon, camel, bighorn sheep, reindeer, gnu, water buffalo, yak and llama. (Temple *et al.*, 1979; Jessup *et al.*, 1981; Pacetti *et al.*, 1994).

Since the mid 1980s JD has emerged as a potentially serious problem in red and fallow deer on farms in the United Kingdom (Gilmour, 1988; Fawcett *et al.*, 1995;), Germany (Commichau, 1982), New Zealand (Gumbrell, 1986; de Lisle and Collins, 1993; Mackintosh & de Lisle, 1998), Canada (Starke, 1991), Ireland (Power *et al.*, 1993), USA (Manning *et al.*, in press), Argentina (Mereb *et al.*, 1994) and France (Pingard A, pers. comm.).

Avian tuberculosis (ATb) is caused by members of the *Mycobacterium avium-intracellulare*-complex (MAIC). It is normally a disease of birds but it occasionally causes clinical disease in wild deer (Hopkinson & M^{c}Diarmid, 1964; Blaxter *et al.*, 1974) and has been reported in farmed deer in the UK (Blaxter *et al.*, 1974; Reid, 1994; Otler *et al.*, 1995) and New Zealand (de Lisle & Havill, 1985; Mackintosh *et al.*, 1997). Lesions in retropharyngeal, mesenteric and ileo-caecal lymph nodes due to MAIC organisms are commonly found in clinically normal farmed deer at slaughter in New Zealand (de Lisle & Havill, 1985; de Lisle *et al.*, 1995) and Ireland (Quigley *et al.*, 1997).

This paper summarises the situation in New Zealand relating to the incidences of JD and ATb, describes the clinical syndromes and presents information on the value of various tests used to diagnose clinical and subclinical JD and ATb.

JD Incidence

Since the first confirmed case of JD in 1986 the number of reported cases remained low until the early 1990s and then increased sharply. There were 4, 7, 22, 27, 46 and 40 newly infected herds from 1992 to 1997 respectively. The overwhelming majority of reported cases were from lesions in the viscera of clinically normal deer found at meat inspection. However, there have been a small number of clinical cases of JD and recently there have been a few outbreaks of clinical disease in yearling deer.

JD Clinical Syndrome

Typically affected animals fail to thrive, start to scour and develop obvious soiling with green faecal material around the tail, hind quarters and hocks, lose weight, fail to moult their winter coat in spring, become emaciated and die. Sporadic cases of JD occur in all ages and classes of deer, but there is an increasing incidence in young red deer 8-15 months of age. JD in older animals tends to run a chronic course of months, whereas JD in younger animals runs a more rapid course, which can be as short as two weeks from the first appearance of diarrhoea to death.

JD Post Mortem Lesions

Deer which die of JD typically have an emaciated carcass and have enlarged abscessed mesenteric and ileo-caecal lymph nodes. There may be oedema of the mesenterics and there may or may not be any gross thickening of the mid or terminal ileum and/or caecum.

JD Histopathology

Typically with JD in red deer there is extensive cellular infiltration of the intestinal mucosa with loss of normal villus structure and numerous acid fast organisms present within macrophages. There may be foci of calcification and caseation in some cases. Mesenteric lymph nodes show areas of invasion by macrophages and giant cells which usually contain numerous acid fast organisms. There are varying degrees of calcification, caseation and necrosis.

ATb

There have been a few sporadic cases of clinical disease in farmed deer due to ATb reported in New Zealand. (de Lisle & Havill, 1985). However, recently an outbreak of ATb in 7-8 month old red weaners was reported (Mackintosh *et al.*, 1997). Eighty weaners in an experiment to measure the effects of different diets, including silage with various grain and protein supplements, were kept in four indoor and four outdoor feedlot pens. Five animals, 3 indoors and 2 outdoors, developed severe chronic diarrhoea and weight-loss. They were euthanased and showed gross post mortem signs typical of JD, with enlarged abscessed mesenteric and ileo-caecal lymph nodes, oedematous mesenteries and variable degrees of thickening of the ileum. A PCR test for *M. paratuberculosis* was negative and a particular strain of *M. avium* was isolated from all 5 cases. When the remaining clinically normal animals were killed at 10 months of age, a further 35 had ATb abscesses, histologically indistinguishable from JD, in the mesenteric and/or ileo-caecal lymph nodes. The infections were probably acquired when the animals were brought indoors at weaning, exposed to feed and water contaminated by bird faeces and predisposed to infection by the stresses of weaning, transport and adaptation to new diets.

Diagnostic Tests

A small study was undertaken to assess the value of the complement fixation test (CFT), the agar-gel-immunodiffusion test (GD) and the absorbed ELISA test for diagnosing JD in red deer. These tests are commonly used in sheep and cattle in New Zealand but there has been little systematic study of their use in deer. Using serum samples from 9 confirmed cases of clinical JD in red deer and from 103 clinically normal deer from the infected farm which were sampled at slaughter, it was found that the most sensitive test for clinical JD was the GD (9/9), followed by the ELISA (4/9) and the CFT (2-9). Fourteen of the "normal" deer had JD lesions at slaughter and the GD and the ELISA both detected 5 of these, while there was one GD positive and 6 ELISA positives among the 89 gross and histologically negative deer.

In the ATb outbreak described above the GD and CFT were both positive 5/5. When the remaining deer were killed and the necropsy results compared with serology, the ELISA was the most sensitive (72.5%) but the least specific (62.2%) and the GD was 55-65% sensitive and 100-94.6% specific depending on whether the "pos" or the "pos" plus "weak pos" plus "suspicious" endpoints were used respectively. The CFT, using the cut off of $\geq^4/_8$ was 22.5% sensitive and 100% specific, and using $\geq^4/_4$ was 57.5% sensitive and 97.2% specific. These results highlight the problem of differentiating between disease caused by *M. paratuberculosis* and *M. avium* using serology.

Lymphocyte transformation (LT) test and skin tests using avian and/or Johne's PPD can be used to detect cell-mediated reactivity but the results are often difficult to interpret because these tests also do not discriminate between JD and ATb and there is widespread avian sensitisation amongst farmed deer in New Zealand. Research is currently being undertaken to identify antigens which might be specific for *M. paratuberculosis* and *M. avium*.

Lesions at Slaughter

Gross lesions especially in mesenteric lymph nodes, found at slaughter in deer are a continuing problem. (Campbell, 1995). Lesions due to *M. bovis*, *M. paratuberculosis* and *M. avium* are grossly and histopathologically indistinguishable. When detected at meat inspection, samples are taken for submission to the local diagnostic Laboratory and the carcass detained until diagnosis is confirmed. If the lesions are due to *M. bovis* and if the carcass is unaffected, then it can be passed for "local consumption" in New Zealand, but not exported. However, diagnosis of *M. avium* and *M. paratuberculosis* allow the export of venison from unaffected carcasses. The use of PCR tests (de Lisle *et al.*, 1996) has sped up the process of differentiating these three diseases, but culture is still regarded as the "gold standard" and can take up to 12 weeks, and some "sheep" strains of *M. paratuberculosis* may not grow at all. Thus from a meat industry standpoint these lesions are an expensive time-consuming nuisance. From the farmer's point of view it is crucial to determine the causative agent because the epidemiological factors are so different. The finding of bovine tuberculosis, JD or ATb have profoundly different implications, in terms of the status of the farm as well as control and prevention.

Epidemiology of JD and ATb in Deer

There is little known of the epidemiology of JD as it relates specifically to deer. It appears that they are susceptible to cattle and sheep strain of *M. paratuberculosis* (de Lisle & Collins, 1993) and it is presumed that the faecal-oral route of infection is the most common. One major difference between JD in deer and other domestic ruminants, is the relatively young age at which it has appeared. It is suspected that disease in 8 to 12 month old red deer is due to heavy challenge at an early age. One outbreak in this age group resulted in 35 of 300 clinically affected rising yearling animals. It is possible that stressors such as intercurrent disease and parasitic challenge may predispose to infection.

Control

The only effective means of eliminating JD on a deer farm is to destock it for at least 2 years and then restock it with "clean animals". However, this is usually not economically or practically feasible. Repeated "test-and-slaughter", although unlikely to eliminate JD because none of the tests are sensitive enough to detect all subclinically infected animals, is likely to reduce the JD incidence to a low level, if used in conjunction with regular culling of animals for poor condition or performance. Currently this is the most feasible option. Vaccination with live attenuated *M. paratuberculosis* vaccines has been used successfully overseas to control JD and reduce the incidence to a low level, but it does not prevent subclinical infection (Fawcett *et al.*, 1995). This strategy is unlikely to be viable in New Zealand because the currently available vaccine is not licensed for use in deer, it causes cross-reactivity with the bovine Tb skin tests and the lesions at the injection site and the draining lymph nodes may render the carcasses ineligible for export, thereby halving their value. Control of ATb is by reducing faecal contamination of food and water, especially for deer being housed, and to minimise stress by good management practices.

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