Summary of a Johne's disease session: NADVet Conference, Austin, Texas, 19-20 Feb, 2002

C Mackintosh

Abstract

Becky Manning, from the University of Wisconsin, presented a paper on the occurrence of Johne's disease (JD) in North American.

JD has been diagnosed in cattle, sheep, goats, deer and bison in USA. It has been identified in wild Tule elk in California and appears to cause long-standing infections and minimal mortality. JD occurs in a range of captive cervid species in many zoos throughout USA, where it is expensive and causes major problems. JD has caused disastrous outbreaks in a large trophy hunting elk herd and in a herd of fallow deer.

Manning and colleagues have developed an ELISA test for serological diagnosis of JD in cattle, and it uses a Protein G conjugate, which binds to cattle IgG1 and IgG2. They have tested it in a range of species and found that the binding in white-tailed deer is comparable to cattle, poorer in elk and red deer and very poor in muntjac. They believe that it is useful for screening deer herds, but not for diagnosing individuals.

Colin Mackintosh, from AgResearch Invermay, reviewed the occurrence, aetiology, clinical signs, pathology and diagnosis of JD in deer in New Zealand. The paper then briefly discussed possible prevention and control options.

Peter Wilson, from Massey University, presented a paper on JD from the perspective of individual deer farmers and from the deer industry. Individual farmers may or may not view JD as a serious issue, depending on a range of circumstances. The deer industry is concerned about JD and the potential for it to adversely affect overseas markets. The industry is faced with a number of options including: do nothing, attempt eradication or introduce a Market Assurance Programme. The pros and cons of these options were discussed. It was concluded that "there is no better time to implement a management programme than while the herd prevalence of the disease is low".

Diagnosis of Johne's disease in North America deer

Becky Manning

Introduction

JD has been diagnosed in cattle, sheep, goats, deer and bison in USA. As well as being found in farmed elk, it has been identified in wild Tule elk in California and appears to cause long-standing infections and minimal mortality. Also found in captive cervids in many zoos throughout USA, including the following deer species: axis, barasingha, hog, sambar, mule, fallow, white-tailed deer (WTD) and *Cervus elaphus*. In zoos it is expensive and causes major problems because it halts exchanges, resulting in inbreeding and lost genetics, and causes bad publicity.

JD has caused a disastrous outbreak in a large trophy hunting elk herd. Initially it caused widespread skin-test reactivity, and the animals were cleared negative for tuberculosis with a CCT. The following year there were over 50 confirmed cases of clinical JD, especially deaths in yearlings. The operation is now out of business.

It caused an outbreak of clinical JD in a herd of fallow deer. Some animals were shedding *M. paratuberculosis* by 6 mo, and 51/54 animals faecal sampled were culture positive. Samples of pasture, soil and pond water were taken from the farm for culture and 26/36 were positive.

Risk factors

Investigations of these outbreaks indicated the following risk factors for the introduction of JD onto deer farms:

Purchasing deer from untested/unknown sources

- Cross-grazing with cattle, sheep or goats infected with JD
- Using unpasteurised milk for hand-rearing fawns

Diagnosis

The diagnosis of JD is currently limited to the following:

- Histopathology and culture of material from necropsies.
- Faecal culture and serology from live animals.

Serology: Their laboratory has developed an ELISA for serological diagnosis for JD in cattle using a Protein G conjugate, which binds to cattle IgG1 and IgG2. In order to see if this ELISA could be adapted for use in deer, they have tested it in a range of species and found that the binding in WTD is comparable to cattle, poorer in elk and red deer and very poor in muntjac. They believe that it is useful for screening deer herds, but not for testing individuals.

Paratuberculosis (JD) in deer

C Mackintosh

Introduction

Since the mid 1980s paratuberculosis or Johne's disease (JD) has emerged as a problem on deer farms in the United Kingdom (Gilmour, 1988; Fawcett et al., 1995;), Germany (Commichau, 1982), New Zealand (Gumbrell, 1986; de Lisle and Collins, 1993; Mackintosh and de Lisle, 1998), Canada (Starke, 1991), Ireland (Power et al., 1993), USA (Manning et al., 1998), Argentina (Mereb et al., 1994) and France (Pingard A, pers. comm.). In New Zealand the first confirmed case of JD in deer was reported in the mid-80s. Passive surveillance, principally by the examination of suspect 'tuberculous' lesions identified in deer slaughter plants has resulted in *Mycobacterium avium* subsp. *paratuberculosis* (*M. ptb*) being identified in over 600 farmed deer on 300 properties (de Lisle, pers. comm.). The herd prevalence based on this information is ~6% (300/~5000), but the true prevalence of infection amongst New Zealand's 2 million deer is expected to be higher than this figure.

Aetiology and epidemiology

Cattle are generally only infected with "bovine" strains of *M. ptb*, while sheep are generally only infected with "ovine" strains. Deer appear to be susceptible to both strains (de Lisle and Collins, 1993).

M. ptb may be introduced to deer farms by faecal contamination from infected sheep, cattle or deer. The finding of *M. ptb* in rabbits in the United Kingdom and some wild animals in Australia indicates that wildlife may also be a source of infection for farmed deer. Many deer farms are established by deer-fencing areas of existing sheep farms or cattle farms. The use of sheep and cattle on deer farms to assist with pasture management and weed control may also introduce infection. Some local environmental spread from runoff is also theoretically possible. *M ptb* organisms are thought to persist in the environment for long periods, although the number of viable organisms probably declines exponentially, so the greatest risk is in the first 3 to 6 months.

Two clinical syndromes have been recognised in farmed red deer; a) sporadic cases in mixed age deer, with an incidence of 1 – 3% per annum, and b) outbreaks in 8 – 15 month old deer, that may involve up to 20% of a group.

The exact risk factors for the development of clinical JD in deer are not known, but it is likely that stress plays a major role in exacerbating the disease. Sheep and cattle generally do not

develop clinical disease until they are 2 - 4 years old, although under experimental conditions young lambs (<14 days old) exposed to very heavy challenges (>10^9 colony forming units) develop severe disease in under 6 months. It is assumed that animals exposed to early heavy challenge are more likely to develop clinical disease. Older animals appear to become more resistant to infection and are less likely to develop disease. The development of clinical signs of JD in deer as young as 8 months of age suggests an early heavy challenge. Genetic susceptibility to JD plays a role in dairy cattle (Koets et al., 1999) and it is likely to be important in sheep and deer as well. The relative susceptibility of deer to JD, compared with sheep and cattle is not known. The level of faecal shedding, intrauterine infection, systemic infection of the udder or external contamination of the hind's udder, have not been established for deer.

Clinical signs

Sporadic cases of clinical JD, characterised by chronic granulomatous enteritis, occur in all ages and classes of farmed deer. Affected animals typically lose weight over a period of a few months, and the majority develop diarrhoea. There is usually low morbidity (<1%) but high mortality (~100%) with little or no response to symptomatic treatment, which is very similar to paratuberculosis in cattle and sheep.

Outbreaks of JD in young red deer, involving 5 - 20 % of the 8 – 15 month-old animals, have occurred on 5 to 10 farms a year for the last 6 years in New Zealand. The affected animals initially "fail to thrive", stop growing and then rapidly start to lose weight and condition. They invariably develop diarrhoea and became soiled with green faecal material around the tail, hindquarters and hocks. In spring they only partially moult their winter coats and take on a patchy or "moth-eaten" appearance. The clinical disease has a course of a few weeks, and it appears that the younger the animal, the quicker the progression to emaciation and death. The differential diagnosis includes yersiniosis (in weaners in winter), abomasal parasitism, avian tuberculosis and chronic malignant catarrhal fever (Mackintosh, 1998).

The prevalence of subclinical *M. ptb* infections in farmed deer is not known, but up to 10% of animals in some lines of apparently normal deer from infected properties have had lesions in mesenteric lymph nodes at slaughter.

Pathology

Necropsy examination of paratuberculosis cases typically reveals enlarged jejunal and ileo-caecal lymph nodes, often with white or cream caseous lesions. Unlike sheep and cattle, there may not be any gross thickening of the terminal ileum, but there are often prominent lymphatic drainage vessels from the jejunum to the adjacent lymph nodes. There is usually no fat in the omentum, which is often oedematous and may be adherent to the affected jejunum and lymph nodes in severe cases. Histopathological examination of lesions typically reveals extensive areas of invasion of affected lymph nodes by macrophages, often with caseation with foci of calcification and numerous small acid-fast organisms present in the macrophages. The ileo-caecal valve may show loss of villus structure with mixed cellular infiltrate and contain numerous acid-fast organisms, but it appears that lesions are also commonly found further up the ileum and/or jejunum.

Subclinical paratuberculosis infection in deer is often detected at slaughter and typically a single tuberculoid lesion is detected in the jejunal lymph node without any macroscopic evidence of enteric lesions. Occasionally there is a generalised lymphadenitis with normal lymph node constituents replaced by bizarre giant cells. This condition is sometimes misdiagnosed as a lymphoid neoplasm (H. Montgomery, pers. comm.).

Diagnosis

A trial conducted in New Zealand showed that, of the tests available, the most sensitive and specific serological test for confirming a diagnosis of clinical JD in red deer is the agar gel immuno-diffusion (AGID or GD) test (Mackintosh et al, 1999). However, none of the

currently available serological or cell-mediated immunological tests is sufficiently sensitive or specific for detecting subclinical *M. ptb* infections in deer to be useful for control of JD on infected properties. Serological tests suffer from lack of sensitivity because antibody is only produced late in the disease. Cell-mediated tests, such as the intra-dermal skin test, the lymphocyte transformation test, suffer from poor specificity because of widespread sensitisation of deer exposed to closely related *M. avium* complex organisms and none of the currently available antigens are specific enough to differentiate between them. Faecal smears, culture or PCR may be used to detect infected individuals. Bulk faecal cultures, whereby faecal pellets from 50 – 100 animals are blended together and cultured, have been used successfully to detect infected sheep flocks in Australia (Whittington et al, 1999). If this technique is shown to be as sensitive for deer as sheep, it could be used to screen deer herds for infection in a cost-effective manner.

At commercial deer slaughter plants in New Zealand, the discovery of lesions in the mesenteric or ileocaecal lymph nodes causes considerable problems because of the similarity between lesions due to *M. bovis*, *M. avium* and *M. ptb* when examined grossly and histopathologically (Campbell, 1995). 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 a PCR test has markedly sped up this process (de Lisle et al., 1996). In addition, the PCR test can equally well detect the bovine and ovine strains of *M. ptb*. However, culture remains the "gold standard" for differentiating *M. bovis*, *M. avium* and *M. ptb* because it is 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. The introduction of the liquid culture-based BACTEC system has increased the sensitivity and also sped up the process of culturing mycobacteria from slaughter plant material.

Prevention

If JD has never been diagnosed in a deer herd and the farmer has no reason to suspect that his herd is infected, it would be wise to take all sensible precautions to prevent its introduction. This means:

Keep a closed herd, avoid buying in animals and use AI to bring in new blood lines.

Only purchase animals from "low risk" herds. A market assurance programme, which screens herds for infection, would provide a mechanism for assessing risk and provide a premium for replacements from low risk herds.

Avoid grazing sheep or cattle on the deer farm unless they are known to come from flocks or herds that are low risk.

Control

Control options on infected deer farms are currently limited to either culling affected stock, culling test-positive animals, changing from a breeding operation to a weaner fattening or velvet operation or depopulation and restocking after two years. All these options should be subjected to a rigorous cost/benefit analysis to determine the most economic and practical alternative. It is hoped that vaccination may provide an alternative means of control if a vaccine can be developed that a) is efficacious, b) does not interfere with the national Tb control programme and c) does not cause carcass blemishes or downgrading. A live attenuated oil-adjuvanted vaccine has been used successfully to control JD in deer in the United Kingdom (Fawcett et al., 1995), but recent trials in New Zealand suggest that oil-adjuvanted vaccines are unlikely to be acceptable due to interference with Tb testing (Mackintosh et al, 2001).

NOTE: *This paper is a summary of the paper in the proceedings of the World Deer Farmer's and NADeFA Conference Feb. 2002. The references are available in that proceedings*