Animal Health/ Diseases

Deer Diseases - Otago and Southland

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(From cases submitted to Invermay Animal Health Laboratory 1994-1997)

The total number of deer cases submitted to the Invermay Animal Health Laboratory has increased steadily over the past few years from 678 in 1994 to 938 in 1997. Laboratory data can often give a rough picture of the relative prevalence of the various diseases seen in a region. Approximately 30% of the cases have been submitted from deer slaughter plants (DSP's) - usually as suspect tuberculosis.

Diseases caused by Mycobacteria

The majority of these are submitted by DSPs. The number of *Mycobacterium bovis* cases (diagnosed by histology/culture) was 44 in 1994 and 53 last year. In 1995 the numbers rose for some reason to 82.

M avium (diagnosed by culture) prevalence has always been low. Eleven cases were recorded in 1994 and about the same number in1997. Infected animals invariably show enlarged ileo-jejunal nodes but some cases can mimic *M bovis* cases with involvement of the retropharyngeal nodes.

The number identified as *M paratuberculosis* has shown a rise from 12 in 1994 to 22 last year. These cases invariably show at meat inspection with enlarged ileo-jejunal nodes which, on histopathological examination, are packed with acid fast bacilli but sections of ileum, when taken, are normal.

The occasional *M. paratuberculosis* case in adult deer, possibly more in the last few years has been diagnosed by practitioners, with clinical signs similar to those seen in cattle - wasting and diarrhoea. Alarmingly this disease appears to have entered a new phase in the last few months where we have seen at least three outbreaks of acute Johne's disease in yearling deer. Affected deer develop severe diarrhoea and wasting followed in a few weeks by death. A high percentage of deer in the affected mob show signs and a large tail end of scouring yearlings develops.

As *M. paratuberculosis* can be cultured from these deer and there is Johne's in sheep on these farms a sheep strain of *M. paratuberculosis* is probably the cause.

Why this condition has suddenly been seen only in the last few months and why large numbers of weaners have been affected is unknown. Presumably these young deer have been infected in the first few weeks of life - the incubation period is thus very short compared with the disease in other species.

Deer have been farmed for a large number of years now and outbreaks of clinical Johne's in yearlings have not been seen before until last year.

Malignant catarrhal fever

There has been a slight rise in the number of cases diagnosed at this laboratory from 9 cases in 1994 to 15 cases in 1977.

The disease is seen in the winter and affected animals are usually found dead with minimal gross lesions. Fixed brain remains the best tissue for diagnosis, even in moderately decomposed animals. The use of PCR on blood from live suspect cases is not advised.

There appears to be a species susceptibility with Pere David's deer being the most susceptible, then red deer and the least susceptible are wapiti.

Very occasionally a clinical syndrome of chronic MCF can be seen in individual animals as a progressive weight loss culminating in death after a few weeks.

Yersinia pseudotuberculosis

In spite of vaccination we have seen a steady increase in the numbers of outbreaks of yersiniosis in weaners over the winter. In 1994 eight outbreaks were seen and there were 27 in 1997. The general impression is that the numbers dying in these outbreaks are much less than pre-vaccination outbreaks. Deaths do occur in vaccinated groups and are probably due to a severe challenge or, less often, faulty technique. AgResearch checked 20 isolates for us last year and all were vaccine strains of *Y. pseudotuberculosis*. The risk factors associated with outbreaks of this disease are large mobs, nutritional stress and bad weather conditions.

The diagnosis can be simply made by identifying the organism in faeces or intestinal contents from affected animals. Histopathological examination of fixed intestine from very recently dead animals can also help but most veterinarians rely on culture for diagnosis.

Cryptosporidiosis

This is probably a major cause of deaths in fawns in this region. Although the number of cases diagnosed remain low (six outbreaks in 1994, four in 1997) the numbers of fawns dying in each outbreak can be very high, up to 30% of a mob.

The risk factors for developing the disease appear to be poor weather, stress on the hinds and the presence of ponds or wallows in the calving paddock. Affected fawns are usually found dead showing signs of severe dehydration. Often there is clotted milk in the stomach and colostrum-like fluid distending the intestine/caecum/colon. This usually has a very offensive odour.

A ZN stained smear of rectal contents is usually the easiest method of making a diagnosis, although multiple sections of fixed intestine from moribund calves can be useful.

Trace element deficiencies

Copper

Deer appear to be the most susceptible of the farmed species to copper deficiency, with osteochondrosis seen in deficient weaners and enzootic ataxia in adult animals. Weight gains and suspected improvement in velvet quality have also been reported in deficient animals given copper.

Clinical signs are seen in deer with liver copper concentrations of <50 μmol/kg and serum copper of <3-4 μmol/l.

In 1995 85% of the liver copper cases requested through the Optigrow programme through meat plants had one or more livers with a liver copper concentration of <100 μmol/kg and there has been a steady rise in cases of deficiency diagnosed at this laboratory - 33 in 1997.

Selenium

Confirmed cases of selenium deficiency are rare although there have been anecdotal reports of unthrifty young deer with low GSHPx concentrations of <1-2 KU/l responding to selenium supplementation.

The very occasional case of white muscle disease in young 4-12 week deer from Central Otago has been seen. These small outbreaks have been associated with wet summers, good grass growth and a lack of selenium supplementation of the hinds.

Affected calves show depression and stiffness. Pale streaking of the skeletal muscles and heart similar to that seen in lambs with WMD, can be seen at necropsy.

In affected fawns liver selenium concentrations range from as low as 73 nmol/kg to 300 nmol/kg and blood selenium from 46-74 nmol/l (GSPHx concentrations can be undetectable).

The diagnosis can be confirmed by histopathological examination of affected muscle.

Miscellaneous diseases

Clostridial disease

Clostridial disease is relatively rare, perhaps because of vaccination? Only a few small outbreaks have been seen per year (although in one outbreak 7/120 deer died).

Deaths are associated with yarding and fighting. *Cl. novyi* and *Cl septicum* are most commonly involved.

Dictyocaulus viviparus

Overall control of this parasite is good in this region but there is the occasional outbreak of deaths caused by lungworm (as late as May) in weaned deer, often associated with reliance on intermittent drenching with white drenches. In one outbreak a white drench had been given only 3 weeks before the deaths began.

Affected deer are found dead or are seen barely able to stand with a severe dyspnoea. A necropsy reveals a markedly emphysematous lungs with immature lungworm packing the smaller airways in all areas of both lungs.

It is not known wether a cattle or deer strain of lungworm is involved.

Fading elk syndrome

This condition is characterised by a marked, sometimes irreversible loss of condition in individual elk in late winter- early spring. Low albumin concentrations (20-25 g/l) are identified as the only biochemical abnormality in the serum of affected deer. Total protein concentrations are normal, ruling out a protein losing enteropathy.

Although abomasal parasitism has been reported as a cause. Fading elk syndrome often occurs on deer farms with good parasite control and in these cases appears to be associated with poor pasture length and a lack of dietary roughage.

Because affected animals are valuable and often recover, farmers are reluctant to sacrifice them for necropsy. With improved management and parasite control, samples from "fading elk" are seldom submitted to the laboratory nowadays.

Stillbirths

Stillbirths may be relatively common. Necropsy shows a full term, often moderately decomposed calf which has not breathed with no gross pathological changes.

The most likely cause is management factors with stocking density too high in the calving paddock.

Duodenal ulceration

This is seen only occasionally nowadays. With better management and feeding deer are less likely to be under stress. In this condition stress, possibly over some time, creates an ulcer which perforates the duodenum a few centimetres from the pylorus. Peritonitis and death follows.

Porphyria

There have been at least two cases where "brown bones" were found in clinically normal deer at slaughter. The entire skeletal system was involved and affected bones fluoresced red under UV light. A possibly genetic disorder of heme metabolism is suggested as the cause. It has been recorded in other farm animals.

Brucella ovis

How this disease was transmitted from rams to stags is anyone's guess.

It has been identified serologically on two farms in Otago and on one farm there was a possible association with a low calving percentage.

A survey of stags at deer slaughter plants in this area is presently underway. So far *Br. ovis* affected stags have not been identified at DSPs in this region.