CRYPTOSPORIDIA AND OTHER PROTOZOA IN DEER

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INTRODUCTION

We know little about protozoa in deer. In most stock the protozoa discussed cause asymptomatic infections. Occasionally, cases of clinical disease occur, but these are more the exception than the rule. In the small amount of literature available, clinical disease appears to occur only when other contributory factors are present; or when there may be particular host susceptibility.

In this paper the information has been set out in a similar way to The Veterinary Handbook[13] to try to make it more useful. Much of the content is speculative, but this speculation is unavoidable at this stage.

CRYPTOSPORIDIUM

Cryptosporidium is a very small coccidial organism with no host specificity, which parasitises the epithelium of the villi of the small and large intestine. It can produce an acute diarrhoea of varying severity, usually in immunodeficient animals.

Hosts: All species are probably susceptible.

Distribution: Probably New Zealand wide, but unknown.

Occurrence: Unknown.

Herd incidence: Probably up to 100%.

Aetiology: Cryptosporidium sp.

Mode of infection: Ingestion of oocysts.

Incubation period: 2 to 3 days.

Age susceptibility: Probably all ages, but particularly neonates.

Clinical signs: Acute diarrhoea in severe cases[16,21].

Pathology: All stages of the parasite in the epithelial cells of the large intestine and small intestinal villi. As they are just under the cell wall they may appear to be attached to the outside of the epithelial cells.

Diagnostic aids: Oocysts, 4-6 microns long, in faeces. Histopathology of small intestine.

Laboratory specimens: Faeces, fixed small and large intestine.

Differential diagnosis: Other causes of diarrhoea.

Prognosis: Good in immunocompetent animals, poor in those that are immunodeficient.

Treatment: None.

Control: Isolate clinically affected animals. Decontamination is difficult as Cryptosporidium is resistant to most commonly used disinfectants. 10% formalin is said to be effective if used after thorough cleaning.

Prophylaxis: Ensure that hand reared animals have received colostrum. Avoid stress as much as possible.

Public health: This organism is zoonotic and can be contracted from the faeces of infected animals. Human infections may be asymptomatic or may feature abdominal pain and watery diarrhoea. Can be a severe disease in immunologically compromised humans.

Comment: Three cases of Cryptosporidium associated with disease in deer have been described and each has involved housed, artificially reared, red deer calves[16,21]. One case[21] was in Scotland, the other two were in Otago, New Zealand[16].

In the Scottish case 82 red deer calves up to one week of age were captured and brought to the research station. Over 4 weeks 56 calves developed diarrhoea that lasted 2 to 14 days and 20 animals died. Cryptosporidium oocysts were observed in 27 of 34 faecal smears from calves with diarrhoea and 11 of 22 smears from apparently healthy animals.

In a necropsied calf the small intestine was slightly congested and the mesenteric lymph nodes were enlarged. Both small and large intestines were infected with cryptosporidia. They were most numerous in the caecum and colon, with smaller numbers in the jejunum and upper ileum. Very few organisms were seen in the terminal ileum. Atrophy was almost total in the villi of the ileum and there were focal areas of villous atrophy in the villi of the upper jejunum.

Mice inoculated with faeces containing cryptosporidia excreted oocysts 7 to 10 days later. Sera collected from deer 2 weeks after the outbreak of diarrhoea contained antibody to bovine Cryptosporidium.

Not all the infected deer developed diarrhoea, suggesting that contributing factors (e.g. inadequate passive immunity) may play a significant role in precipitating diarrhoea.

The two Otago cases were similar. The first involved about 10 abandoned calves of recently captured hinds that were being hand reared indoors. The second involved a group of 10 farm bred calves that were picked up at two days old and hand reared indoors. In both cases the calves developed a pasty yellow scour and mortality was high - 100% and 70%

respectively - and occurred within a few days of onset of the scour; despite treatment with electrolytes, antibiotics and kaolin in the second case. In neither case was there a loss of appetite.

One calf from the first case and five calves from the second were necropsied and showed patchy reddening of the small intestine mucosa and oedematous mesenteric lymph nodes. Histologically there was a moderately severe to severe acute enteritis of both the small and large intestines. The villi were stunted, fused and swollen and there was infiltration of the lamina propria by macrophages and neutrophils. Cryptosporidia, 4 to 6 u in diameter, were seen primarily in the small intestine in the first case, and the large intestine in the second case. In neither case were any significant bacterial or viral pathogens cultured.

Following the second case, the pens were cleaned thoroughly and five more deer calves were brought in, but they were given 30 ml bovine colostrum and 20 ml adult deer serum with each feed for 3 weeks. These calves showed no sign of illness at any stage.

In each of these cases no cause-and-effect relationship was established between diarrhoea and the Cryptosporidium organism, but in the absence of any other causative factor the authors consider that Cryptosporidium played a significant role.

COCCIDIA

Infection by host specific Sporozoa of the genus Eimeria. No clinical cases have been reported from deer in New Zealand. As in other animals, infections are generally asymptomatic either because the species of coccidia are not normally pathogenic or because initial infections are light and allow development of immunity without disease. Can occur concurrently with other enteric disease.

Hosts: Eimeria oocysts have been reported from red and fallow deer in New Zealand[15], but not from other species.

Distribution: Probably New Zealand wide in red deer. Low numbers of oocysts were recovered from 27 of 108 red deer farms surveyed in 1981[15]. Distribution for other deer species unknown.

Occurrence: Probably common at low levels in red deer. Unknown for other deer species.

Herd incidence: Probably up to 100% in red deer. Unknown for other deer species.

Aetiology: Eimeria spp.

Mode of infection: Ingestion of sporulated oocysts.

Incubation period: 1 to 3 weeks.

Age susceptibility: Probably more common in young animals. Clinical disease would probably occur mainly in young stock.

Clinical signs: Bloody diarrhoea and elevated temperatures have been reported for mule deer[1], none for other deer species.

Pathology: None described for deer.

Diagnostic aids: Microscopic demonstration of oocysts in faeces, or schizonts or oocysts in intestinal scrapings or sections.

Laboratory specimens: Fresh faeces, fixed intestine.

Differential diagnosis: Bacterial or viral enteritis. Helminthiasis.

Prognosis: Probably good. Should resolve in 1 to 3 weeks without treatment. When severe, treatment may be needed.

Treatment: Sulphonamides or specific anticoccidial drugs should work.

Control: If necessary remove animals from contaminated environment; avoid moist conditions, overcrowding and stress.

Prophylaxis: Avoid overcrowding of young animals under warm moist conditions.

Comments: Overseas, Eimeria species have been reported from white-tailed deer, fallow deer and elk[5,17,18,20]. In all these reports there was no association with clinical disease. There is one report of clinical disease developing in mule deer fawns 8 to 9 days after being given sporulated oocysts of E. mccordocki[1]. The fawns exhibited elevated temperatures and bloody diarrhoea, but it was also reported that the fawns were in poor condition when they were obtained. It would seem therefore, that coccidia have the potential to cause disease in deer, but generally only when the animals are under stress from some other cause.

TOXOPLASMA

A widespread, most frequently asymptomatic, infection with the zoonotic protozoan Toxoplaszma gondii. Little work has been done with this organism in deer, but the most common clinical manifestations found in other stock are placentitis and abortion.

Hosts: Has been reported from feral red deer from the Rotorua area[2]. All species are probably susceptible.

Distribution: Probably New Zealand wide, but unknown.

Occurrence: Unknown, but probably common as asymptomatic infection.

Herd incidence: Unknown.

Aetiology: Toxoplasma gondii.

Mode of infection: Ingestion of sporulated oocysts.

Incubation period: Unknown.

Age susceptibility: Probably mainly younger animals.

Clinical signs: Abortion?

Pathology: Insufficiently studied. Severe necrotizing and haemorrhagic enteritis, and mesenteric lymphadenitis in mule deer[11].

Diagnostic aids: Histological demonstration of lesions and Toxoplasma cysts. Transmission to mice. Serological tests (see comments).

Laboratory specimens: Fixed tissues from major organs. Foetus and placenta?

Differential diagnosis: Other causes of abortion? Other causes of enteritis and lymphadenitis.

Prognosis: May differ with species. Probably good for red deer. May be poor for white-tailed deer (see comments).

Treatment: None.

Control: None practicable.

Prophylaxis: Epidemiology and/or significance in farmed deer unknown, so no recommendations can be made.

Comment: Two experiments have been described in which elk and mule deer were given the same dose of sporulated oocysts[10,11]. A pregnant elk, her foetus and a calf all became infected with Toxoplasma, but no clinical signs were exhibited. Despite this the Toxoplasma remained virulent. The mule deer (Odocoileus hermionus) developed acute toxoplasmosis and died. This may indicate a difference in host susceptibility, or possibly that mule deer are more easily stressed than elk. If the former, then the related white-tailed deer (O. virginianus) may be more susceptible than red deer.

It is not known what role Toxoplasma may play in abortion in the various deer species - more work is required in this area.

Serological tests are a problem. The dye test works well with elk but there are problems with IHA[10]. Animal Health Laboratories currently use IFA or agglutination tests for detection of Toxoplasma in sheep, but these have not been evaluated for deer.

SARCOCYSTIS

Sarcocystis spp. are protozoan parasites with a two-host life cycle. typically the coccidia-like sexual stage occurs in the intestine of a carnivore and the cystic stage occurs in the muscles of a herbivore. Species are usually host specific, but taxonomy is something of a nightmare. Infections are usually asymptomatic but clinical conditions do sometimes occur in other domestic animals.

Hosts: Sarcoystis sp has been recovered from 25 of 75 feral red deer from the Rotorua area[3,4]. It is not known if species infecting other deer are in the country.

Distribution: Unknown, but will probably eventually be throughout the country.

Occurrence: Unknown.

Herd incidence: Unknown.

Aetiology: Sarcocystis spp.

Mode of infection: Ingestion of sporocysts shed in faeces of carnivore. A red deer-dog cycle has been demonstrated in N.Z.[2].

Incubation period: Unknown.

Age susceptibility: Probably all ages, but unknown.

Clinical signs: Unknown. Abortion?

Pathology: Unknown.

Diagnostic aids: Histopathology. Peptic digestion of muscle. Transmission studies.

Laboratory specimens: Fixed muscle.

Differential diagnosis: Other causes of acute febrile illness, abortion and myositis.

Prognosis: Probably good.

Treatment: Supportive therapy if severe case should develop, otherwise none.

Control: Not practicable.

Comment: Overseas, Sarcocystis has been reported from elk in North America[8,14,19,20], red deer in Europe[19] and white-tailed deer[6,7,9]. Prevalence in elk and white-tailed deer is reported to be high in some areas. No clinical signs described. Sarcocystis sp from elk have been shown to have only low infectivity for cattle[12].

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