A CRYPTOSPORIDIOSIS OUTBREAK IN FAWNS

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In an outbreak of cryptosporidiosis on a Southland deer farm during December 1993, 10 out of 73 fawns died. Clinical aspects of this case are described and discussed in relation to cryptosporidial epidemiology. The apparently successful treatment of sick fawns with toltrazuril is also discussed.

Cryptosporidiosis was first diagnosed in Western Southland in a batch of dairy calves brought down from the North Island in spring 1992 The farmer concerned was an inexperienced stockman and about 40 out of 240 calves died despite electrolyte treatment. Concurrently there were deaths in lambs at pasture on a neighbouring farm.

In September 1993 we saw many outbreaks in dairy calves and in most a traceback to the original farm could be made One farmer treated 90 of 110 calves with 850 L electrolyte (and diligent nursing), and lost only 5.

By contrast, intensive treatment of fawns* is not practicable and for this reason cryptosporidiosis is an even greater threat to the deer industry Soon after we had witnessed these outbreaks in dairy calves there were two confirmed outbreaks on deer farms

This report details one of those outbreaks on a 23 ha smallholding near Otautau This is something of a model farm: tidy, with good fencing and shelter The clay soil drains poorly and is prone to pugging in winter The farmer is a capable and caring stockman Hinds are wintered on autumn-saved ryegrass pasture with no supplementary feed. By fawning time they are in good (but not fat) condition

History

Seventy nine hinds, 40 stags and 60 weaners were carried through to spring 1993. In November, 3 weeks before fawning started, two groups of first fawners were set-stocked on to 7 5 cm of lush grass. There was subsequently very little problem in these hinds The mixed age hinds were likewise set stocked on two paddocks of 3 5 ha, each containing 23 and 24 hinds, 1 week before they started fawning These paddocks are adjacent to a golf course and fawning tends to be concentrated at the ends where there is least disturbance and most shelter - in one paddock a gully which becomes muddy in wet weather The weather over fawning was cold and wet

In spring 1991 several fawns died on this farm Extensive sampling revealed K99 *Escherichia coli* as the likely cause, and the scours and deaths ceased following injection of all sick and at-risk fawns with a long-acting oxytetracycline formulation.

* It is the author's preference to use the term "fawns" to distinguish them from (bovine) calves

Clinical investigation, initial treatment and response

I was called in on 4th December when a fawn in the gully mob was found dead At post-mortem the fawn had a full stomach of milk and there was no scour Lesions were confined to the ileum which had mucosal haemorrhages and pasty grey contents (I have observed this grey colouration in two fawns subsequently shown to have had cryptosporidiosis - counter to the yellow or greenish custardy contents usually reported (1) (10) Perhaps this is due to mud ingestion by dehydrated animals.) The farmer was reluctant to pay for a full range of tests so only ileal contents (some fresh and a swab in transport medium) were sent to Invermay Animal Health Laboratory.

Whilst awaiting results several other fawns looked sick, so the whole mob was injected with oxytetracycline* on the premise that this was a revisitation of the 1991 problem. Despite the injections in this and the other mixed age hind mob, where fawns were also dying, the losses continued.

Results

From the swab a heavy growth of K99 *E. coli* was grown Also present were "significant numbers of cryptosporidia oocysts"

Clinical signs

In this outbreak the fawns were affected at 10 - 14 days old At this stage fawns should either be planted, playing or running behind their mothers Sick fawns were often observed wandering behind their peers (? gut pain) Frequently the tail was up, and the fawn appeared slightly pot-bellied, though there sometimes was no scour visible There was a rapid loss of condition over 24 hrs Some deaths occurred without scouring - but often there was an explosive white/yellow diarrhoea Dehydration, recumbency and death ensued without treatment

Treatment with toltrazuril[3]

Over a period of 10 days 9 fawns died and many others were sick. It was decided to try drenching the sick fawns with the anticoccidial drug toltrazuril,† available as a 2 5% water-miscible solution (2). This product is not licenced for use in deer, but overseas literature suggests that a single dose is an effective treatment for Coccidiosis in calves (3), goats (4) and lambs (5) and that the effect is independent of the level of infection (2)

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* "Oxytetrin LA" Pitman-Moore

† "Baycox" Bayer

At this stage we were unaware of this literature which recommends doses of between 10 and 20 mg/Kg L W , with better results at the higher rate. We used 16 ml (diluted in an equal volume of water) by mouth, which for a 10 Kg fawn equates to 40 mg/Kg of toltrazuril. The water was added solely because of a warning of irritancy to mucous membranes.

I have not been able to find any reference supporting toltrazuril's efficacy against cryptosporidiosis. There is an unsupported observation of lack of efficacy (2)

Of the remaining 38 fawns in the two affected mobs some 18 sick fawns were drenched with 32 ml of this 1.25% toltrazuril solution by the farmer over a 2 week period Six of these were recumbent and unable to walk The rest were caught and treated as soon as they were observed to be ill. In most cases only a single dose was used, but occasionally it was repeated several days later. Despite the severity of the signs, only 1 fawn died after toltrazuril treatment The recovered fawns seem to have suffered no long term effects

[The response to toltrazuril on another farm with fawns dying with a mixed cryptosporidiosis/rotavirus infection was less spectacular, 9 out of 18 fawns treated survived. The total loss in this outbreak was 47 out of 150 fawns]

Discussion

Cryptosporidium parvum is an organism that has the ability to infect many species (6), and has a low minimum infective dose (2) (7). Although the persistence of oocysts in the environment is unknown, they appear to survive dispersal but are susceptible to temperatures below 0 deg.C and dessication (7). The oocysts are infective when passed and the life cycle is very short (8). It has recently been demonstrated that healthy beef cows whose calves had had cryptosporidial diarrhoea continued to excrete oocysts in their faeces for 18 months (the duration of the trial) at a rate calculated to yield between 750,000 and 720 million per cow daily (9) The long term carrier status of hinds is a matter of surmise, but asymptomatic hinds may excrete oocysts around the time of parturition (6)

Bearing in mind these characteristics, it could be envisaged that the organism, following a large number of outbreaks of cryptosporidiosis in calves in September and October 1993, has become ubiquitous in Western Southland In the outbreak described, the resistance of the fawns was probably weakened by an intercurrent *E. coli* infection and cold, wet conditions Although the concentration of hinds in the paddocks was not excessive, they tended to camp and fawn in an unsuitable muddy gully - away from interference of a public golf course. The water from this gully drains across to the paddock of the second mob whose fawns were subsequently affected. Water borne transmission is a possibility (8).

Two outbreaks in fawns have been described in the U K (1) (6) and two in New Zealand (10), all under different circumstances Both New Zealand cases were in housed fawns One of the U.K outbreaks affected fawns 24 - 72 hours old, i e younger than the fawns under discussion, the other at 3 - 11 days

In the neonatal fawns there were low liver vitamin E levels which could have depressed the immune status of the affected fawns. The issue of neonatal liver vitamin E concentrations in ruminants is contentious (M.B Orr, pers. comm.) but the principle could be relevant to New Zealand conditions in selenium deficient herds. Cold wet weather was also a feature of the British outbreaks

The immune response is obviously a critical factor in the pathogenesis of cryptosporidial infections. In SPF lambs cryptosporidial oocysts were excreted in the presence of high IgM and IgG antibody levels. Oocyst output fell as IgA peaked (11) Furthermore colostral antibody concentrations in the sera of fawns in one of the British outbreaks was considered to be adequate (1). It would appear that intestinal IgA produced by the fawn or possibly derived from colostrum could be the key to protection against infection. In ruminants IgA is almost totally derived from the intestine (11) and serum concentrations would be expected to appear only in response to intense IgA synthesis in the intestine. For these reasons the source of adult deer serum and bovine colostrum used to treat fawns (10) may be more important than their use *per se*

Other treatments for cryptosporidiosis have been ineffective (8) (10) The apparent success of toltrazuril in this outbreak can only be regarded as inconclusive under such uncontrolled circumstances, but warrants further investigation An effective single dose treatment would be extremely useful to farmers facing a disease with potentially disastrous financial implications

At present the farmer who has had an outbreak can only rely on such preventive measures as: provision of good shelter, dry fawning paddocks and ensuring the selenium and nutritional status of the hinds pre-fawning is adequate. If intercurrent infections are a problem the use of a commercially available rotavirus/K99 vaccine on the hinds could be considered, though none is currently licensed for use in deer

Finally there are two other factors that the practising veterinarian must appreciate It is important to warn the client about the zoonotic potential of *C parvum* It is also important not to be implicated as a vector - bearing in mind that the organism is resistant to most commonly used disinfectants (8) (12).

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