A Brief Review of Bovine Coccidiosis in Western Canada

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SUMMARY

Coccidiosis of beef cattle, in both its enteric and nervous forms, seen in feedlots in Western Canada is discussed. Cases of coccidiosis accompanied by nervous signs, occasionally up to 30% of those affected enterically, are most common during the coldest winter months. The pathogenesis of the nervous form of the disease is unknown. Clinical management of disease outbreaks using various chemotherapeutics is described. The importance of using anticoccidial drugs before the onset of clinical signs in cattle in contact with sick animals is discussed.

RÉSUMÉ

Une brève revue de la coccidiose bovine, dans l'ouest du Canada

Cet article commente les formes entérique et nerveuse de la coccidiose bovine, telles qu'elles sévissent dans les parcs d'engraissement de l'ouest du Canada. Les cas de coccidiose, accompagnés de signes nerveux, surviennent surtout durant les mois les plus rigoureux de l'hiver et représentent une complication d'environ 30% des cas de coccidiose intestinale. On ignore la pathogénèse de la forme nerveuse de la coccidiose bovine. Les auteurs décrivent l'approche thérapeutique des éclosions de cette maladie, à l'aide de divers médicaments. Ils commentent aussi l'importance d'utiliser des médicaments contre la coccidiose, avant l'apparition de signes cliniques, chez les bouvillons qui vivent avec des congénères atteints de cette maladie.

INTRODUCTION

Coccidiosis is a common disease of beef cattle in Western Canada, which may affect 25-50% of a herd. The disease occurs most commonly in calves from six to 12 months of age during the fall and winter months when the calves are confined in farm or commercial feedlots. The disease is characterized clinically by dysentery which lasts for a few days and most affected animals recover spontaneously without treatment. The case fatality rate is usually below two percent. Treatment using sulfonamides and other chemotherapeutics is practised widely although spontaneous recovery is common.

Some major unsolved problems are: 1) the "nervous form of coccidiosis" in which the case fatality rate is much higher than those without nervous involvement and 2) the clinical management (treatment and control) of herd outbreaks of the disease.

Life Cycles and Pathogenesis

The two species of coccidia which are considered most pathogenic to cattle are *Eimeria zuernii* and *E. bovis* and their life cycles are similar (12). Both organisms have two asexual and one sexual stage. The first asexual stage of both organisms is a giant schizont (up to 300 μm in diameter) found in the lower ileum of the small intestine. The first asexual schizonts of *E. bovis* are found in endothelial cells lining the lacteals of villi while those of *E. zuernii* are found in the lamina propria usually close to the muscularis mucosa (5, 17). It is not always possible to differentiate the two species, as schizonts of *E. zuernii*, in the villal lamina propria, may bulge into the lacteals but they have not been reported in the endothelial cells lining the lacteals. Also, it is fairly easy to confuse first generation schizonts of *Eimeria auburnensis* with those of *E. zuernii* as both are found close to the muscularis mucosa of the lower ileum. However, the first generation schizonts of *E. auburnensis* are reported to parasitize epithelial cells of the crypts (1).

The second asexual stage of both *E. bovis* and *E. zuernii* occurs in epithelial cells of the cecum and colon. Both the second generation schizonts and merozoites of *E. zuernii* are larger than those of *E. bovis* (8, 17). It is unlikely that any confusion can arise with *E. auburnensis* at this stage as second generation schizogony and gametogony in this species occurs in the lamina propria of the ileum (2).

Gametogony of *E. bovis* and *E. zuernii* usually occurs in epithelial cells of the cecum and colon but in heavy infections of *E. bovis* may occur also in the lower ileum (5).

The stages of the life cycle that cause functional and structural lesions of the large intestine are the same in both species of coccidia, that is second generation schizogony and gametogony.

Development of the second generation schizonts and gamonts of both species within the epithelial cells causes mechanical disruption of the cytoplasm. This in turn prevents normal absorptive function of the cecal and colonic epithelia and in the case of *E. zuernii* the nonabsorption of sodium ions (11). *Eimeria bovis*, on the other hand, causes nonadsorption of fluid but there is apparently less effect on sodium ion resorption (3). As the second generation schizonts or gamonts mature the cells containing them slough from the basement membrane and either leave a few attenuated epithelial cells to cover the lamina propria or actually expose the lamina propria with engorged capillaries. Depending upon the extent to which the exposed capillaries are damaged this may result in the loss of plasma or whole blood.

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The histopathogenesis is as follows: The epithelial cells of the glands can be visualized as rows of schizonts or gamonts indicating the lines of former glands. As these cells are sloughed only lamina propria can be seen as connective tissue, capillaries, smooth muscle cells and infiltrating leucocytes with a few epithelial cells here and there. Usually the surface of the now exposed lamina propria is covered with a fibrinous membrane often in several layers in which erythrocytes, leucocytes and oocysts are trapped. The epithelium regenerates from islands of epithelial cells scattered through and on the surface of the lamina propria. The regenerating cells have large pale basophilic nuclei and there are many mitotic figures present in their nuclei (18).

The gross lesions resulting from the above changes are a diphtheritic membrane covering the exposed lamina propria. Often the lumen of the large intestine contains blood and occasionally sausage-shaped fibrin casts up to 0.5 m in length. Generally, the submucosa and external muscular layers beneath the affected areas of the large intestine are greatly thickened, up to 1 cm by edema. Occasionally petechiae are seen along the longitudinal ridges of the large intestine and rectum (18).

"Nervous" Coccidiosis in Cattle

The nervous form of coccidiosis in cattle has been recognized for many years but with increased frequency in the last ten years (10, 14). In herd epidemics of coccidiosis, about 20 to 30% of affected cattle may have nervous signs. In addition to the acute dysentery which is characteristic of coccidiosis, those with nervous signs show muscular tremors, convulsions, opisthotonus, nystagmus, occasional blindness, and there may be a case fatality rate of about 50%. Those with nervous involvement may die during a convulsion on the first day of illness or they may survive for three or five days and then die. A terminal convulsion may occur unexpectedly or the animal may recover within one week.

A retrospective study of 102 cases of bovine coccidiosis admitted to the large animal clinic of the Western College of Veterinary Medicine from November 1972 to January 1979 revealed that 21% were affected with nervous signs; more than 90% of those with nervous signs occurring during the coldest months of the year from January to March.

Laboratory examination of cattle with the nervous form of coccidiosis did not reveal any abnormality significantly different from those usually present in bovine coccidiosis.

There were no epidemiological differences between cattle with or without nervous signs. Of all cattle that died of coccidiosis, only 75% had gross evidence of colitis and typhlitis whereas 91% of those with nervous signs had evidence of colitis and typhlitis. Of all cases which died from coccidiosis, 66% had nervous signs. There were no significant gross or histopathological lesions present to explain the nervous signs.

Treatment and Control

Bovine coccidiosis is reportedly controlled by a number of drugs, all of which have been reported as successful (Table I). Most animals have clinical signs due to coccidia during the gametogenous stages of species such as *E. bovis* and *E. zuernii* and thus are probably already at or just past the most pathogenic phase of the disease. Thus, treatment will usually appear to be effective. Most cases recover without treatment. Animals which are anorexic, dehydrated and weak, should be isolated from the herd and treated with fluids orally and parenterally. Severe dysentery may persist for up to four to six days but if hydration is maintained with fluid therapy, recovery usually occurs within one week.

Corticosteroids are contraindicated for use in clinical coccidiosis of cattle because they contribute to death and increase the case fatality rate (15).

There is no satisfactory, reliable treatment for the nervous form of coccidiosis. Fluid therapy with a balanced electrolyte and glucose solution given orally and parenterally appears to be a rational choice for treatment. Affected cattle should be brought into a well-bedded, weather protected shelter during treatment. Claims have been made for the use of thiamine hydrochloride and injectable vitamin A. However, in this clinic these have not provided any

TABLE I
CHEMOTHERAPEUTICS WHICH HAVE BEEN RECOMMENDED FOR THE TREATMENT AND CONTROL OF COCCIDIOSIS IN CATTLE

Chemotherapeutic Agent	Treatment	Control	Cost per 500 lb (225 kg) Calf (for control)
Sulfamethazine	140 mg per kg body weight orally, daily for three days individually.	In water 140 mg per kg body weight first day followed by 70 mg per kg body weight next six days followed by 35 mg per kg body weight for next ten days	C $2.89 for 17 days
Amprolium 25% powder	Individual dose at 10 mg per kg body weight daily for five days or 65 mg per kg body weight one dose	In feed at 5 mg per kg body weight for 21 days Withdrawal — seven days	C $0.91 for 21 days
Monensin		In feed at 33 g/tonne for 31 days No withdrawal required	C $ 0.51 for 31 days
Chlortetracycline and Sulfadimidine		Each drug in feed at 350 mg per animal per day for 35 days beginning five days before exposure Withdrawal - ten days	C $2.10 for 35 days

beneficial response. Long-term sedation over a period of two to four days, to control the convulsions may be a possible treatment and should be evaluated. Convulsions or mild muscular tremors may persist for up to five days and be followed by spontaneous recovery. An economical method of controlling convulsions over a three to five day period would probably be useful and may improve the survival rate.

Management of Herd Outbreaks

1. Identify and isolate all affected animals and treat them for the effects of the dysentery. Intensive fluid therapy over a period of three to five days may be necessary in severe cases.
2. Attempt to reduce the stocking rate of the animals in affected pens or corrals. Overcrowding is a common occurrence in epidemics of coccidiosis.
3. Ensure that all feed is fed in troughs to avoid fecal contamination.
4. Provide extra bedding in an attempt to reduce oocyst concentration and to minimize the stress of cold weather when most epidemics occur.
5. Consider mass medication of the feed or water supplies of all contact animals for up to 21 days. The drugs and their dosages which have been recommended for the control of the disease are summarized in Table I.

Hammond *et al* (6) treated calves infected experimentally with *E. bovis*, with multiple small doses and single large doses of sulfamethazine. They reported that treatment with either of the above drugs at a dose rate of 0.15 g/lb body weight every other day for a period of ten to 18 days after infection or a single treatment of either drug 13 days after infection with a dose of 1.5 g/lb body weight effectively controlled coccidiosis. They found that sulfamethazine given at a dose rate of 21.5 mg/kg on days 12 and 14 after infection reduced the likelihood of the occurrence of dysentery and the length of duration of diarrhea (7).

Sulfamethazine and other derivatives of sulfanilamide have long been regarded as highly effective for control of bovine coccidiosis. More recently, amprolium and monensin have also been reported as effective drugs for the control of bovine coccidiosis. Hammond *et al* (9) reported that amprolium was effective against experimental bovine coccidiosis due to *E. bovis* at rates of 143, 36 or 22 mg/kg body weight if given daily for 21 days beginning on the day of dosing the calves with oocysts. They also stated that the highest dose rate (143 mg/kg) was effective if given for five days beginning day 13 after infection.

Two reports have been made on the efficacy of monensin for the treatment of coccidiosis due to *E. bovis*. In the first (4) it was reported that monensin fed throughout the incubation period of an experimental infection of *E. bovis* at a rate of 1 mg/kg body weight/day prevented the development of the clinical signs of the disease but that a few oocysts were produced. In the second, McDougald (13) described the prevention of coccidiosis due to *E. bovis* by feeding monensin at the levels of 16.5 or 33 g/metric ton of feed for 31 days. The animals were dosed with 100,000 oocysts of *E. bovis* on the third day after the incorporation of the drug into the feed. Monensin has also been shown to be effective in the prevention of the disease caused by *E. zuernii* (19). Animals experimentally infected with *E. zuernii* were treated at a dose rate of 1 mg/kg daily for a period of ten days beginning the tenth day after infection. None of the treated animals died or became sick and they continued to gain weight uninterruptedly.

The sulfa drugs and chlortetracycline are probably most useful as therapeutic agents as they aid in permitting normal epitheliogenesis to take place. The lesions of *E. bovis* and *E. zuernii* are produced mainly during gametogony of the parasite so that specific anticoccidial drugs at this stage of the parasites' life cycle are probably not of great value. However, cattle in contact with the clinically sick animals will almost certainly be incubating the protozoa at varying phases of their life cycles. It is likely that mass treatment of these in contact animals with amprolium or ionophone anticoccidials, such as monensin, would be useful. It should be realized that this mass medication will be less effective if the coccidia are in the later phases of their life cycles as these phases may not be susceptible to the action of those drugs.

Resistance to reoccurrence of bovine coccidiosis due to *E. bovis* and *E. zuernii* after a primary infection has been reported (9, 16, 19). Hammond *et al* (9) challenged calves with inocula of *E. bovis* after they had earlier been infected with the same organism and successfully treated with dose levels of amprolium of 22 mg/kg and 36 mg/kg daily for 21 days beginning on the day of infection. They reported that only one calf of their experimental group had a transient diarrhea and all had low fecal oocyst counts after the challenge. In experimental infections with *E. zuernii*, resistance to reinfection with the organism has been reported in calves after they had been experimentally infected and treated with either amprolium or monensin (19).

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Répertoire de vaccins antirabiques pour animaux, vendus au Canada

Cette liste s'adresse aux vétérinaires et à ceux dont les activités touchent la lutte contre la rage au Canada. Des mises à jour seront publiées lorsque de nouveaux vaccins arriveront sur le marché ou que des modifications seront apportées aux produits homologués.

Les vaccins antirabiques doivent être administrés par la voie intramusculaire, dans une région de la cuisse, afin d'assurer une immunisation efficace. Les chiens ou les chats vaccinés avant l'âge de trois mois doivent être revaccinés à cet âge pour devenir entièrement immunisés. Le Tableau I donne plus de détails à cet égard.

Au Canada, il n'existe actuellement aucun vaccin antirabique homologué pour les bêtes sauvages ou les animaux gardés en captivité dans les jardins zoologiques. On ne possède pas de documentation sur leur utilisation. Dans les cas où la vaccination de tels animaux s'avère nécessaire, on ne doit employer que des vaccins inactivés. On déconseille de garder des animaux sauvages comme animaux de compagnie, à cause de l'incidence élevée de la rage chez ces derniers.

Une exposition accidentelle à des vaccins inactivés ne représente aucun risque pour l'homme. Les données empiriques recueillies au fil des ans nous permettent également de conclure que les vaccins atténués ne semblent pas communiquer la rage. Toutefois, la documentation en la matière est incomplète. En cas de contact avec ces vaccins, il est préférable de prévenir son médecin ou les agents de la santé publique.

Certains pays envisagent de plus en plus de restreindre l'usage des vaccins antirabiques aux vétérinaires. Pour l'importation et l'exportation d'animaux de compagnie, les postes frontaliers canadiens n'acceptent que les certificats de vaccination émis par des vétérinaires. Au Canada, la politique des fabricants de vaccins consiste à ne vendre leurs produits qu'aux vétérinaires.

TABLEAU I
VACCINS ANTIRABIQUES POUR ANIMAUX, VENDUS AU CANADA

Vaccin, Fabricant, Distributeur	Espèces animales	Posologie[a] (mL)	Age des 1ères vaccinations (mois)	Intervalle avant revaccination (année)
A. Virus vivant atténué				
Culture de tissu porcin, souche ERA, Laboratoires Connaught	Canine	2	3 et 15	3
	Féline	2	3 et 15	2
	Bovine	2	4	4
	Équine	2	4	2
	Ovine	2	4	1
	Caprine	2	4	1
Culture de tissu canin, souche SAD, Philips Roxane Inc./ Pitman-Moore Inc.	Canine	1	3 et 15	3
Lignée cellulaire canine, souche Flury Laboratoires Norden Inc.	Canine	1	3 et 15	3
	Féline	1	3	1
B. Virus inactivé				
Origine murine, Laboratoires Rolynn/ Laboratoires Ayerst	Canine	1	3 et 15	3
	Féline	1	3	1

[a]Tous les vaccins doivent être administrés par la voie intra-musculaire, en une seule injection, dans la cuisse.

Préparé par le personnel de la section des vaccins vétérinaires. Direction de la santé des animaux, Direction générale de la production et de l'inspection des aliments, ministère de l'Agriculture du Canada, C.P. 11300, Station H, Nepean, Ontario K2H 8P9.