Saskatchewan

Abortion due to *Coxiella burnetii* in a goat

In the spring of 1990, in Saskatchewan, the first confirmed case of disease caused by *Coxiella burnetii* occurred in a mature female goat. The infection resulted in a late-term abortion and dystocia of three fetuses, one of which was alive, but nonviable, and two were dead.

Significant gross or microscopic lesions were not found in one fetus examined. The placenta had severe inflammation, characterized grossly by thickened leathery patches within the intercotyledonary areas, which were partially mineralized and edematous. Macroscopically, the cotyledons appeared normal. The abundant grey-brown exudate often associated with this disease was not seen (1). Large numbers of rickettsia-like organisms were present on placental impression smears. The organism was not isolated using standard techniques. Histologically, the cotyledonary and intercotyledonary areas had a moderate infiltrate of neutrophils, macrophages, lymphocytes, and plasma cells. Many cotyledonary epithelial cells were distended by intracytoplasmic organisms. The intercotyledonary areas had multifocal fibroplasia, superficial necrosis, mineralization, and edema, with total loss of epithelium. Indirect immunoperoxidase stain for *Chlamydia* sp. was negative. Abortion due to *C. burnetii* was confirmed by serology of the dam (Dr. G.H. Lang, Department of Microbiology and Immunology, Ontario Veterinary College, University of Guelph, Guelph, Ontario) and ultrastructure of the intracellular organisms (1).

Coxiella burnetii is the cause of the zoonotic disease, Q fever in humans, and was reviewed recently (2). In North America, the disease in animals has been associated mainly with abortions, stillbirths, and weak neonates in goats and sheep (California, Idaho, Ontario) (1). There have also been reports of stillbirths in cats (Maritimes) and rare abortions in cattle (1,3). Infection can often be asymptomatic in these animals, and in a wide variety of other species (1).

A 1964 review of Q fever in Canada reported that cattle in Saskatchewan had a reactor rate of 1% (3), indicating that *Coxiella* sp. may have been present for some time in this province. **In abortions caused by this agent, the most significant lesions occur in the placenta and, without placenta, confirmation of disease is very difficult.**

References

1. Zeman DH, Leslie-Steen P, Peacock MG. Ovine abortion caused by *Coxiella burnetii*. In: Kirkbride CA, ed. Laboratory Diagnosis of Livestock Abortion. 3rd ed. Ames: Iowa State University Press, 1990: 175-183.
2. Marrie T J Q fever — A review. Can Vet J 1990; 31: 555-563.
3. McKiel J A Q fever in Canada. Can Med Assoc J 1964; 91: 573-577.

Shelagh Copeland, Department of Veterinary Pathology, Manuel Chirino-Trejo, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0; Phil Bourque, Andrew Biernacki, Rosthern Veterinary Clinic, Rosthern, Saskatchewan S0K 3R0

Alberta

Suspected ivermectin toxicity in kittens

Four seven-week-old domestic kittens, owned by a pet store, were presented to the clinic for vaccination. All were found to be healthy except for an infestation of ear mites in each kitten. The kittens were vaccinated against feline rhinotracheitis, calicivirus, panleukopenia, and chlamydia, and were also given, by injection, 1 mg ivermectin used for cattle. Eighteen hours later, the owner of the pet store reported that the kittens were ill, and returned them to the clinic. All kittens had mild diarrhea, and the only female kitten also had posterior ataxia and mild miosis. By the next morning, two kittens were in sternal recumbency, with head bobbing and vocalization. Diarrhea continued in all four kittens. In 48 hours, diarrhea had stopped, but the two kittens in sternal recumbency on the previous day were semi-comatose. One other kitten was mildly ataxic and the fourth kitten was moderately ataxic. By 60 hours, two kittens had shallow, regular respirations, and were in a deep coma. The other two kittens remained ataxic. By 72 hours, two kittens remained in a deep coma, whereas one other kitten was almost clinically normal, and the fourth kitten was only mildly ataxic.

The comatose state persisted in two kittens for a further 48 hours before signs of recovery were noted. When physically stimulated, the two kittens that had been comatose responded with muscular twitches and faint vocalizations. The other kittens were now clinically normal. One of the two comatose kittens was the last to recover fully, seven days after administration of ivermectin.

During this period of illness, the kittens were placed in an incubator at 28°C, rotated in lateral recumbency each hour, and given 1 mg dexamethasone by injection daily. If unable to eat or drink, kittens were given q12h 10 mL physiological saline subcutaneously, and 1 mL Aminodex (Austin Laboratories, Joliette, Quebec), 5 mL ClinicCare feline supplement (Pet Ag Inc., Hampshire, Illinois) by stomach tube.

After recovery, the kittens were returned to the pet store and sold. One kitten was later returned to the clinic as a patient, and has been a normal, healthy animal. We believe that patience and perseverance are important factors in the resolution of such a problem. **As well, we are aware that the dosage of ivermectin used in these animals was excessive, but the range of clinical signs was startling, and in our opinion, needs further explanation. Finally, the risks of off-label use of ivermectin must be considered prior to its use.**

Harold Frischke, Laura Hunt, Glamorgan Animal Clinic Ltd., 3919 Richmond Road S.W., Calgary, Alberta T3E 4P2