Ontario

Q fever abortions in a goat herd

From mid-January to mid-April 1992, reproductive failure characterized by abortion or premature kidding afflicted 11 of 33 pregnant does in a goat herd in southwestern Ontario. Affected does were of different parities and various ages (1–9 years old), and all were in late pregnancy. A cesarian section was performed when the cervix of one doe failed to dilate, while she was straining to abort. The cervices of three other does had to be manually dilated for the same reason. The uterus in one of these goats ruptured during manipulation.

Aborted fetuses were unremarkable. Purulent intercotyledonary placentitis was prominent in all placentae. Purulent segmental cotyledonary placentitis was an occasional finding in a few placentae. The placentae from six of the does were submitted to the Huron Park laboratory for histological examination. Microscopic lesions consisted of coalescing areas of marked purulent to necrotizing placentitis and placental mineralization, most pronounced in the intercotyledonary areas. Chorionic epithelial cells in the intercotyledonary areas were swollen and had a "soap bubble" appearance. Faintly acid-fast-positive, pleomorphic organisms consistent with *Coxiella burnetii* were present in the chorionic epithelial cells (1). Sera from 34 of the 40 adult goats in the herd tested positive for antibodies to *C. Burnetii* in April 1992, using an ELISA test (SANTEIA, Animal Health Support, Guelph, Ontario). Eight of the 10 does that aborted tested positive; the eleventh doe was euthanized because of the ruptured uterus, so she was unavailable for testing. Q fever was diagnosed as the cause of the abortions (1).

Eight of the 11 goats that aborted had attended the annual Royal Winter Fair in Toronto in November 1991, where they were housed with a group of parturient goats. Q fever was diagnosed as the cause of abortion in several goat herds in the Niagara region in the winter of 1992. Goats from each of those herds had also been exhibited at the Royal Winter Fair. **To date, at least six people in the Niagara region of Ontario have contracted clinical Q fever in the 12 months following the 1991 Royal Winter Fair (2).** All six people had had either direct contact with parturient goats at the Royal Winter Fair or exposure to parturient goats that may have been infected at the Royal Winter Fair (2). A manuscript is in preparation documenting the outbreak of reproductive failures in several goat herds, all associated with exposure to the parturient goats at the 1991 Royal Winter Fair.

References

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Alberta

Porcine clostridial enteritis

Clostridium perfringens Type C is responsible for necrotizing, hemorrhagic enteritis in suckling animals of most livestock species (1). The organism is present in the soil and maternal feces, and infection of neonates occurs within the first hours of life. In swine, the disease takes one of four forms. Peracute infection results in death within 48h from a hemorrhagic enteritis, which may or may not result in diarrhea. Acute infection is manifest as hemorrhagic to necrotizing enteritis with variable diarrhea and has a clinical course of two to three days. A subacute form of disease results in death within seven days with progressive emaciation and diarrhea, as a result of necrosis throughout the small intestine. Infrequently, a chronic form of the disease occurs resulting in a failure to thrive. The disease usually strikes in the first week of life but has been reported in two to three-week-old piglets, as well as in weaners (2). The disease has a tendency to occur sporadically, although it will persist on affected farms, and it is a disease that we rarely confirm as being present in swine operations in Alberta.

In January 1993, two piglets were received by the Airdrie Regional Laboratory from a farrow to finish operation with 60 sows. The farm had begun operation in October 1992, on a new site, with purebred Yorkshire bred sows purchased from a closed, high-health herd. Bred and pregnant sows were housed in outdoor pens; farrowing took place and growing pigs were housed in a new barn. Four of the first five litters born were normal, but all pigs in the fifth litter died by three weeks of age. By mid-January, piglet deaths had occurred in one-third of the litters born on the premises with an average of half the litter lost by

three weeks of age. There were occasional signs of scours, but most of the affected piglets found dead were large, well-doing individuals.

The piglets submitted to the laboratory were from two litters and had similar lesions. Both were markedly dehydrated but in good flesh. The stomach contained small mild clots and showed mild congestion of the mucosa. The content of the duodenum and proximal jejunum was scant and dark yellow. **From mid-jejunum through the ileum, the intestinal loops were plump to turgid, and the serosa had a 'ground glass' appearance, with ecchymotic and coalescing hemorrhages prominent over the jejunum.** There was transmural thickening with mild focal emphysema, and pale linear streaks were visible through the serosa, corresponding to adherent necrotic membranes on the mucosa. The intestinal lumen contained a moderate quantity of reddish-tan material with flecks of sloughed necrotic debris. The spiral colon was distended with reddish, watery fluid and had a similar, but less developed, necrotic mucosal membrane. One piglet also had multiple, acute intussusceptions.

Clostridium perfringens was isolated in heavy growth from intestinal contents incubated anaerobically. Typing was not possible in this laboratory.

Microscopic examination of sections prepared from the intestines revealed diffuse, severe, epithelial necrosis. Neutrophil exudation and fibrin accumulation were present where epithelial sloughing had occurred, and mixed inflammatory cells were present through the muscular layers of the intestinal wall. Hemorrhage was evident in many regions. Gram's stain demonstrated a large number of gram-positive rods, both adherent to remnants of villi and beneath the mucosa of intact villi. Lesions in the colon were similar but less severe.

A diagnosis of hemorrhagic, necrotizing enterocolitis due to infection with *Clostridium perfringens* was made. The changes seen were consistent with type C, in which piglet death is due to intestinal damage combined with the effects of toxins elaborated by the bacterium (2). Two more litters were subsequently affected, but once the diagnosis had been made, sows were vaccinated with a type-C toxoid. Since the organism is present in feces and soil, the producers were advised to wash sows before placing them in farrowing crates, to reduce environmental contamination for young piglets. No further instances of enteritis have occurred.

References

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British Columbia

Aspergillosis outbreak in Stellar's jays (*Cyanocitta stelleri*) from central Vancouver Island

During September and October 1992, there was a considerable increase in the number of injured Stellar's jays admitted to the Island Veterinary Hospital, Nanaimo, British Columbia. Twenty-seven jays were admitted from the region of central Vancouver Island between Ladysmith and Qualicum. A number of the birds had been attacked by cats or had old injuries, and many were thin and experiencing respiratory difficulty. Previous records from 1987 to 1990 showed that only six Stellar's jays had been admitted. It was recognized that there was a larger population of jays in the area in 1992, which may have accounted for the increase in the number of admissions, but nine of the birds had aspergillosis lesions on postmortem examination.

The birds were dehydrated, lethargic, hypothermic, and often emaciated. Gross postmortem examination revealed variously sized yellow nodules (1–5mm) scattered throughout the lungs and, frequently, within the airsacs. The airsacs were visibly thickened, and in a few birds, there were mycotic plaques. Histological examination of tissues revealed multiple granulomatous areas distributed throughout the pulmonary parenchyma. There were irregular areas of necrosis within the lungs, surrounded by atelectatic pulmonary alveoli and numerous epitheliod cells. Extensive necrotic debris was found within granulomatous areas, and numerous mycotic hyphae were present. Silver stains revealed large numbers of fungal hyphae typical of *Aspergillus* sp. within the necrotic debris.

The hematological and biochemical values for one extremely emaciated Stellar's jay were examined. This bird had complete obliteration of the right lung lobe by a large caseous mass. There was a moderate anemia, severe leucocytosis with marked heterophilia, and a mild monocytosis, consistent with the inflammation in the lung. There was evidence of liver damage with elevation of alanine aminotransferase and aspartate aminotransferase. Uric acid and creatinine kinase were extremely high at 5170 umol/L and 30060 IU/L, respectively. Calcium levels were decreased. The hepatic and renal enzyme elevations may have been secondary to anoxic damage, since there were no observable lesions in these organs.

Aspergillosis is an infectious disease of the respiratory tract most commonly caused by *Aspergillus*