Feline Infectious Peritonitis

The owner of a small cattery breeding Persian cats sent 3 dead kittens (one 4 months old, and two 4½ months old) to the necropsy service of the Faculty of Veterinary Medicine, University of Liège. These kittens constituted the 3rd litter of a 3-year-old female. Feline infectious peritonitis (FIP) was diagnosed in all 3 kittens on the basis of characteristic macroscopic and microscopic lesions.

Five months later, the same female gave birth to 3 kittens. One of them died of FIP at the age of 2½ months; the diagnosis was based on macroscopic findings. We then proceeded with clinical examinations of the mother and the 2 other kittens. The mother had lost weight and was weak. The 2 kittens were clinically normal.

Two months later, the kittens had the same pattern of weight loss and weakness, accompanied by retarded growth. Five months later, the mother and the 2 kittens appeared clinically normal. During the whole observation period, no other cat in the cattery had clinical signs of FIP. Specific precautions were not taken to avoid contagion in the cattery.

The etiology of FIP is slowly becoming clear, based on the experimental transmission of the disease agent in filtrates of organ extracts and the electronmicroscopic observation of viral particles in diseased tissues. Some specific characteristics of the etiologic agent have been reported.

Up to now, all theories concerning spread of FIP have been based on clinical observations, serologic tests being only recently available. Neonatal FIP has occurred among kittens produced by certain queens. In a recent review, the different forms of the disease as well as the possible modes of transmission were described. Transplacent transmission of the disease agent seems applicable to the kittens of this report.

Unfortunately, the feline leukemia status of the affected kittens was unknown. Concurrent diseases, especially feline leukemia, apparently can
suppress a cat's resistance enough to allow the FIP virus to cause disease when it might otherwise pass unnoticed.¹

One of the affected kittens died during a period in which it should have been protected by maternal immunity. Regardless of this fact, it cannot be excluded that the kittens obtained the infection by direct contact at around 6 to 9 weeks of age, either from the mother or from clinically normal carriers in the immediate environment.—P.-P. Pastoret, M. Gouffaux, M. Henroteaux, F. Schoenaers, Faculté de Médecine vétérinaire ULg, 45, rue des Vétérinaires, 1070 Brussels, Belgium, and J. Tepper, 2 Penn Ct, Dix Hills, NY 11746.


Equine Intestinal Clostridiosis

Clostridium perfringens (type A) infection in the horse was studied because this organism had been isolated in large numbers from the intestinal content and feces of horses with acute and often fatal disease.

Affected horses typically had an acute onset, with apathy, foul-smelling diarrhea, discolored mucous membranes, and elevated pulse rate and temperature. Not infrequently, affected horses died shortly after onset of clinical signs. Case histories revealed that affected horses had been subjected to stress prior to onset of the disease. There were widespread angiopathies with hyperemia, edema, hemorrhages, and poorly coagulated blood. The changes were most marked in the cecum and large colon and were diagnosed as acute typhilitis and colitis. Some horses had lesions in the myocardium, liver, and kidneys.

In horses that survived, the disease regressed, and parallel to this the C perfringens counts decreased to normal levels. A positive correlation between the numbers of C perfringens in feces and the severity of illness was established; C perfringens was detected only in exceptional cases in the intestinal contents and feces of healthy horses and then only in very low numbers.

Immunologic investigations indicated that sick horses and horses exposed via the intestinal tract to C perfringens were immunologically stimulated by its extracellular antigens.

The cause of the rise in the C perfringens counts in the sick horses could not be established with certainty. Treatment with antibacterial drugs could not be excluded as a contributing factor in a few of the cases. It was proposed that fodder rich in protein with a relatively low content of cellulose and in combination with stress might be of etiologic importance.

The disease resembles colitis X and similar disorders. However, the prevalence of C perfringens above a certain level combined with the described clinical and pathoanatomic findings was regarded as the best diagnostic criterion for the disease. Because C perfringens seemed to be of etiologic importance, the disease was named equine intestinal clostridiosis.