1975

Feline Infectious Peritonitis

Chuck Lemme
Iowa State University

Johnny D. Hoskins
Iowa State University

Follow this and additional works at: https://lib.dr.iastate.edu/iowastate_veterinarian

Part of the Gastroenterology Commons, Small or Companion Animal Medicine Commons, and the Veterinary Infectious Diseases Commons

Recommended Citation
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol37/iss1/4

This Article is brought to you for free and open access by the Journals at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
Feline Infectious Peritonitis

by
Chuck Lemme*
and
Johnny D. Hoskins D.V.M.†

Summary

Feline infectious peritonitis was diagnosed in a two-year-old Ragdoll cat at Bay Cities Pet Hospital, Torrance, California in August of 1974. The article describes the specific case and discusses the current knowledge of the disease.

Introduction

Feline infectious peritonitis was first described as a separate disease entity in 1966, although the disease had been present long before that. It is most commonly seen in young cats and has been known to affect whole litters. A corona virus is said to be the etiologic agent. The clinical signs include fever, anorexia, ascites, and listlessness. The disease is diagnosed by clinical signs and examination of aspirated peritoneal exudate. Although affected cats may live for several weeks, the disease is always fatal in spite of vigorous therapy.

Most of the lesions of infectious peritonitis are found in the abdominal cavity. There is a diffuse fibrinous inflammation of the serosal membranes and areas of focal necrosis in some of the organs. The disease can be reproduced experimentally by inoculation with tissues from infected cats, even though the etiologic agent has never been isolated.

Case Report

On August 12, 1974, a two-year-old intact female Ragdoll cat was admitted to the Bay Cities Pet Hospital in Torrance, California. The owners complained of lethargy and inappetence of a few days duration. On physical examination the cat appeared depressed, anemic and had a rectal temperature of 105°F. Penicillin (200,000 units) was given intramuscularly.

The next day the cat's condition appeared about the same. Her temperature was still 105°F., she was still depressed, and now appeared six percent dehydrated. The cat had not eaten or drunk any water during the night. A blood sample was taken for a complete blood count and a fluorescent antibody test for Feline Leukemia virus. Lactated Ringer's solution (150 ml.) was given subcutaneously and 100 milligrams of chloramphenicol was given parenterally three times that day.

On the third day the cat was in the hospital she appeared more depressed than before. Her rectal temperature, however, had decreased to 102.2°F. The results of the laboratory work showed:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb.</td>
<td>6.0gm%</td>
</tr>
<tr>
<td>PCV</td>
<td>17.9%</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.8%</td>
</tr>
<tr>
<td>WBC</td>
<td>3.9x10³/cmm</td>
</tr>
<tr>
<td>Heinz bodies</td>
<td>slight</td>
</tr>
<tr>
<td>FeLV</td>
<td>neg.</td>
</tr>
</tbody>
</table>

The chloramphenicol was continued and another 150 milliliters of lactated Ringer's solution was given subcutaneously. Paracentesis was attempted but no abdominal fluid could be collected.

On the fourth day of hospitalization the cat had still not eaten any food. She was still very depressed, and the rectal temperature stayed at 102.2°F. Another attempt was made to collect abdominal fluid and a thick, honey-colored fluid was obtained. Feline infectious peritonitis was diagnosed and the owner's permission for euthanasia and post mortem examination was ob-

Issue No. 1, 1975

* Mr. Lemme is a fourth year student in the College of Veterinary Medicine at Iowa State University.
† Dr. Hoskins is an Assistant Professor of Veterinary Clinical Sciences at Iowa State University.
tained. On necropsy the peritoneal cavity was found to be filled with the honey-colored fluid. There were granular deposits of fibrin on the surface of the abdominal viscera.

**Discussion**

Feline infectious peritonitis was first recognized as a separate disease in 1963 by Feldman and Holzworth who called the disease “chronic fibrinous peritonitis.” In 1966 Wolf and Greisemer studied sixteen cases of the disease histologically and named it “feline infectious peritonitis.”

Since that time the disease has been reported in many countries throughout the world. The viral etiology of the disease was confirmed in 1968.

The disease is caused by a virus of the corona group. At this time the virus has not been isolated. Attempts to grow it on tissue culture or in chick embryos have failed. However, the virus has been seen in mesothelial cells of affected cats using an electron microscope.

The virus of infectious peritonitis has been spread experimentally by many routes. Lesion material from an infected cat has been injected intraperitoneally, intravenously, subcutaneously, and intracerebrally, with disease produced in the injected animal. The natural route of transmission is unknown, but the infectious agent can be found in the blood and urine of an infected cat. More than one cat in a litter has been affected in some instances. Cases have also been reported of a cat being infected three to four months after another cat in the same household had the disease.

The greatest incidence of feline infectious peritonitis occurs in cats less than three years old. There is no evidence of sex or breed predisposition.

The clinical signs seen in most cases of feline infectious peritonitis are anorexia, depression, fever from 103 to 105°F., dehydration, ascites, progressive weight loss, and pale mucous membranes. In other isolated cases vomiting, diarrhea, icterus, sneezing, coughing, and pleural effusion may be seen. The incubation period can vary from a few days to a few weeks. Experimentally, the incubation period is from one to six days. The duration of clinical signs can vary from two weeks to two months.

Many conditions have to be considered in the differential diagnosis of a cat showing the above signs. Early in the disease the depression seen may resemble infection with feline respiratory viruses or panleukopenia virus. An enlarged abdomen could be due to pregnancy or a closed pyometra. A bacterial peritonitis must also be considered.

Feline infectious peritonitis is diagnosed mainly on the basis of clinical signs and laboratory data. A CBC should show a moderate leukocytosis with a left shift, lymphopenia, eosinopenia, and anemia. A leukopenia has also been reported in some cases, especially in those near death. Total serum protein is usually greater than 7.8 grams%, due to an absolute increase in the amount of beta and gamma globulins. Serum electrophoresis is a helpful diagnostic tool in this disease. The beta fraction of the globulin appears as a very prominent spike, or it displays altered electrophoretic mobility. It is not known if this spike represents altered IgM or IgA, or an aberrant IgG, but coupled with other clinical signs and laboratory data serum electrophoresis can provide useful diagnostic information.

In about 50% of the cases the cat is found to be positive for feline leukemia virus. A laboratory analysis of the abdominal fluid is also helpful in diagnosis. It can be anywhere from clear, to straw-colored, to blood-tinted. It may clot on exposure to air. The specific gravity is usually greater than 1.017 and can be as high as 1.047. There can be from 1,000 to 10,000 WBC’s per cubic millimeter with 12 to 99 percent of these cells being neutrophils. Total protein levels in the fluid are quite high, usually approaching serum levels. There are generally no bacteria found. In some cases an elevated van den Bergh (total, direct, and indirect) is seen, and the BSP clearance time is prolonged.

There are a number of treatments recommended for feline infectious peritonitis, although it is considered a fatal disease. Most authors recommend aspiration of ab-
dominal fluid, and administration of corticosteroids, fluid therapy, and antibiotics. The type of antibiotic recommended varies. Sulfadimethoxine, Tylosine, and chloramphenicol are all suggested.

The post mortem lesions seen with feline infectious peritonitis divide the disease into two different types. The first type is the "classic" or "wet" type of the disease. In this form of the disease up to one liter of the previously described abdominal fluid can be found. Granular gray-white fibrinous exudate can also be found on the surface of the abdominal viscera in this form. Focal areas of necrosis can be found in the spleen, kidneys, liver, pancreas and muscular layers of the intestine. There can be an associated meningitis and uveitis.

The second type of feline infectious peritonitis is known as the "parenchymal" or "dry" form. It differs from the above type in that the characteristic abdominal fluid is not found. Any of the abdominal organs can be invaded as can thoracic organs. Histologically, affected organs show multifocal pyogranulomas. The kidneys, liver, and pancreas are the most commonly affected organs. Even when the thorax is involved clinical signs of this are seldom seen. Ocular problems are also common in this form of the disease. The damage can vary from corneal edema to retinal detachment. A characteristic lesion is chorioretinitis. CNS involvement is often seen. This can result in posterior incoordination, tremors, or convulsions. Damage to any organ often will result in spread to the subcapsular veins, resulting in a phlebitis.

Since the etiologic agent involved in feline infectious peritonitis has not been isolated, no immunizing product can be produced. Therefore, the only way to prevent the spread of the disease is to isolate suspect animals. Cages in which suspects are kept should be sterilized after use and should be kept vacant as long as is practical. People who have had a cat with infectious peritonitis in their home should be warned about bringing in a new cat to the premises. A minimum period of 30 days should be observed.

References

* The author wishes to acknowledge the help of Dr. Charles O. Gardner, ISU '53, of Torrance, California.

Ectopic Ureter—A Case Report

by Char Slindee* and Russell Mitten, B.V.Sc., M.R.C.V.S., D.V.R.†

Ectopic ureters are characterized as terminating outside the urinary bladder as a result of faulty differentiation of the Wolffian ducts. The condition can be unilateral or bilateral, and is almost always seen in the female dog.