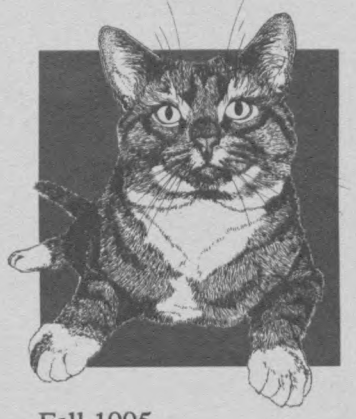

Feline Health Topics

for veterinarians

Fall 1995 Volume 10, Number 4

Feline Exocrine Pancreatic Disease

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and

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Traditionally, the frequency of exocrine pancreatic disease in cats has been considered to be low. However, a recent study has shown that the frequency of significant pathologic lesions of the exocrine pancreas at necropsy is almost as high in cats as it is in dogs (1.4 % in cats and 1.7 % in dogs).[1] Other studies report even higher frequencies.[2, 3] This is contrasted by an infrequent clinical diagnosis of exocrine pancreatic diseases in cats. Cats suffer from various exocrine pancreatic diseases. However, the most common condition appears to be pancreatitis. Chronic forms of pancreatitis are more common than acute forms. Exocrine pancreatic insufficiency and pancreatic neoplasia are less common than inflammatory disease. Finally, other conditions have been reported in the cat such as pancreatic pseudocyst, pancreatic bladder and pancreatic parasites (i.e., *Eurytrema procyonis* and *Amphimerus pseudofelineus*).

The following discussion focuses on pancreatitis and exocrine pancreatic insufficiency. Limited evidence shows that while there are broad similarities between these diseases in cats and dogs, there are also some important differences, particularly with regard to etiology and diagnostic testing.

Pancreatitis

Recent reports have described that acute necrotizing pancreatitis in cats is similar to that seen in dogs, as well as a histologically distinct suppurative form.[4] This contrasts with traditional reports of chronic mild interstitial pancreatic inflammation in cats, characterized by inflammation of interstitial tissue apparently spreading from the ducts, often accompanied by cholangiohepatitis, and sometimes by interstitial nephritis.

Etiology

The inciting cause of spontaneous feline pancreatitis is not determined in most cases. However, several cases of pancreatitis caused by trauma (i.e., high rise syndrome, traffic accidents and abdominal surgery) have been described. Also several infectious organisms, such as *Toxoplasma gondii,* feline Herpesvirus I, feline infectious peritonitis virus, and the feline parvovirus have been named responsible for cases of pancreatitis. Rare cases of pancreatitis caused by organophosphate intoxication have also been described. Coexistent interstitial pancreatitis and

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cholangiohepatitis have been observed in cats, and although the relationship between the changes in these two organs is not clear, convergence of feline biliary and pancreatic ducts may be a factor. Pancreatitis has also been reported in a high proportion of cats with hepatic lipidosis.[5] Suspect drugs associated with pancreatitis in feline patients include furosemide, sulfonamides and tetracyclines. Finally, the infestation of pancreatic tissue by the hepatic parasite *Amphimerus pseudofelineus* has been identified as a rare cause of pancreatitis in the cat.

In summary, as in other species, nutrition/hyperlipoproteinemia, drugs/toxins/hypercalcemia, duct obstruction, duodenal reflux, pancreatic trauma, pancreatic ischemia/reperfusion, viral, mycoplasmal and parasitic infections, and uremia may all be inciting or contributory causes of pancreatitis, but their exact role in feline pancreatitis is unknown.

Diagnosis

Clinical signs of pancreatitis in cats are non-specific. Fever, tachycardia and variable signs of abdominal pain were observed with only rare episodes of vomiting during an experimental study.[6] A survey of 40 cases of fatal feline pancreatitis revealed that nearly all were severely lethargic and anorexic, and that more than 50% were dehydrated or hypothermic. Vomiting was noted in 35% of cases and signs of abdominal pain or an abdominal mass were apparent in 25%of patients.[4]

History and clinical signs associated with pancreatitis are also non-specific and are common to numerous gastrointestinal and metabolic disorders. Abdominal radiographs may provide evidence leading to one of these alternative diagnoses, or support a tentative diagnosis of pancreatitis by revealing ascites or local peritonitis in the anterior right quadrant. Ultrasonographic imaging is proving increasingly useful in identification of patients with pancreatitis. Nonhomogeneous masses and loss of echogenicity have been reported.[1]

Until recently the usefulness of serum amylase and lipase, which serve as markers of pancreatitis in dogs and humans, was unknown. An experimental study demonstrated that while serum lipase increased significantly in cats following induction of pancreatitis, amylase activity was never increased above normal, but rather decreased significantly during the course of the disease.[6] A recent study reported serum amylase and lipase concentrations of 12 cats with pancreatitis. These cats did not have elevations or decreases in either serum amylase or serum lipase concentrations.[8] Hence, serum amylase or serum lipase seems to be of little help in the diagnosis of pancreatitis in the cat. The same study evaluated the use of feline trypsin-like immunoreactivity (fTLI) in serum. The reference range for fTLI has been reported to be 17-49 µg/L.[9] The mean serum fTLI concentration was significantly higher in the group of 12 cats suffering from pancreatitis compared with healthy cats or cats suffering from other diseases. Thus fTLI is the first serum marker for pancreatitis which has shown any promise in the cat.

Feline Health Topics

A publication for veterinary professionals

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere, by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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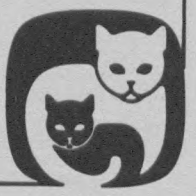

Hyperglycemia is common in cats with necrotizing pancreatitis, probably as a result of hyperglucagonemia and stress-related increases in the concentrations of catecholamines and cortisol. Some affected animals are diabetic following recovery from acute episodes of pancreatitis. In contrast, cats with suppurative pancreatitis often develop hypoglycemia.[4]

The concentration of trypsinogen activation peptides (TAP) in plasma or urine provides a specific marker for pancreatitis, since in the absence of pancreatitis only free trypsinogen is present in the plasma.[10] The TAP concentration correlates with the disease severity and clinical course of pancreatitis in rats and human beings. Recent research has confirmed that the (Asp)4-Lys-peptide sequence, common to TAP in all vertebrates, is also preserved in the cat.[11] Therefore, immunoassays for TAP may also prove to be of practical value in the cat.

Treatment

Supportive care is the mainstay of therapy for pancreatitis in the cat. In the very few cases in which a cause can be identified, the specific cause should be eliminated if possible. The importance of withholding all oral intake in cats with pancreatitis is questionable, especially given the reported high prevalence of concurrent hepatic lipidosis and the attendant desirability of aggressive nutritional support. These cats perhaps should ideally be fed via a jejunostomy tube, and perhaps total parenteral nutrition may be considered. If abdominal pain is marked, analgesic drugs should be given. Plasma is likely to be advantageous in cats with pancreatitis, as in other species, by virtue of both its albumin and protease inhibitor content. The value of other therapeutic agents including antibiotics, glucococorticoids, and antisecretory drugs is highly questionable.

TLI Assay Submissions

Assay of feline TLI is presently only available from Dr. Williams' laboratory. Additional information and sample submission forms can be obtained from Dr. David A. Williams, GI Lab, Purdue University, West Lafayette IN 47907-1248. Fax (317) 496-1796, Telephone (317) 494-0331.

Exocrine Pancreatic Insuffciency

Etiology

The most common cause of feline EPI is chronic pancreatitis. Since both endocrine and exocrine pancreatic cells are destroyed in pancreatitis, feline EPI is often accompanied by diabetes mellitus. Other, less commonly reported causes of EPI in the cat are obstruction to the flow of pancreatic juice secondary to pancreatic adenocarcinoma or other tumors, or secondary to the infestation of the pancreas with pancreatic flukes. EPI has also been reported as a complication of proximal duodenal resection. Dual pancreatic ducts are usually absent in the cat and therefore damage to the major duodenal papilla blocks pancreatic secretion. There are no well documented reports of idiopathic pancreatic acinar atrophy in cats. However, the authors are aware of at least three cases in which this diagnosis was confirmed by direct examination of the pancreas.

Pathophysiology

There are no reports of studies in cats, but naturally occurring and experimental EPI in several other species leads to abnormal activities of intestinal mucosal enzymes and impaired absorption of sugars, amino acids and fatty acids. These functional abnormalities often occur without histologic evidence of mucosal damage. The absence of the trophic influence of pancreatic secretions, concurrent overgrowth

of bacteria in the small intestine, and endocrine and nutritional factors may all be contributory to this dysfunction. Malabsorption of fat-soluble vitamins also occurs secondary to steatorrhea, and vitamin K-responsive coagulopathy has been reported in association with feline EPI.[12] It has recently been reported that the pancreas is the major source of intrinsic factor in the cat, and so affected cats are at risk of developing cobalamin deficiency.[13]

Clinical Signs

Clinical signs of feline EPI are similar to those seen in dogs and include soft, pale and voluminous stools and weight loss. Greasy soiling of the perineal area and sometimes of the entire hair coat appears to be more common in cats.

Diagnosis

Several pancreatic function tests have been recommended in the past (bentiromide absorption, fat absorption and/or plasma turbidity, microscopic examination of feces for evidence of undigested food, such as fat droplets, starch grains or muscle fibers). All of these tests are cumbersome and unreliable. The only exception is fecal proteolytic activity which is more reliable, but also very cumbersome. Fecal proteolytic activity (FPA) using radial enzyme diffusion or azoprotein digestion will identify most affected cats, but will give false positive or equivocal results in a proportion of patients that have small intestinal disease. FPA is present in all normal cats but results are consistently low in most cats with EPI. At least three fecal specimens must be assayed because normal cats occasionally pass feces with low activity. Since proteolytic activity in feces is relatively labile, samples need to be stored frozen prior to transit to the laboratory by express delivery.[14, 15]

A recent study has evaluated the use of serum feline trypsin-like immunoreactivity in the diagnosis of EPI in the cat.[9] A total of 11 cats with a significantly decreased serum fTLI (≤8 µg/L) were identified and all 11 cats had severe classical clinical signs of EPI. All but 2 cats responded favorably to therapy with pancreatic enzyme replacement. These remaining two cats had marked reductions in acinar tissue on direct examination of the pancreas. It was therefore concluded that all 11 cats had EPI. As with the diagnosis of pancreatitis, further studies are needed to evaluate the sensitivity of fTLI in the diagnosis of feline EPI. However, the specificity appears to be high from this study.

The same study evaluated serum cobalamin and serum folate concentrations in these cats with EPI. The serum cobalamin concentrations were markedly subnormal in most cats affected with EPI.[3] Ten of the 11 cats had undetectable serum cobalamin concentrations (normal range at the GI laboratory at Purdue University: 200-1680 ng/L; detection limit 27 ng/L), while the last cat had a significantly decreased serum cobalamin concentration (127.5 ng/L). In contrast to dogs with EPI, none of these cats had an elevated serum folate concentration; however, 6 cats had subnormal serum folate concentrations indicating associated small intestinal dysfunction.

Treatment

Most cats with EPI can be managed by supplementing each meal with an initial dose of 1 teaspoon of powdered non-enteric-coated pancreatic extract. Parenteral cobalamin administration (initially 250 µg subcutaneous every 7 days for 1 to 2 months) will usually normalize serum cobalamin concentration. If the response is suboptimal, instituting antibiotic therapy for possible small intestinal bacterial overgrowth, glucocorticoid therapy for possible associated small intestinal mucosal disease, dietary modification, and inhibition of gastric acid output may be helpful. ■

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(Continued on page 8)

Research Briefs

Estrous Length, Pregnancy Rate, Gestation and Parturition Lengths, Litter Size, and Juvenile Mortality in the Domestic Cat

(Authors: M. V. Root, S. D. Johnston, P. N. Olson)—Reproductive performance in a feline research colony of 14 queens was reported. Average estrous length in 38 cycles was 5.8±3.3 days, with a range of two to 19 days. Estrous length in 23 bred cycles was not shorter (p greater than 0.05) than in 15 nonbred cycles, suggesting that induction of ovulation does not decrease estrous length. Pregnancy rate in 23 bred cycles was 73.9%. Gestation length averaged 66.9+ 2.9 days with a range of 62 to 71 days (n=15). Average parturition length was 16.1± 14.3 hours (n=7), with a range of four to 42 hours. Litter size ranged from one to five kittens, with an average of 3.7 kittens per litter (n=15). Percent mortality by eight weeks of age was 29.1%, with 4.7% stillbirths.—*(Resource: J Amer Anim Hosp Assn 1995)*

A Review of the Clinical Diagnosis of Feline Infectious Peritonitis Viral Meningoencephalomyelitis

(Authors: M. Baroni, Y. Heinold)—A study was carried out on 19 cats which had been primarily presented for neurological signs caused by the feline infectious peritonitis (FIP) virus. Seven animals only showed central nervous system (CNS) involvement without symptoms of a systemic disease. The main neurological signs were depression, tetraparesis, head tilt, nystagmus, and intention tremor. Neuroanatomical localization was multifocal in the majority of the cases, with brain stem involvement in all but one. Blood biochemical changes mainly consisted of total protein elevation and hypergammaglobulinemia. Serum antibodies against FIP virus, detected through indirect fluorescence procedure, showed high titer in only 58% of the animals tested. The test was negative in two cats. CSF analysis was consistently characterized by a high protein content and marked pleocytosis which primarily consisted of neutrophils. We concluded that biochemical blood changes and antibody testing have a limited value in diagnosing the neurological form of FIP, while CSF analysis is essential for a correct diagnosis.—(Re*source: Prog Vet Neur 6:3, 1995)*

Measurement of Serum Bile Acids Concentration for Diagnosis of Hepatobiliary Disease in Cats

(Authors: S. A. Center, H. N. Erb, S. A. Joseph)—Serum bile acid concentrations were measured after food had been withheld for 12 hours (fasting serum bile acid [FSBA] concentration) and 2 hours after a meal (post-prandial serum bile acid [PSBA] concentration) using a direct enzymatic procedure in 108 cats clinically suspected of having hepatobiliary disease. In all cats, liver tissue was examined histologically to confirm the diagnosis. Twenty-six cats did not have histologic evidence of hepatobiliary disease and served as controls. The remaining 82 cats had hepatobiliary disease including hepatic lipidosis (n=20), portosystemic vascular anomaly (n=24), hepatic necrosis (n=13), hepatic neoplasia (n=8), or cholestatic hepatic disease (n=17). Sensitivity and specificity of measuring FSBA and PSBA concentrations were calculated for each test alone and when results were interpreted in combination (i.e., in series and in parallel), and were compared with sensitivity and specificity of routinely used serum biochemical tests, including measuring serum activities of alanine phosphatase, and y-glutamyltransferase, and measuring serum concentration of cholesterol, BUN, and total bilirubin. *(Continued on next page)*

When tests were considered individually, determination of FSBA and PSBA concentrations had higher specificity than did the other tests (using a cutoff of 15 µmol/L for FSBA concentration and of 20 µmol/L for PSBA concentration). Determination of PSBA concentration had the highest sensitivity of all single tests in cats with hepatic lipidosis, portosystemic vascular anomaly, or cholestasis; determination of alanine aminotransferase activity or PSBA concentration had the highest sensitivity for cats with hepatic neoplasia; and determination of aspartate aminotransferase activity had the highest sensitivity for cats with hepatic necrosis. For all cats with hepatobiliary disease, determination of PSBA concentration had the highest sensitivity of any single test.

Combination testing using results of measuring total serum bile acid concentrations in conjunction with results of measuring serum enzyme activity or total bilirubin concentration gave the best overall test performance. Results indicate that both the FSBA and PSBA concentration should be determined, but that if paired samples cannot be collected, the PSBA concentration should be preferentially measure. In a small number of cats (6/108), the FSBA concentration exceeded the PSBA concentration.—*(Resource: JAVMA 207 (8):1048, 1995)*

Other Research Articles of Interest:

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Cornell Appoints New Dean of Veterinary College

Dr. Franklin M. Loew, former dean of Tufts University School of Veterinary Medicine, was appointed dean of Cornell's College of Veterinary Medicine this past summer. He began his new duties on September 1. Dr. Loew is an alumni of Cornell University (D.V.M. , class of 1965). He succeeds Dr. Robert D. Phemister (D.V.M., class of 1960) who has been the college's dean since 1985.

Dr. Loew is an internationally known lecturer and writer on animals and society. He has addressed important issues facing the veterinary profession, including the use of animals in research, the changing moral status of animals, and how Americans and Europeans view domestic animals and wildlife.

Dr. Loew returned to Cornell earlier this summer to celebrate his thirtieth reunion with the DVM class of 1965. In an address to alumni he stated that, "Deans are not the people who make institutions great. It begins with the faculty, staff, and students and encompasses their work in both service and scholarship here on campus. Then our graduates go out into the world and it is their dedication and commitment to animals and the people who care for them that continually reinforces Cornell's reputation for excellence in veterinary medicine." ■

Solving Feline Behavior Problems

May 11–12, 1996 Ithaca, New York

Program

This intensive course is designed for veterinary practice staff members, cat breeders, boarding facility owners, and other people with a serious interest in cats. It will be taught by faculty and staff members of the Cornell University College of Veterinary Medicine.

Topics to be covered include:

- **Behavioral History**
- **The Cat Brain and Drug Therapy**
- **Feline Communication and Spraying**
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- **Feline Social Structure**
- **Aggression**
- **Feeding and Pica**
- **Sleep, Sex, and Maternal Behavior**
- **Development and Temperament Testing**
- **Recent Advances in Feline Medicine**

Faculty

Program instructors are faculty and staff members of the College of Veterinary Medicine, Cornell University:
Dr. Katherine A. Houpt, Director of the Animal Behavior Clinic and Professor of Veterinary Physiology;
Dr. Soraya Juarbe-Díaz, Resident in Behavior Medicine at the Veterinary Medical Teaching Hospital;
Dr. Ilana R. Reisner, Resident in Behavior Medicine in the Animal Behavior Clinic;
Dr. John E. Saidla, Director of Continuing Education; Chief of Dental Services at the Veterinary Medical Teaching Hospital.

Accommodations

Rooms have been reserved at the following locations: Best Western University Inn, (607) 272-6100, $60 single/double; Ramada Inn, (607) 257-3100, $65 single/double; Sheraton Inn, (607) 257-2000, $79 single/double. The above reduced rates are available if, when you make your reservations, you mention the program by name.

Program Charge

The program charge is $285 and includes tuition; course materials; a formal Cornell University certificate of completion; continental breakfasts on Saturday and Sunday; lunch and dinner on Saturday; lunch on Sunday; and refreshment breaks. Persons whose cancellations are received in writing by April 26 will receive a full refund. Cancellations received after April 26 are subject to a $100 cancellation fee. Substitutions may be made at any time before the program begins. Program costs may be tax deductible.

Travel Planning

Participants should arrive by 8:30 a.m., Saturday, May 11. The program will conclude by 4:00 p.m. on Sunday, May 12.

Further Information

Solving Feline Behavior Problems, Cornell University, B20 Day Hall, Ithaca, NY 14853-2801; phone (607) 255-7259; fax: (607) 255-8942; e-mail: sp@sce.cornell.edu.

Registration Form

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Feline Exocrine Pancreatic Disease *(continued from page 4)*

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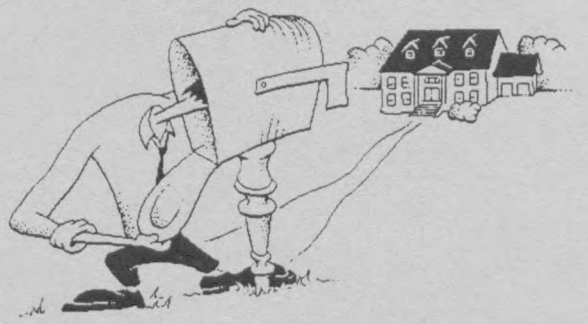

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