Underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis: 51 cases (1990–2001)

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Objective—To determine the underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis and identify differences between cats that survived following treatment and cats that did not survive despite treatment.

Design—Retrospective study.

Animals—51 cats with septic peritonitis.

Procedure—Medical records were reviewed for clinical findings; results of clinicopathologic testing, microbial culture, and radiography; diagnosis; treatment; and outcome.

Results—Signs of pain during palpation of the abdomen were reported for only 29 of 47 (62%) cats. Eight (16%) cats had relative bradycardia (heart rate < 140 beats/min). The most commonly isolated organisms included *Escherichia coli*, *Enterococcus* spp, and *Clostridium* spp. The most common cause of peritonitis was gastrointestinal tract leakage (24 cats). No definitive source could be identified in 7 cats. Treatment, including exploratory surgery, was pursued in 23 cats, of which 16 (70%) survived and were discharged. There were no significant differences between survivors and nonsurvivors in regard to heart rate, age, rectal temperature, serum lactate concentration, WBC count, PCV, blood glucose concentration, or serum albumin concentration.

Conclusions and Clinical Relevance—Results suggest that clinicopathologic abnormalities and outcome in cats with septic peritonitis are similar to those reported for dogs. However, certain features may be unique, including an absence of signs of pain during abdominal palpation, relative bradycardia, and apparent spontaneous peritonitis in some cats. (*J Am Vet Med Assoc* 2004;225:897–902)

Septic peritonitis is a life-threatening clinical condition that requires prompt recognition and aggressive medical and surgical treatment. Potential sequelae of septic peritonitis include hypovolemia, electrolyte and acid-base alterations, **systemic inflammatory response syndrome (SIRS)**, sepsis, and septic shock.

Although several studies[1-9] have described clinical signs of septic peritonitis in dogs and outcomes associated with various treatments, there is little information in the literature concerning septic peritonitis in cats. In addition, many studies[1,3-5,8,9] that do include cats combine data for cats and dogs, providing little specific data about cats.

Our clinical experience as well as results of a recent study[10] of cats with severe sepsis suggests that clinicopathologic responses to a septic insult in cats are different from responses in other species. For example, while dogs typically develop tachycardia in response to septic shock, cats with sepsis or SIRS have a varied cardiovascular response and may develop bradycardia. Thus, it may be inappropriate to make clinical decisions about cats with septic peritonitis on the basis of data from studies involving dogs.

The purposes of the study reported here, therefore, were to determine the underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis; identify differences between cats that survived following treatment and cats that did not survive despite treatment; and determine whether cats with septic peritonitis fulfilled proposed criteria for the diagnosis of SIRS in cats.[10]

Criteria for Selection of Cases

Medical records of the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania were searched to identify cats examined between 1990 and 2001 for which a diagnosis of peritonitis had been made. Additionally, the Department of Pathobiology database for the same period was searched to identify cats for which a necropsy diagnosis of septic peritonitis had been made. Records were reviewed, and cats were included in the study only if intracellular bacteria had been seen during cytologic evaluation of peritoneal fluid, microbial culture of peritoneal fluid had yielded bacterial growth, or a necropsy diagnosis of septic peritonitis had been made. Cats for which medical records were incomplete were excluded, along with cats with aseptic peritonitis (eg, cats with feline infectious peritonitis or bile peritonitis).

Procedures

Data extracted from the medical records included signalment, indoor-outdoor status, physical examination abnormalities, clinicopathologic data, radiographic findings, cytopathologic findings, results of microbial culture, definitive diagnosis, treatment, clinical course, and outcome. Information recorded in the medical record prior to surgery and within 24 hours of the diagnosis of septic peritonitis was used.

Cats included in the study were considered to have SIRS if they met 3 or more of the following criteria: rec-

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tal temperature > 39.7°C (103.5°F) or < 37.8°C (100°F), heart rate > 225 or < 140 beats/min, respiratory rate > 40 breaths/min, and WBC count > 19,500 or < 5,000 cells/µL or band neutrophil fraction > 5%.

Cats were considered to have survived if treatment was attempted and the cat survived long enough to be discharged from the hospital. Cats were considered to have not survived if treatment was attempted but the cat died or was euthanatized without being discharged. Cats in which treatment was not attempted were excluded from analyses of factors associated with outcome.

Statistical analyses—For continuous variables, the Shapiro-Wilk or skewness-kurtosis test was used to determine whether data were normally distributed. Mean and SD were reported for continuous variables that were normally distributed; median and range were reported for continuous variables that were not normally distributed. Unpaired Student *t* or Wilcoxon rank sum tests (depending on data distribution) were used to compare continuous variables between survivors and nonsurvivors. The Fisher exact test was used to compare proportions between groups. For all analyses, values of $P < 0.05$ were considered significant. Standard statistical software[a] was used for all analyses.

Results

The initial records search yielded records for 253 cats for which a diagnosis of peritonitis had been made during the study period. Of these, 51 cats met the criteria for inclusion in the study. For 49 of the 51 cats, the diagnosis was made and data were collected on the first day of hospitalization. The remaining 2 cats developed septic peritonitis while hospitalized, and for these cats, data were collected from the morning of the day on which septic peritonitis was diagnosed.

Median age of the 51 cats included in the study was 5 years (range, 7 days to 19 years). Thirty (59%) were castrated males, 12 (23%) were spayed females, 6 (12%) were sexually intact males, and 3 (6%) were sexually intact females. Forty-one (80%) were domestic shorthair cats, 5 (10%) were domestic longhair cats, and 5 (10%) were purebred cats. Thirty-eight (74%) lived strictly indoors, 12 (24%) lived indoors and outdoors, and 1 (2%) lived strictly outdoors.

Median heart rate was 192 beats/min (range, 100 to 256 beats/min); 8 cats (16%) had a heart rate < 140 beats/min. Median respiratory rate was 40 breaths/min (range, 18 to 120 breaths/min). Median rectal temperature was 38.1°C (100.6°F; range, 32.4° to 42.2°C [90.4° to 108°F]). Signs of pain during palpation of the abdomen were reported for 29 of 47 (62%) cats. No other pertinent physical examination abnormalities were recorded.

A CBC was performed in 36 of the 51 cats. Median WBC count was 14,600 cells/µL (range, 1,500 to 64,000 cells/µL; reference range, 5,500 to 19,500 cells/µL). Band neutrophilia was identified in 23 of 36 (64%) cats; median band neutrophil count was 240 cells/µL (range, 0 to 18,000 cells/µL). Toxic WBC changes were reported for 13 (36%) cats and were designated as mild in 3 cats, moderate in 5, and marked in 5. Anemia was found in 16 of 46 (35%) cats; median PCV was 32% (range, 9% to 63%; reference range, 27% to 45%).

Venous pH was measured in 34 of the 51 cats. Median pH was 7.33 (range, 7.08 to 7.51; reference range, 7.36 to 7.47). Acidemia (pH < 7.36) was identified in 21 of the 34 cats, and alkalemia (pH > 7.47) was identified in 2. Serum lactate concentration was measured in 33 of the 51 cats. Median lactate concentration was 2.9 mmol/L (range, 0.5 to 11.3 mmol/L; reference range, 0.6 to 2.5 mmol/L). Hyperlactatemia was identified in 21 of 33 (64%) cats.

Serum albumin concentration was measured in 37 of the 51 cats. Mean ± SD serum albumin concentration was 2.14 ± 0.67 g/dL (reference range, 2.7 to 3.9 g/dL). Thirty (81%) cats had hypoalbuminemia. Median blood glucose concentration (n = 46 cats) was 125 mg/dL (range, 25 to 494 mg/dL; reference range, 75 to 199 mg/dL). Hypoglycemia was identified in 10 cats, and hyperglycemia was identified in 6. Serum ionized calcium concentration was measured in 34 cats and was low in 20 (59%). Mean ± SD serum ionized calcium concentration was 1.09 ± 0.11 mmol/L (reference range, 1.13 to 1.33 mmol/L). Median total bilirubin concentration (n = 36) was 0.5 mg/dL (range, 0.1 to 9.9 mg/dL; reference range, 0.1 to 0.5 mg/dL). Hyperbilirubinemia was identified in 16 of 36 (44%) cats. Serum **alanine transaminase (ALT)** activity was high in 10 of the 36 (28%) cats in which it was measured. Median serum ALT activity was 65 U/L (range, 13 to 912 U/L; reference range, 20 to 107 U/L). Median serum **alkaline phosphatase (ALP)** activity (n = 36) was 29.5 U/L (range, 10 to 246 U/L; reference range, 23 to 107 U/L). Serum **γ-glutamyltransferase (GGT)** activity was measured in 27 cats and was high in 17. Median serum GGT activity was 11 U/L (range, 5 to 26 U/L; reference range, 1 to 5 U/L).

Abdominal radiographs were obtained in 27 of the 51 cats. Loss of serosal detail attributed to a peritoneal effusion was identified in 19 cats, and pneumoperitoneum was visible on radiographs of 10 cats. Pneumoperitoneum was associated with rupture of an abdominal viscus (n = 6), penetrating abdominal trauma (3), and rupture of the bladder following perineal urethrostomy surgery (1). Six cats had a mass effect on radiographs, and 4 had an obstructive gastrointestinal tract pattern. Abdominal ultrasonography was performed in 21 cats, and although results were used as an adjunct to results of other diagnostic tests, in no cats were results of abdominal ultrasonography directly used in the diagnosis of septic peritonitis.

Peritoneal fluid from 31 of the 51 cats was examined cytologically. Intracellular bacteria were identified cytologically in 28 of the 31 (90%) cats. In the remaining 3 cats, suppurative inflammation was seen with degenerate neutrophils but without bacteria. Degenerate neutrophils were characterized by marked cytoplasmic swelling and karyolysis.

Peritoneal fluid from 31 cats was submitted for bacterial culture. Organisms identified included *Escherichia coli* (n = 17); *Enterococcus* spp (14); *Clostridium* spp (9); *Pseudomonas* spp (2); *Acinetobacter* spp (2); and coagulase-negative *Staphylococcus* spp, *Enterobacter* spp, α-

hemolytic *Streptococcus* spp, *Pasteurella multocida*, and *Proteus* spp (1 each). Polymicrobial infection was identified in 17 of the 31 (55%) cats.

Identification of intracellular bacteria in peritoneal fluid was the most common method by which a definitive diagnosis of septic peritonitis was made (n = 29). In 11 cats, the diagnosis was made at necropsy, and in 8, the diagnosis was made during exploratory surgery. In the remaining 3 cats, the diagnosis was made on the basis of pneumoperitoneum on abdominal radiographs without any history of abdominal surgery.

In 24 of the 51 (47%) cats, septic peritonitis was a result of leakage from the gastrointestinal tract. Gastrointestinal tract leakage occurred because of neoplasia (n = 13 [adenocarcinoma, 6; lymphosarcoma, 5; lymphohistiocytic lymphoma, 1; and undetermined neoplasia causing a jejunal mass, 1]), leakage from a previous surgery site (3), perforation of megacolon (2), gastric necrosis (2), perforation of the bowel secondary to a linear foreign body (1), gastric rupture (1), ileal perforation (1), and severe colitis with colonic rupture (1). In 8 cats, septic peritonitis developed secondary to trauma, including bite wounds (5), gunshot wounds (2), and vehicular trauma (1). In 4 cats, the urinary tract was the source of contamination resulting in septic peritonitis. Two of these cats developed septic peritonitis following urinary bladder rupture; 1 of these cats had undergone perineal urethrostomy 5 months earlier. Histologic examination of biopsy specimens from the urinary bladder of 1 cat revealed transmural inflammation, suggesting that septic peritonitis resulted from leakage of urine into the peritoneal cavity. *Enterococcus* organisms were isolated from urine and a peritoneal fluid sample. The fourth cat was being treated for pyelonephritis, and *Escherichia coli* was isolated from urinary bladder and peritoneal fluid samples; at necropsy, no source of leakage was found. In 3 cats, septic peritonitis developed secondary to septic pancreatitis or a pancreatic abscess. In 2 cats, septic peritonitis was a result of uterine rupture secondary to pyometra, and in 1 cat, septic peritonitis developed secondary to severe omphalitis. In 1 cat, septic peritonitis resulted from liver lobe necrosis, and in 1 cat, septic peritonitis was associated with pyogranulomatous disease with gram-positive filamentous bacteria found at necropsy. In the remaining 7 (14%) cats, the cause of the septic peritonitis was not identified during surgery or necropsy.

Rectal temperature was > 39.7°C or < 37.8°C in 33 cats, heart rate was > 225 or < 140 beats/min in 13 cats, respiratory rate was > 40 breaths/min in 24 cats, and the WBC count was > 19,500 or < 5,000 cells/μL or the band neutrophil fraction was > 5% in 25 cats. Eight cats were considered to have SIRS on the basis of having 3 or more of these criteria.

Median hospitalization time was 2 days (range, 0 to 13 days). Treatment was not pursued in 28 cats. Of these, 11 were euthanatized at the time of diagnosis of septic peritonitis. Eight cats were euthanatized and 1 died before the diagnosis of septic peritonitis was made, and the diagnosis was made at necropsy. In the remaining 8 cats, treatment was initially attempted, but the cats were euthanatized at the time of exploratory surgery because of the extent of disease.

Treatment, including exploratory surgery, was pursued in the remaining 23 cats. In all 23 cats, attempts were made during surgery to identify and treat the underlying cause. Seventeen cats were managed by means of abdominal lavage and primary closure. The remaining 6 cats were managed with open abdominal drainage. All of the treated cats received routine postoperative care consisting of IV administration of fluids and antimicrobials, intensive monitoring, and nutritional support. Various antimicrobials were used, including ampicillin (n = 18); enrofloxacin (10); metronidazole (9); cefazolin (5); cefoxitin (4); amikacin (2); gentamicin (2); and imipenem and cilastatin, doxycycline, clindamycin, and ciprofloxacin (1 each). Median duration of hospitalization for treated cats was 5 days (range, 1 to 13 days). Sixteen of the 23 (70%) cats in which treatment was attempted survived long enough to be discharged from the hospital. Three of the 6 cats that were treated with open abdominal drainage and 13 of the 17 cats treated with primary closure survived.

There were no significant differences between cats that survived and cats that did not survive despite treatment in regard to preoperative heart rate, age, rectal temperature, venous pH, serum lactate concentration, WBC count, band neutrophil fraction, PCV, serum ionized calcium concentration, serum albumin concentration, serum total bilirubin concentration, serum GGT activity, or serum ALP activity. There were also no significant differences between survivors and nonsurvivors in regard to cause of septic peritonitis or whether infection was polymicrobial. In addition, there were no significant differences in survival rates among cats that fulfilled 2, 3, or 4 of the criteria for a diagnosis of SIRS. Median serum ALT activity was significantly (P = 0.016) lower in cats that survived (median, 55.5 U/L; range, 15 to 176 U/L) than in cats that died (median, 179 U/L; range, 68 to 269 U/L). Mean ± SD hospitalization time for cats that survived (6.3 ± 2.8 days) was significantly (P = 0.037) longer than mean hospitalization time for cats that did not survive (3.6 ± 2.2 days).

Discussion

Results of the present study suggest that clinicopathologic abnormalities and outcome in cats with septic peritonitis are similar to those reported for dogs. However, certain features were unique, including the findings that only 29 of 47 (62%) cats had signs of pain during abdominal palpation and that 8 of 51 (16%) cats had bradycardia. In addition, in 7 (14%) cats, septic peritonitis appeared to be spontaneous because an underlying cause could not be identified.

Median heart rate of cats in the present study was 192 beats/min, but 8 cats (16%) had heart rates < 140 beats/min. Similarly, in a previous study,[10] 19 of 29 (66%) cats with severe sepsis had relative bradycardia, that is, a heart rate that was inappropriately low for the hemodynamic status of the patient. The mechanism of this inappropriate bradycardia is unclear, but it has been postulated to be secondary to increased vagal tone or cytokine-associated myocardial depression.[10-13] Treatment was not attempted in any of the 8 cats with bradycardia in the present study.

Surprisingly, although all cats in the present study had septic peritonitis, only 29 of 47 (62%) had signs of pain during abdominal palpation. Thus, results of physical examination can be deceptive in cats with septic peritonitis, and an absence of signs of pain during abdominal palpation should not be used to rule out septic effusion.

Median WBC count for cats in the present study was lower than values reported in previous studies.[2,5,6,8] Importantly, a CBC was not performed in all cats in the present study, and it is possible that some of the most severely affected cats were euthanatized before a CBC could be performed. For this reason, the median WBC count may have been artificially low.

Sixteen of 46 (35%) cats in the present study were anemic, and anemia has previously been reported to be a common clinicopathologic abnormality in cats with severe sepsis or septic peritonitis.[3,10] The pathogenesis of anemia associated with sepsis and critical illness in cats is likely complex. Factors that have been proposed to play a role in humans include frequent blood sampling, nutritional deficiencies, gastrointestinal tract blood loss, mechanical and antibody-mediated hemolysis, renal and hepatic insufficiency, and bone marrow infiltrative disorders.[14,15] In addition, inflammatory mediators such as tumor necrosis factor-α and interleukin-1 inhibit erythroid precursor cells, reduce the formation of erythropoietin, and blunt the response of the bone marrow to erythropoietin.[14-16] Decreased RBC lifespan also likely plays a role in the pathogenesis of anemia of inflammation,[17] and RBCs in cats are particularly susceptible to oxidative damage because the feline hemoglobin molecule contains 8 reactive sulfhydryl groups.[18]

Low serum ionized calcium concentration is common in human patients with sepsis.[19-22] Proposed mechanisms of hypocalcemia include parathyroid gland suppression, inadequate vitamin D concentrations, and parathyroid hormone and vitamin D resistance.[21,22] However, the underlying causes of these changes in patients with sepsis are still under investigation.

Serum glucose concentrations varied widely among cats in the present study. Six cats were hyperglycemic, potentially as a result of a stress response, and 10 were hypoglycemic (< 75 mg/dL). Three of these hypoglycemic cats underwent surgery, and 1 survived and was discharged from the hospital. Although blood glucose concentrations were not significantly different between survivors and nonsurvivors in the present study, abnormalities (increases and decreases) in blood glucose concentrations have been associated with higher mortality rates among dogs with postoperative sepsis.[23] In dogs, alterations in blood glucose concentration associated with acute sepsis include transient hyperglycemia followed by hypoglycemia.[24,25] Multiple factors are thought to play a role in the development of hypoglycemia, including increased peripheral glucose use, impaired gluconeogenesis, and depleted glycogen stores.[26]

Hypoalbuminemia is common in animals with septic peritonitis and has been reported as a complication of open peritoneal drainage in dogs and cats.[5,8,23] It is also common in cats with severe sepsis.[10] Hypoalbuminemia may be caused by increased losses, malnutrition, hepatic dysfunction, increased capillary permeability, and shifting of hepatic synthetic pathways away from albumin production and towards production of acute phase proteins.[10,23] Hypoalbuminemia can lead to peripheral edema but has not been associated with decreased survival rates in animals with peritonitis.[5] Interestingly, although serum albumin concentration was low in 30 of the 37 (81%) cats in which it was measured, peripheral edema was not identified in any cats.

Hepatic dysfunction can be a serious sequela of sepsis and septic peritonitis. Hepatosplanchnic perfusion is impaired in septic shock, which leads to decreased oxygen and nutrient delivery to the liver.[27] High serum ALT and GGT activities have been associated with high mortality rates in dogs with septic peritonitis,[2] and in the present study, median serum ALT activity was significantly higher in cats that died despite treatment than in cats that survived. However, the difference in median ALT activities between groups was not clinically relevant.

Peritonitis is associated with the release of vasoactive substances such as histamine, serotonin, cellular proteases, and endotoxins, which ultimately leads to increased capillary permeability and vasodilation.[1,28] This inflammation, in turn, leads to loss of isotonic fluid into the abdominal cavity. Of 27 cats in the present study in which abdominal radiography was performed, however, only 19 had radiographic evidence of peritoneal effusion. An effusion may not be visible on radiographs if the fluid is loculated or if a small volume is present.

Pneumoperitoneum was found radiographically in 10 cats. In animals without any history of recent surgery or abdominocentesis, free air in the abdomen is an indication for immediate exploratory surgery. Septic peritonitis associated with neoplasia is well documented.[2,3,5,6,9] In the present study, a mass effect was seen on abdominal radiographs from 6 cats, and in 4 of these 6 cats, an intestinal mass was discovered at surgery.

Cytologic examination of peritoneal fluid is an excellent tool for making a diagnosis of septic peritoneal effusion. Types of bacteria identified may provide a basis for initial antimicrobial treatment, and detection of neoplastic cells can provide insight into the underlying cause. Although intracellular bacteria were identified cytologically in peritoneal fluid from 28 of 31 (90%) cats in the present study, 3 (10%) cats had only degenerate neutrophils without visible bacteria. In 2 of these cats, microbial culture of the peritoneal fluid yielded bacterial growth, and in the third, intracellular bacteria were seen in peritoneal fluid collected at necropsy. In a recent study[3] evaluating the use of closed suction drains to treat generalized peritonitis, 9 of 21 dogs and cats had neutrophils without evidence of intracellular bacteria during cytologic evaluation of peritoneal fluid, but microbial culture of peritoneal fluid from 6 of the 9 yielded bacterial growth. In another study,[6] 2 of 15 dogs did not have bacteria identified during preoperative cytologic examination of peritoneal fluid, but septic peritonitis was discovered at surgery. Finally, in a study[5] of dogs and cats with septic peritonitis, 2 of 13 animals had degenerate neutrophils

in peritoneal fluid with no evidence of bacteria. Thus, degenerate neutrophils or evidence of chronic inflammation, even without intracellular bacteria, may be an indication for further diagnostic testing or exploratory laparotomy.

Most of the organisms isolated from cats in the present study were gram-negative enteric organisms, which is consistent with results of previous studies.[2-5,8,9] *Clostridium* organisms were isolated from 9 of 31 (29%) cats; therefore, initial empiric antimicrobial treatment should probably include drugs effective against anaerobes. Additionally, *Enterococcus* spp tend to be resistant to a number of common antimicrobials, including fluoroquinolones and aminoglycosides. Polymicrobial infections have previously been associated with higher mortality rates in people and animals with septic peritonitis,[3,29] possibly because of a synergistic action between endotoxin-producing facultative anaerobes such as *E coli* and obligate anaerobes such as *Bacteroides* spp. Polymicrobial infection was identified in 17 of 31 (55%) cats in the present study but was not associated with a worse outcome.

The most common cause of septic peritonitis in the present study was gastrointestinal tract leakage, which was consistent with findings of previous studies[1-7,9] of septic peritonitis in dogs and cats. Gastrointestinal tract leakage was most commonly a result of neoplasia, and of the 13 cats with neoplasia in the present study, 2 were euthanatized at the time of diagnosis of septic peritonitis, 2 were euthanatized prior to diagnosis of septic peritonitis, and 5 were euthanatized at the time of surgery because of the severity of disease. Of the 4 cats with neoplastic septic peritonitis in which surgical treatment was attempted, 3 survived to discharge.

The urinary tract appeared to be the source of contamination in 4 cats in the present study. One cat had histologic evidence of transmural inflammation, suggesting that bacteria may have migrated across the inflamed bladder wall and colonized the abdomen. One cat had pyelonephritis, and although *E coli* was isolated from the urinary bladder and peritoneal fluid, no source for leakage from the urinary tract was identified at necropsy. This cat may have developed bacteremia secondary to pyelonephritis, with septic peritonitis a result of hematogenous spread.

The pancreas was the source of bacteria in 3 cats in the present study and in 1 cat in a previous study.[4] In cats, the pancreatic and bile ducts share a single opening into the duodenum through the major duodenal papilla. In addition, the accessory pancreatic duct generally does not persist, so that approximately 80% of cats have only 1 pancreatic duct.[30,31] Cats have a higher number of bacteria in the proximal portion of the small intestine than do dogs[31,32]; therefore, duodenal reflux into the pancreatic duct may have more severe consequences in cats than in dogs. In cats with experimentally induced pancreatitis, bacteria were shown to colonize the pancreas from multiple sources, including the colon.[33] Treatment was attempted in 2 of the cats with pancreatitis in the present study, but the third cat was euthanatized before the diagnosis was made. Of the 2 cats in which treatment was pursued, 1 died and 1 was euthanatized.

In 7 cats in the present study, the cause of septic peritonitis was not identified during surgery or necropsy. To our knowledge, there are no reports of spontaneous peritonitis in dogs, although a recent report[3] of dogs and cats with peritonitis described a cat in which the source of bacterial infection could not be identified at surgery. Treatment was attempted in 4 of the 7 cats with no obvious cause of peritonitis in the present study, and 3 of the 4 survived and were discharged from the hospital. The fourth developed a recurrence of septic peritonitis 3 days after the initial surgery. Exploratory surgery was repeated, but the cat died 6 hours after surgery.

In humans, bacterial peritonitis is classified as spontaneous, primary, or secondary.[29,34] Spontaneous bacterial peritonitis is defined as diffuse bacterial peritonitis with no obvious primary focus of infection or disruption of intra-abdominal hollow viscera.[29,34] It is thought that spontaneous bacterial peritonitis in humans occurs by hematogenous spread and may respond to medical treatment.[34] In contrast, primary and secondary peritonitis mandate immediate medical and surgical treatment.[29] Even if spontaneous peritonitis is suspected in a cat with septic peritonitis, extensive efforts must be made to ensure that there is no ongoing leakage in the abdominal cavity.

Previously reported mortality rates for dogs and cats with septic peritonitis range from 20% to 48% for animals undergoing open abdominal drainage[2,4,7-9] and from 33% to 46% for animals undergoing lavage and primary closure.[6,8] Mortality rate was 30% for animals in 1 study[3] in which closed suction drains were used. In the present study, the mortality rate for cats in which treatment was attempted was 30% (7/23). The small number of cats in each group made it impossible to compare mortality rates for cats undergoing open abdominal drainage with rates for cats treated with primary closure, but in a study[8] comparing the outcome of dogs and cats treated with open peritoneal drainage versus primary closure, there was no significant difference in survival between groups. Unfortunately, no specific data on the outcome of cats were provided in that study.

The term SIRS refers to a generalized inflammatory response to a severe localized inflammatory process. Progression to a generalized inflammatory response may be a result of loss of local control of inflammation or excessive activation of systemic inflammatory mediator and cytokine responses. A diagnosis of SIRS implies some degree of endothelial injury and disruption, and SIRS is frequently thought to indicate imminent risk of septic shock. However, this term remains a purely clinical diagnosis, as there is no clinical test that can determine the extent to which a localized inflammatory process has become generalized.

In the present study, all cats had a severe inflammatory process involving the abdomen, and it could therefore be argued that all had some degree of activation of inflammatory mediators. Thus, clinical criteria for the diagnosis of SIRS simply represent a means by which clinicians may be alerted to the most severely affected patients. The criteria for diagnosis of SIRS in the present study were based on recommendations

from a previous study[10] of cats with severe sepsis. The authors of that study proposed that a tentative clinical diagnosis of SIRS might be made in cats that fulfilled at least 3 of the 4 criteria. It is important to remember that these criteria were developed on the basis of findings for cats in which a diagnosis of severe sepsis was made at necropsy, and prospective studies to determine the sensitivity and specificity of these criteria have yet to be performed. In dogs, similar criteria were found to be a sensitive, but nonspecific, tool for the diagnosis of sepsis,[35] and similar criteria are routinely used for the diagnosis of SIRS in human patients.[36] Further studies are needed to investigate the sensitivity and specificity of these criteria in cats. In the present study, 8 cats were considered to have SIRS on the basis of having 3 or more of the specified criteria, but there was no significant difference in survival rates between cats with and without SIRS.

There were a number of limitations to the present study. In particular, the retrospective nature and small number of cases limit the study's utility. In addition, by excluding cats that were euthanatized without undergoing treatment from analyses of outcome, we may have had a bias towards less severely affected cats. Further prospective studies would be helpful to examine these factors.

[a]Intercooled Stata 7.0 for Windows, StataCorp LP, College Station, Tex.

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