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CHAPTER 122
PERITONITIS

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KEY POINTS

- Peritonitis is inflammation of the peritoneal cavity and is most commonly the result of gastrointestinal rupture, perforation, or dehiscence in small animals.
- Clinical signs in patients with peritonitis may be mild to severe and are often nonspecific.
- Abdominocentesis is the preferred diagnostic method for confirming peritonitis.
- When abdominal fluid cytology reveals degenerative neutrophils and intracellular bacteria, confirming a diagnosis of septic peritonitis, emergency surgical exploration of the abdomen is indicated.
- Open peritoneal drainage or closed suction drainage should be considered for management of septic peritonitis in which the source of contamination cannot be controlled completely, or if significant contamination or inflammation remains after surgical debridement and lavage.
- Prognosis is guarded for patients with peritonitis. Reported survival rates are highly variable and depend on the cause, presence of infection, and development of systemic inflammatory response syndrome and/or organ dysfunction.

Peritonitis is defined as inflammation of the peritoneal cavity and may be classified according to the underlying cause (primary or secondary), extent (localized or generalized), or the presence of infectious agents (septic or nonseptic). Primary peritonitis refers to a spontaneous inflammatory condition in the absence of underlying intraabdominal pathology or known history of penetrating peritoneal injury. Secondary peritonitis occurs more commonly in the dog and cat and is the consequence of a preexisting aseptic or septic pathologic intraabdominal condition. Because of the multitude of conditions that may lead to peritonitis the types of clinical signs and their severity vary.

Hematogenous dissemination of infectious agents has been postulated as the mechanism of development of primary peritonitis and likely is facilitated by impaired host immune defenses. The most common form of primary peritonitis is the effusive form of feline infectious peritonitis, caused by feline coronavirus, which should be included on any differential diagnosis list for cats with peritoneal effusion. Other infectious agents reported to cause primary peritonitis in dogs and cats include *Salmonella typhimurium, Chlamydia psittaci, Clostridium limosum, Mesocestoides* spp., *Bacteroides* spp., *Actinomyces* spp., *Blastomyces* spp., and *Candida* spp. Given the common occurrence of isolated *Bacteroides* and *Fusobacterium* spp. from cats with primary septic peritonitis, these bacteria may be translocating from the oral cavity through either unrecognized direct penetration (bites) or a hematogenous route.[1]

Inflammation of the abdominal cavity in the absence of infectious pathogens (aseptic peritonitis) most commonly occurs in response to exposure of the peritoneum to sterile fluids (i.e., gastric, biliary, or urine), pancreatic enzymes, or foreign material. Aseptic bile and urine cause minimal peritoneal inflammation, whereas gastric fluid and pancreatic enzyme leakage lead to a more intense peritoneal reaction. Microscopic and macroscopic foreign material, including surgical glove powder, surgical materials (suture, cotton swabs, surgical sponges), hair, and impaled objects (sticks, plant material, metal) may elicit a granulomatous response. To minimize iatrogenic causes of aseptic peritonitis, surgeons should rinse surgical gloves preoperatively with sterile saline or use powder-free gloves, perform a surgical sponge count before opening and closing a celiotomy, and use surgical sponges with radiopaque markers.

More commonly, secondary peritonitis is identified as a septic process, most commonly secondary to contamination from the gastrointestinal (GI) tract. Leakage of GI contents may occur through stomach and intestinal walls that have been compromised by ulceration, foreign body obstruction, neoplasia, trauma, ischemic damage, or dehiscence of a previous surgical incision. Spontaneous gastroduodenal perforation may be associated with nonsteroidal antiinflammatory drug administration but also may be seen with corticosteroid administration, neoplastic and nonneoplastic GI infiltrative disease, gastrinoma, and hepatic disease.[2,3] Neoplasia was found to be the underlying pathology in 25% of cats with septic peritonitis secondary to GI leakage in one study, with adenocarcinoma and lymphosarcoma the most common types.[4] Septic peritonitis secondary to surgical site dehiscence occurs in 6% to 16% of postoperative patients requiring intestinal enterotomy or resection and anastomosis.[5-8] GI linear foreign bodies in dogs have been reported as the inciting cause of peritonitis in 41% of cases, higher than that previously reported for cats.[9] One canine study found that two or more of the following conditions increased the risk for leakage after intestinal anastomosis: preoperative peritonitis, intestinal foreign body, and a serum albumin concentration of 2.5 g/dl or less.[8] In addition, a recent study suggests that intraoperative hypotension is also a risk factor for the development of septic peritonitis after gastrointestinal surgery.[5] Interestingly, this retrospective of 225 surgeries found the presence of a foreign body to be a protective factor. Other causes of septic peritonitis can be found in Box 122-1.

CLINICAL SIGNS

Historical information may provide clues regarding the underlying cause of peritonitis. Previous and current maladies and surgical procedures (including neutering), current medications (particularly those that may predispose to GI ulceration), and duration of current clinical signs should be investigated. Owners should be questioned specifically regarding the potential for trauma exposure and foreign body ingestion. A history of recent abdominal surgery should raise suspicion for septic peritonitis, particularly if gastrointestinal surgery was performed.

Clinical signs of dogs and cats with peritonitis vary in type and intensity and may reflect the underlying disease process. Peritoneal effusion is a consistent finding but may be difficult to appreciate on physical examination if only a small volume of fluid is present; it also

BOX 122-1 *Differential Diagnoses of Septic Peritonitis in Dogs and Cats*

Primary

Feline coronavirus (feline infectious peritonitis)
Salmonella typhimurium
Chlamydia psittaci
Clostridium limosum
Mesocestoides spp.
Blastomyces spp.
Candidiasis spp.

Secondary

Penetrating abdominal wounds
Surgical peritoneal contamination
Peritoneal dialysis
Gastrointestinal conditions
Gastric rupture secondary to GDV, neoplasia, perforating ulcer
Intestinal leakage
Perforating foreign body, ulcer, or neoplasia
Bacterial translocation secondary to obstruction (foreign body, neoplasia, intussusception, or bowel incarceration)
Dehiscence of intestinal surgical wound
Ischemic intestinal injury
Hepatobiliary condition
Liver abscess
Liver lobe torsion with abscess formation
Ruptured biliary tract with bacterobilia
Pancreatitis or pancreatic abscess
Hemolymphatic conditions
Splenic abscess
Splenic torsion with anaerobic bacterial colonization
Mesenteric lymph node abscess formation
Urogenital conditions
Renal abscess
Septic uroabdomen
Pyometra (ruptured or with mural bacterial translocation)
Uterine torsion
Prostatic abscess formation

GDV, Gastric dilatation-volvulus.

may be difficult to detect sonographically in animals that are dehydrated. Abdominal pain may be appreciated on palpation, with a small number of dogs exhibiting the "prayer position" in an attempt to relieve their abdominal discomfort. Abdominal pain is a less consistent finding in feline peritonitis patients (38% to 62%).[4,10] Most animals with septic peritonitis are systemically ill and exhibit nonspecific clinical signs such as anorexia, vomiting, mental depression, and lethargy. These patients may arrive in progressive states of hypovolemic and cardiovascular shock, with either injected or pale mucous membranes, prolonged capillary refill time, tachycardia with weak pulses, and with either hyperthermia or hypothermia reflecting poor peripheral perfusion. A significant number of cats (16%) with septic peritonitis exhibited bradycardia (see Chapters 6 and 91).[4] In fact, the combination of bradycardia and hypothermia in cats with primary septic peritonitis has been established as a negative prognostic indicator.[1] Animals with uroperitoneum may continue to urinate with concurrent leakage into the peritoneal cavity.

DIAGNOSTIC TESTS

Although the preoperative diagnosis of peritonitis is confirmed by identification of a septic or aseptic inflammatory process in peritoneal fluid obtained by abdominocentesis, patients with suspected or

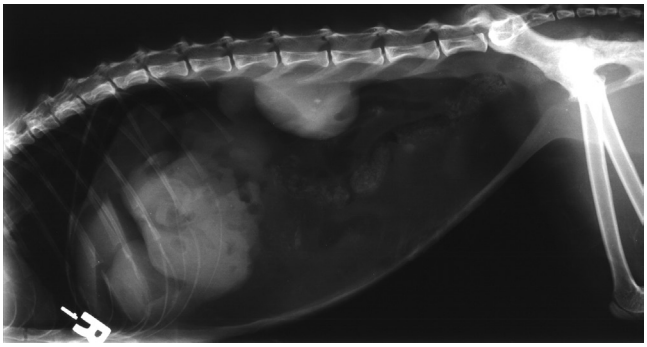

FIGURE 122-1 Lateral abdominal radiograph showing free peritoneal gas and possibly ingesta free within the abdomen. Pneumoperitoneum, without a history of recent surgery or open-needle abdominocentesis, indicates the need for abdominal exploratory surgery. This cat was diagnosed with a ruptured gastric mass at surgery.

confirmed peritonitis should have routine hematologic, biochemical, and coagulation analyses performed. A marked neutrophilia with a left shift is the predominant hematologic finding, although a normal or low neutrophil count may be present. Animals recovering without incident from GI surgery also may have a transient inflammatory leukogram; however, the overall peripheral white blood cell counts typically fall within normal limits.[11] An increasingly left-shifted neutrophilia (or neutropenia) paired with clinical signs of peritonitis may raise the clinician's index of suspicion for postoperative intestinal dehiscence (which typically occurs 3 to 5 days after surgery).

Furthermore, acid-base and electrolyte abnormalities may be noted. Hyperkalemia (and azotemia) may indicate uroperitoneum, particularly if trauma or urinary tract dysfunction has been noted historically. Hypoproteinemia may be a result of the loss of protein within the peritoneal cavity. Patients with a concurrent septic process may be hypoglycemic. Hepatic enzymes, creatinine, and blood urea nitrogen may be elevated, indicating primary dysfunction of these organs or perhaps reflecting a state of decreased perfusion or dehydration. The serum of patients with bile peritonitis is often icteric if the total bilirubin is elevated. Recently, the prevalence of ionized hypocalcemia in cats and dogs with septic peritonitis has been recognized and a failure to normalize calcium levels during hospitalization associated with negative prognosis.[12,13]

Plain radiographs may reveal a focal or generalized loss of detail that also is known as the *ground glass appearance.* A pneumoperitoneum (Figure 122-1) suggests perforation of a hollow viscous organ, penetrating trauma (including recent abdominal surgery) or, less commonly, the presence of gas-producing anaerobic bacteria. Intestinal tract obstruction or bowel plication should be ruled out. Prostatomegaly in male dogs and evidence of uterine distention in female dogs should be noted. Thoracic radiographs should be performed to rule out concurrent illness (infectious, neoplastic, or traumatic). The presence of bicavitary effusion increased the mortality rate of patients 3.3-fold compared with that of patients with peritoneal effusions alone.[14] Ultrasonography may be useful for defining the underlying cause of peritonitis, in addition to its use in localizing and aiding retrieval of peritoneal effusion. In the case of a confirmed uroabdomen, preoperative contrast radiography (excretory urography or cystourethrography) is recommended to localize the site of urine leakage and aid in surgical planning. All patients should be stabilized hemodynamically and medically before diagnostic imaging is performed.

Patients with suspected peritonitis should be evaluated for peritoneal effusion. Little or no fluid may be detected initially if patients arrive early in the disease process or before fluid resuscitation if they are dehydrated (see Table 112-1). Large volumes of effusion may be

obtained via blind abdominocentesis or, alternatively, via ultrasonographic guidance (see Chapter 200). Single paracentesis attempts are successful in only 20% of patients with low volumes of peritoneal effusion (3 ml/kg) and in only 80% with larger volumes (10 ml/kg). Ultrasonographic guidance facilitates the retrieval of smaller volumes of peritoneal fluid. If single-site sampling is negative for fluid, four-quadrant sampling should be performed.

A diagnostic peritoneal lavage (DPL, see Chapter 200) should be performed when peritonitis is suspected despite the absence of detectable effusion or when a minimal volume of effusion makes it difficult to obtain a sample. DPL ideally is performed using a peritoneal dialysis catheter but also can be performed using an over-the-needle, large-bore (14- to 16-gauge) catheter. The technique is performed by placing a catheter sterilely into the abdomen, infusing 22 ml/kg of a warmed, sterile isotonic saline solution, then retrieving a sample for analysis and culture and susceptibility testing. The lavage solution dilutes the sample and therefore alters the fluid analysis. A repeated DPL may increase accuracy of the technique when results of the first procedure are equivocal.

Whether obtained by paracentesis or DPL, cytologic, biochemical, and microbiologic analyses are useful in diagnosing peritonitis and further classifying type (septic or aseptic) and potential underlying cause (see Table 112-1 for overview). Leukocyte morphology has been suggested to be more reliable than cell counts in diagnosing peritonitis.[15] In an experimental study, DPL samples obtained before and after abdominal surgery suggest a nucleated cell count less than 1000 cells/µl (predominantly segmented neutrophils and macrophages) in dogs without intraabdominal pathology, whereas nucleated cell counts increased significantly in postoperative samples.[11] In a second experimental study, DPL cell counts between 500 and 10,500 cells/µl consisting predominantly of nondegenerate neutrophils are seen within the first 3 days after uncomplicated intestinal anastomosis.[15] Peritoneal leukocyte counts in animals with experimentally induced peritonitis exceed 5000 cells/µl (consistent with an exudate), with primarily degenerative neutrophils. Early in the disease process, lower cell numbers or an absence of degenerate neutrophils may occur in the face of septic peritonitis. The presence of intracellular bacteria, plant material/GI ingesta with associated inflammation, and/or free biliary crystals supports the diagnosis of peritonitis. Furthermore, increasing inflammation (numbers of neutrophils or morphologic features of toxicity in these cells) observed in serial samples and correlated with clinical findings may prove more useful than single leukocyte counts in abdominal fluid samples when deciding whether reoperation is indicated. Dogs receiving antimicrobial therapy may have no observable bacteria in peritoneal fluid samples, despite having peritoneal contamination.

In addition to the presence of bacteria and a high nucleated cell count with the presence of degenerate neutrophils, the glucose concentration of abdominal effusion is a useful predictor of bacterial peritonitis in dogs. A concentration difference of more than 20 mg/dl between paired samples for blood and peritoneal fluid glucose is a reliable predictor of a bacterial peritonitis; intravenous administration of dextrose or the presence of a hemoperitoneum may decrease the accuracy of this test. In addition, an abdominal fluid lactate concentration that is 2.0 mmol/L or greater than the blood lactate is predictive of septic peritonitis in dogs but has not been as useful in cats.[16,17] These parameters have been shown to be unreliable indicators of septic peritonitis in the evaluation of postoperative cases in which closed suction drains have been placed.[18] Samples for aerobic and anaerobic cultures should be obtained at the time of initial sampling so that additional samples are not required after confirming the presence of a septic process and initiating antimicrobial therapy.

The diagnosis of uroperitoneum in dogs can be made if the peritoneal fluid creatinine or potassium concentration exceeds that of the serum creatinine (more than 2:1) or potassium concentration (more than 1.4:1).[19] Similarly, biliary rupture leads to a bilirubin concentration that is higher in the peritoneal fluid than in the serum. In addition, bile pigment or crystals may be visible on cytologic examination of the peritoneal effusion in animals with bile peritonitis (Figure 122-2). These changes may not be seen in patients with bile peritonitis secondary to a ruptured gallbladder mucocele because the gelatinous bile often fails to disperse throughout the abdomen.

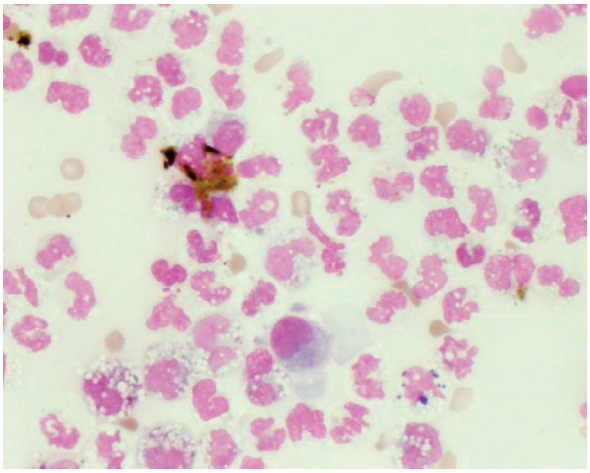

FIGURE 122-2 Microscopic examination of Wright-stained peritoneal fluid reveals markedly degenerative neutrophils, activated macrophages, and extracellular gold-brown pigment. One neutrophil in this high-powered field contains large bacterial rods (lower right hand side). This cytologic evaluation, together with elevated total bilirubin concentration in the peritoneal fluid relative to the serum concentration, confirms a diagnosis of a septic bile peritonitis.

TREATMENT

Medical Stabilization

The goals for animals with septic peritonitis are to identify and address the source of contamination to resolve the infection and treat the systemic consequences as quickly as possible (i.e., fluid and electrolyte abnormalities and hypoperfusion). Before surgical intervention, a decision must be made whether additional hemodynamic stabilization is indicated before proceeding, or whether this additional time and continued contamination of the abdominal cavity will result in further clinical decline that outweighs the benefits of additional medical treatment.

The goals of medical therapy are to restore normal fluid and electrolyte balance and minimize ongoing contamination. Fluid resuscitation is initiated after obtaining pretherapy blood samples for a minimum database (packed cell volume, total solids, BUN, dextrose), hematology, serum chemistry, and coagulation evaluation. Urine should be collected, if possible, for analysis with or without culture and susceptibility testing. Shock doses of crystalloids (up to 90 ml/kg in the dog, 50 ml/kg in the cat) or a combination of isotonic crystalloids (up to 20 to 40 ml/kg) and synthetic colloids (hydroxyethyl starch up to 20 ml/kg in the dog or up to 10 ml/kg in the cat; or 7% to 7.5% hypertonic saline in synthetic colloid solution (1 part 23.4% hypertonic saline to 2 parts synthetic colloid), 3 to 5 ml/kg IV over 5 to 15 minutes) should be administered to effect (see Chapter 60). Because significant amounts of protein are lost into the peritoneal cavity, plasma and/or albumin administration also may be warranted. Judicious fluid therapy is recommended to avoid volume overload. Electrolytes and glucose should be supplemented if indicated (see Electrolyte and Acid-Base Disturbances, Chapters 50 through 56, and Chapter 66). After appropriate volume resuscitation,

vasopressor therapy may be necessary to alleviate hypotension further. A urinary catheter may aid in diversion of infected urine in the case of a ruptured bladder or proximal urethra and allow time for the necessary correction of any metabolic derangements (typically hyperkalemia and acidosis) before surgery. Analgesia is an important component of preoperative management for peritonitis patients. Opioids often are used as a first-line choice for pain management; however, they must be used with caution because of their negative effects on GI motility, as well as their dose-dependent respiratory depression (see Chapters 144 and 163).

Broad-spectrum antimicrobial therapy should be initiated immediately after confirming the diagnosis of septic peritonitis (see Chapters 93 and 94). *Escherichia coli, Clostridium* spp., and *Enterococcus* spp. are common isolates. A second-generation cephalosporin such as cefoxitin (30 mg/kg IV q6-8h) may be used as a single agent or combination antimicrobial therapy such as ampicillin or cefazolin (22 mg/kg IV q8h) administered concurrently with either enrofloxacin (10 to 20 mg/kg IV q24h [dog], 5 mg/kg IV q24h [cat]) or an aminoglycoside (amikacin 15 mg/kg IV, IM, SC q24h [dog], 10 mg/kg IV, IM, SC q24h [cat] or gentamicin 10 mg/kg IV, IM, SC q24h [dog], 6 mg/kg IV, IM, SC q24h [cat]). If extended anaerobic coverage is necessary, metronidazole (10 mg/kg IV q12h) may be considered. Aminoglycosides usually are avoided until renal insufficiency or acute kidney injury has been ruled out and the patient is well hydrated. Antimicrobial therapy should be tailored to the results of culture and susceptibility testing.

Surgical Treatment

The goals of surgical treatment for patients with septic peritonitis include resolving the cause of the infection, diminishing the infectious and foreign material load, and promoting patient recovery with aggressive supportive care and nutritional supplementation, if indicated. A ventral midline celiotomy from xiphoid to pubis allows a thorough exploratory laparotomy to determine the underlying cause. Monofilament suture material is advocated in animals with a septic process, and surgical gut is avoided because of its shortened half-life in this environment. Placement of nonabsorbable suture material or mesh within the abdominal cavity is not recommended in cases of septic peritonitis because these materials may serve as a nidus for infection. If possible, the surgeon should isolate the offending organ from the rest of the abdomen with laparotomy sponges to prevent further contamination during correction of the problem.

Surgical treatment is tailored to the individual case and the underlying cause of the septic peritonitis. If a GI leakage is identified, adjunctive procedures such as serosal patching or omental wrapping of the repaired site are recommended to reduce the incidence of postoperative intestinal leakage or dehiscence. Although heavily contaminated or necrotic omentum may necessitate partial omentectomy, preservation of as much omentum as possible is advised to promote venous and lymphatic drainage from the peritoneal cavity. In addition, potential benefits of surgical applications of the omentum (e.g., intracapsular prostatic omentalization for prostatic abscess formation[20] pancreatic abscess omentalization,[21] omentalization of enterotomy or intestinal resection and anastomosis sites, and around gastrostomy or enterostomy tube sites) relate to its immunogenic, angiogenic, and adhesive properties. Because enteral nutrition directly nourishes enterocytes and decreases bacterial translocation across the intestinal wall, feeding tube placement (gastrostomy or jejunostomy) should be considered during initial surgical exploration.

After addressing the underlying cause to prevent further contamination of the peritoneum, clinicians must reduce the infectious and foreign material load by a combination of debridement and lavage. Localized peritonitis should be treated with lavage of the affected area initially to minimize dissemination of the infection. A thorough lavage of the entire abdominal cavity with sterile isotonic fluid (warmed to body temperature) is warranted to remove bacteria, as well as GI contents, urine, or bile. The addition of antiseptics and antibiotics to lavage fluid is not beneficial and actually may be detrimental by inducing a superimposed chemical peritonitis. Lavage of the abdominal cavity is continued until the retrieved fluid is clear. All lavage fluid should be retrieved because fluid accumulation in the abdominal cavity impairs bacterial opsonization and clearance.[22]

If debridement and lavage can resolve gross foreign material or GI spillage and the source of contamination can be controlled, the abdomen should be closed primarily because of the potential complications associated with continued abdominal drainage (described below). All patients with open abdominal drainage are susceptible to superinfection with nosocomial bacteria and may experience massive fluid and protein losses.

Open peritoneal drainage is accomplished with a simple continuous pattern of nonabsorbable suture material in the rectus abdominis muscle, placed loosely enough to allow drainage through a gap of 1 to 6 cm in the body wall (Figure 122-3). A preassembled, sterile bandage that comprises a nonadherent contact layer, laparotomy sponges or gauze pads, roll cotton or surgical towels, roll gauze, and an outer water-impermeable layer is placed to absorb fluid and protect the abdominal contents from the environment. Initially, this bandage is replaced twice during the first 24 hours and daily thereafter, although the amount of drainage produced by an individual patient may dictate more frequent changes. A sterile-gloved finger may have to be inserted through the incision to break down adhesions and to allow thorough drainage of the peritoneal cavity. Alternatively, patients with severely contaminated tissues may require daily general anesthesia for repeated abdominal exploration and lavage before reapplying the bandage. The quantity of fluid can be estimated by the difference in weight of the bandage before application and after removal. Abdominal closure typically is performed 3 to 5 days after the initial surgery. The placement of a urinary catheter and collection system helps to limit urine soaking of the bandage and underlying exposed tissues.

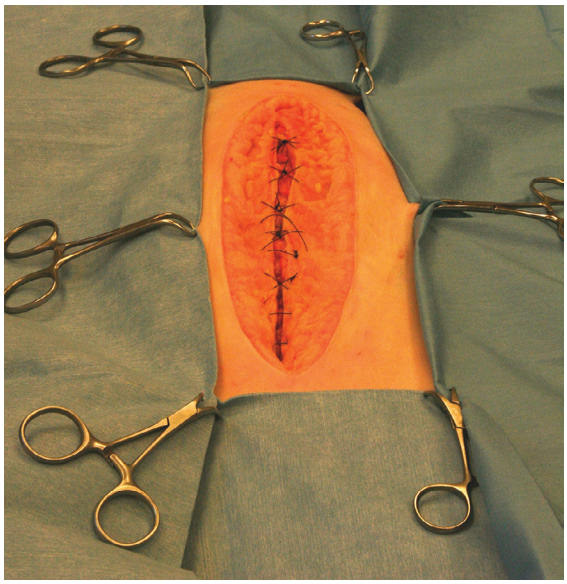

FIGURE 122-3 Open abdominal drainage incision. The incision should be closed with a single layer of nonabsorbable suture material to provide an opening that allows drainage but does not allow abdominal viscera or omentum to herniate through the open incision. A preassembled sterile bandage is placed over this incision and is changed daily, or more frequently as required to prevent strike-through.

The use of vacuum-assisted peritoneal drainage (VAPD) recently has been described as a means to provide continued postoperative abdominal drainage (see Chapter 139). Although the caudal one third to two thirds of the abdominal incision is closed primarily, the remainder of the incision is reapposed loosely (as described earlier in the chapter) and subatmospheric pressure applied to the cranial portion of the incision. This approach has been used successfully in human patients and its success demonstrated by significant reductions in open abdominal drainage duration times, number of dressing changes, re-exploration rate, and successful abdominal closure rates.[23,24] Superiority of this approach has yet to be established in small animal surgical patients. Survival rates for canine and feline septic peritonitis patients treated with VAPD has been reported as 37.5% (3/8)[25] and 50% (3/6),[26] which is similar to that seen with other abdominal drainage techniques. However, at this time, insufficient case numbers have been examined to draw conclusions as to whether the success of VAPD seen in human patients can be achieved in veterinary medicine. Dressings are available commercially that provide a barrier between the abdominal wall and viscera to protect the abdominal organs.

Alternatively, the abdomen may be closed primarily and drainage accomplished with closed suction (e.g., Jackson-Pratt) drains.[27] Closed suction drainage has been advocated for treatment of patients with generalized peritonitis because it has several advantages over open abdominal drainage, including a decreased risk of nosocomial infection, less intensive nursing care and bandaging requirements, decreased risk for evisceration, and the need for only one surgical procedure.[27] Disadvantages are that the drains may induce some fluid production and may become occluded, although active drainage was maintained for up to 8 days with this technique in 30 dogs and 10 cats in one study.[27] In addition, closed suction drains allow daily quantitative and qualitative assessment of retrieved fluid for evaluating the progression of the peritonitis. Typically, one drain placed between the liver and diaphragm is sufficient for small dogs and cats, whereas two drains are more appropriate for larger dogs (the fenestrated portion of second drain is placed in the caudal abdomen along the ventral body wall). The drain tubes exit the body wall through a paramedian stab incision and are sutured to the abdominal skin with a pursestring and Chinese finger-trap sutures (Figure 122-4). After routine closure of the abdomen, the suction reservoir bulb is attached to the tubing with vacuum (negative pressure) applied. A protective abdominal bandage is placed with sterile contact material around the tube-skin interface and is changed daily to allow assessment of this site. Fluid collected within the bulbs is emptied using aseptic technique, and the volume is recorded every 4 to 6 hours, or more frequently if needed. Drains are removed by applying gentle traction when the volume of fluid production has decreased significantly and cytologic analysis suggests resolution of the peritonitis (i.e., decreasing cell numbers and nondegenerative neutrophils, absence of bacteria). A sterile bandage is reapplied to cover the drain exit site for 24 hours.

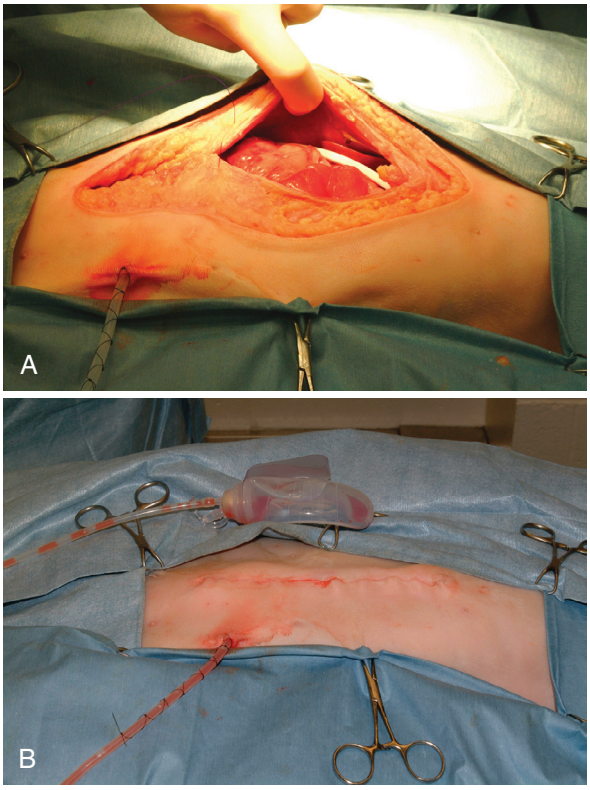

FIGURE 122-4 A, Closed suction drainage may be accomplished by placing a single Jackson-Pratt drain cranial to the liver (a second drain also may be placed in the caudal abdomen along the ventral body wall in large dogs), exiting paramedian to the abdominal incision. **B,** The tubing is secured to the body wall with a purse-string and Chinese finger trap sutures. Once the abdomen is routinely closed, the suction reservoir is attached and a vacuum is created by compressing the bulb. An abdominal bandage is placed to allow attachment of the drainage tubing and reservoir to prevent entanglement and premature removal by the patient.

Postoperative Care

Postoperative care for patients with peritonitis is typically intense because these patients are critically ill and subject to a variety of complications (see Chapter 131).[28] Aggressive intravenous fluid therapy is a necessity, particularly in patients with continued fluid losses from the inflamed peritoneal cavity. Electrolytes and acid-base status should be assessed routinely during the postoperative period and corrected as needed. Because anemia and hypoproteinemia are common complications in these patients, blood component therapy and synthetic colloidal support are often necessary, with a goal of maintaining a packed cell volume greater than 20% to 25%, serum protein over 3.5 g/dl, and colloid osmotic pressure higher than 16 mm Hg.

Proper nutrition provides a much-needed source of protein and energy in these patients. Failing to meet nutritional demands, either with parenteral or enteral nutrition, may contribute to impaired wound healing and immune defenses. In fact, early nutritional support is associated with shorter hospitalization in dogs.[29] Enteral feeding is preferred over parenteral feeding but may be stymied by the anorectic patient unless GI feeding tubes were placed at the time of surgery. If this was not done, a nasoesophageal tube can be placed easily in patients unable to tolerate repeated anesthesia. Alternatively, an esophagostomy tube may prove beneficial in patients that can tolerate general anesthesia. Animals with refractory vomiting typically require parenteral nutrition (see Chapters 129 and 130).

Postoperative hypotension may be treated with vasopressor therapy but only after addressing any underlying hypovolemia (see Chapters 8, 157, and 158). Proper analgesia is required to ensure patient comfort and to diminish the negative cardiovascular effects associated with overactive sympathetic stimulation (see Chapter 144). Other complications, including cardiac arrhythmias, disseminated intravascular coagulation, and systemic inflammatory response syndrome can be found in other chapters (see Chapters 6 and 91).

PROGNOSIS

The prognosis for animals with peritonitis depends on the underlying cause and whether infection is present. Studies in which patients have benefited from advances in critical care management cite overall

survival rates of 44% to 71%.* Cats were reported to have a lower survival rate than dogs in two studies[3,30]; however, two studies focusing on feline septic peritonitis found an approximate 70% survival in animals in which treatment was pursued.[1,4] Poor prognostic indicators for animals with septic peritonitis have included refractory hypotension, cardiovascular collapse, disseminated intravascular coagulation, and respiratory disease.[27,34] The combination of hypothermia and bradycardia on presentation in feline patients appears to be a negative prognostic indicator.[1] Mortality rates in patients with septic peritonitis secondary to GI leakage have been reported to vary between 30% and 85%.[2,3,7,8] Bacterial contamination was associated significantly with mortality in animals with bile peritonitis.[36] Although survival in dogs with aseptic bile peritonitis was between 87% and 100%, those with septic bile peritonitis had survival rates of only 27% to 45%.[32,36] Overall survival rate in cats with uroperitoneum was 62%.[31] Survival rates appear to be similar in patients with septic peritonitis treated with primary closure, open peritoneal drainage, closed suction drainage, or vacuum-assisted drainage.[25-27,33,35]

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