

ACUTE ABDOMEN: An Ugly Situation

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Acute abdominal pain results from stimulation of pain fibers associated with the abdominal cavity, abdominal organs, or the nerves, muscle, fascia or skin associated with the abdomen. Stimulus of the sensory nerve endings happens when regional tension and/or inflammation occur. Clinical signs associated with acute abdominal pain may include restlessness, abdominal distension, arched back/stiff gait, dehydration, poor perfusion parameters, tachypnea, tachycardia, fever, depressed attitude, anorexia, and hypersalivation.

Historical information that is significant to this syndrome includes vaccination and internal parasite history, administration of medication, history of past medical problems, access to garbage, toys, food, or toxins, and recent trauma or abdominal surgery. If vomiting and diarrhea are part of the history, they should be characterized with respect to color, consistency, and association with to last meal. Vomiting should be distinguished from regurgitation by the presence of active abdominal contractions during the episode.

PRIMARY SURVEY

During the primary survey, particular attention should be paid to the patient's perfusion and hydration status. Tachypnea, labored breathing, and moist lung sounds may indicate aspiration of vomitus. Vomiting can cause a significant vasovagal response, bradycardia and respiratory arrest. Close monitoring, including continuous ECG, pulse oximetry and arterial blood pressure, is necessary when circulatory distress or altered mentation accompanies severe gastrointestinal (GI) disease.

Resuscitation begins with correction of any ventilation or oxygen abnormalities. Assisted ventilation is performed if respiratory failure is imminent or happening. Supplemental oxygen is provided if poor perfusion or labored breathing is present. Immediate intravascular volume replacement to correct hypovolemia requires the placement of peripheral IV catheters and administration of balanced isotonic crystalloids (10-30 ml/kg incremental boluses and synthetic colloids (hydroxyethyl starch 5 ml/kg incremental boluses) until end-point resuscitation parameters are reached. Significant anemia may require a blood transfusion or administration of hemoglobin-based oxygen carriers. Rear-limb abdominal counterpressure may help improve venous return when significant hypotension is occurring despite volume replacement, particularly when intraabdominal hemorrhage is ongoing. End-point parameters desired during resuscitation include normal perfusion parameters, heart rate and mean arterial pressure 60-80 mmHg.

Painful stimuli will induce many of the same cardiovascular responses that are induced by shock, and can contribute to the declining state. Analgesia administration is part of the resuscitation process and can be combined with sedatives for an enhanced effect.(Table 1) Epidural administration of opioids and local anesthetics is most effective for abdominal pain nonresponsive to intravenous opioids. Nonsteroidal anti-inflammatory medication are used with extreme caution until the underlying cause has been established, it has been determined that surgical intervention is not necessary, that the integrity of the GI tract is intact, renal function is normal, and volume resuscitation has been completed.

Blood and urine samples are collected during initial intervention. Immediate evaluation of the packed cell volume (PCV), total solids (TS), serum color, glucose, blood urea nitrogen, venous blood gas, electrolytes, lactate level, platelet estimate, prothrombin/partial thromboplastin times, urine specific gravity and urine dipstick may lead to a diagnosis. Abnormalities may require immediate intervention. Once the patient is stable, samples can be further evaluated for complete blood count, serum biochemical profile including amylase and lipase, coagulation profile, and urine sediment.

SECONDARY SURVEY

On secondary survey following analgesia administration, evaluation of the abdomen might detect ascites or distension, organ abnormalities and localization of the pain. Careful examination of the back should distinguish abdominal pain from referred spinal pain. When bowel sounds are absent during abdominal auscultation, this suggests hypomotility, ileus, fluid accumulation, or diffuse peritonitis. Rectal examination

may reveal masses, foreign objects in fecal matter, or evidence of blood. Dark blood (melena) implies upper GI hemorrhage and frank blood indicates large intestinal hemorrhage. Finally, the muscles, fascia and skin of the abdomen should be examined for evidence of inflammation, infection, trauma, and hemorrhage. Hemorrhage in the umbilical or periscrotal tissue may indicate hemoabdomen.

DIAGNOSTIC EVALUATION

If the patient can be stabilized, a complete blood count, serum biochemistry profile (including amylase and lipase), pancreatic lipase immunoreactivity testing, microscopic fecal examination, parvoviral fecal antigen test, fecal cultures, fecal pathogen profile, urinalysis, and coagulation profile may aid in the determination of cause of vomiting and diarrhea, as well as secondary complications or additional organ dysfunction. Hyperphosphatemia may reflect intestinal ischemia or severe intestinal inflammation. Specific diagnostic tests to identify secondary GI disease are performed as indicated (e.g. adrenocorticotrophic hormone [ACTH] stimulation, preprandial and postprandial bile acids).

If the cardiovascular status of the animal stabilizes during resuscitation and surgical intervention is not immediately required, additional diagnostic testing is performed to rule out a differential diagnosis list (Table 2). Abdominal radiographs are evaluated for detail, gas patterns, unexpected mineral or soft tissue densities, and changes in organ size or shape. An abdominal ultrasound can be invaluable for diagnosing presence of fluid, subtle organ enlargement, mass lesions, metastatic disease, vascular occlusion, pyometra, urinary tract obstruction, gastrointestinal obstruction, and pancreatitis.

If abdominal fluid is suspected based on examination or seen on abdominal ultrasound, the fluid is collected via paracentesis and evaluated cytologically and chemically. If fluid is not easily collected, a diagnostic peritoneal lavage (DPL) may be necessary to collect cellular samples for evaluation. A sample is collected and evaluated. Radiographs are always obtained prior to DPL since introduction of fluid into the abdominal cavity will affect contrast.

Immediate abdominal decompression or surgical intervention may be necessary when medical resuscitative efforts are unsuccessful, or the cardiovascular status decompensates. Surgery in many cases is considered a diagnostic procedure, with the expectation that it will become part of the treatment of the condition. Indications for surgical exploration are listed in Table 3. The patient is made as stable as possible prior to anesthesia.

The surgical team and facility is prepared. Animal preparation should include clipping the hair from and disinfection of the caudal thorax, pubis and inguinal thigh region in case maximum exposure of the abdominal cavity requires a parasternotomy, or central vessel access is desired. Anesthetic machines, lighting, surgical instruments, suction, and suture material should be assembled and in working order. Oropharyngeal and esophageal suctioning may be required if gastric fluid has refluxed during anesthetic induction. Placement of a nasogastric tube for suction prior to induction of anesthesia can reduce the incidence of and complications associated with reflux and aspiration.

Upon entering the abdominal cavity, uncontrollable arterial bleeding is temporarily halted with compression of the aorta, cranial to the celiac artery. While suctioning the abdomen, sterile laparotomy pads/towels are used to pack the cavity to control venous hemorrhage. The towels are systematically removed one at a time starting caudally. Sources of bleeding are located and ligated. Once the hemorrhage has been controlled, a systematic review of the abdominal quadrants and organs is performed. Any severely injured organ, infected or potentially neoplastic tissue is excised. Tissues or fluid samples are obtained for aerobic and anaerobic culture. Biopsy specimens of any abnormal organs are submitted for histopathology. If no gross abnormalities are evident, biopsy specimens of the liver, kidney, pancreas, stomach, small intestine, mesenteric lymph node, and abdominal muscle should be considered. In addition, intestinal contents from the distal duodenum and jejunum are submitted for cytological evaluation and culture.

Once sample removal is completed, the abdomen is copiously lavaged with warmed sterile saline and suctioned. Feeding tubes are placed in the esophagus, stomach or jejunum if postoperative nutritional support is required. This may be the case when prolonged recovery is anticipated, when moderate to severe

malnutrition present, or when hypermetabolism is expected. If septic peritonitis is present, peritoneal drainage may be necessary.

CONTINUED TREATMENT

If the animal is stabilized, further treatment will consist of rehydration over 2-6 hours and maintenance of fluid balance. The need for large fluid volumes is common since rapid fluid shifts into third body fluid spaces (e.g. GI tract, peritoneal space, uterus) can be significant. If diseases causing SIRS (systemic inflammatory response syndrome; severe vomiting and/or diarrhea, peritonitis, pyometras, pancreatitis) are suspected/occurring, the addition of a large molecular weight colloid such as hetastarch (0.8 ml/kg/hr) may be required to maintain colloid osmotic pressure.

Antibiotics should be administered if hypovolemic shock has happened or GI signs exist, since translocation of Gram positive and Gram negative aerobes and anaerobes can occur when perfusion and integrity of the GI tract has been compromised. Cefazolin or ampicillin (20 mg/kg IV q8hr) and metronidazole (10 mg/kg IV q12hr), or ampicillin/sulbactam (20 mg/kg IV q 8h) provide appropriate broad spectrum coverage.

If persistent vomiting is occurring, a nasogastric tube can be placed to reduce the volume of gastric contents and gastric stretch. Microenteral nutrition and early feeding are facilitated with a nasogastric tube. Nasogastric tube suctioning is likely the single best method in our experience that reduces vomiting frequency.

Motility enhancers such as metoclopramide (0.2-0.4 mg/kg SQ q6-8hr or 1.0-2.0 mg/kg/day IV CRI) or cisapride (0.1-0.5 mg/kg PO q8-12hr in the dog; 0.5-1mg/kg q8hr in the cat) can be administered if an unobstructed ileus is occurring. Chlorpromazine (0.05 mg/kg IV in dogs and 0.01-0.025 mg/kg IV in cats) or prochlorperazine (0.05-0.25 mg/kg IV TID-QID) can be administered provided the patient is cardiovascularly stable. Substance-p and serotonin inhibitors, such as maropitant (2-8 mg/kg PO; 1mg/kg SQ q 24h), and ondansetron (0.1-0.3 mg/kg SQ TID, or 0.5 mg IV load then 0.5 mg/kg/hr CRI) or dolasetron (0.5 mg/kg IV, SQ q 24h), may also be helpful and can be used in combination with other antiemetics. Drugs inhibiting GI motility (e.g. anticholinergics such as centrine, or lomotil) are not antiemetics, and not used because of the potential of promoting buildup of toxins and affecting nutrient absorption.

If gastric or esophageal ulceration are suspected, H₂-blockers reduce acid secretion and reflux as well as promote mucosal healing. Pantoprazole (0.7-1 mg/kg IV q 24h) or omeprazole (0.7 mg/kg up to 20 mg q24 hours PO) are the preferred drugs when vomiting frequency is reduced. Other antacids include cimetidine (4 mg/kg IV/SQ q6-8hr), ranitidine (2mg/kg IV q8hr in the dog; 2.5 mg/kg IV q12hr in the cat), and famotidine (0.5 mg/kg IM/SQ q12hr) but they do not increase gastric pH as effectively as H₂-blockers. In addition, liquid sucralfate (0.5-1g PO q6-8hr) will coat the area of erosion/ulcer once vomiting is controlled. Sucralfate requires an acid environment, and should be given at a separate time from H₂-blockers.

Additional patient monitoring should include the parameters listed in the Rule of 20.(Kirby, 1994)

REFERENCES

- Herold L, Devey J, Kirby R, Rudloff E. Clinical evaluation and management of hemoperitoneum in dogs. *J Vet Emerg Crit Care* 2008 18(1):40–53
- Kirby R: The acute abdomen syndrome. *Handbook of Small Animal Medicine*. (Morgan R, ed) 2001
- Kirby, R: Septic Shock. In Bonagura JD (ed): *Kirk's Current Veterinary Therapy XII*. WB Saunders, Philadelphia, 1995
- Mathews, K: Management of pain. *Vet Clin North Am [Small Anim Pract]* 30:4, 2001
- Purvis D, Kirby R: Systemic inflammatory response syndrome: septic shock. *Vet Clin North Am [Small Anim Pract]* 24:1225, 1994
- Rudloff E: Abdominocentesis and Diagnostic Peritoneal Lavage. In Ettinger S, Feldman E, *Textbook of Veterinary Internal Medicine* (4th ed) 2005; pp269-270.
- Kirby R, Rudloff E: Crystalloid and colloid fluid therapy. In Ettinger S, Feldman E, *Textbook of Veterinary Internal Medicine* (4th ed) 2005; pp412-423.

Table 1: Analgesic drugs and doses for treating moderate to severe pain in the critically ill dog & cat

ANALGESIC	CANINE DOSE	FELINE DOSE	FREQUENCY
Opioids			
Buprenorphine		0.01 mg/kg SC, IM, IV 0.02 mg/kg SL	q4-6 hr
Morphine or Methadone	0.5–2.0 mg/kg SC, IM	0.05-0.2 mg/kg SC, IM	q3-4 hr
	0.5–1.0 mg/kg IV SLOW	0.1-0.4 mg/kg IV SLOW	q3-4 hr
preservative-free Morphine	0.1-0.3 mg/kg/hr IV CRI after 0.5 mg/kg IV SLOW	0.1-0.3 mg/kg/hr IV CRI after 0.25 mg/kg IV SLOW	
	0.1-0.3 mg/kg EPI	0.1 mg/kg EPI	q12-24 hr
Hydromorphone	0.1-0.2 mg/kg SC, IM, IV	0.02-0.1 mg/kg SC, IM, IV	q2-6 hr
	0.03-0.04 mg/kg EPI	0.03-0.04 mg/kg EPI	q8 hr
Fentanyl	25 mcg patch < 10 kg 50 mcg patch 10-20 kg 75 mcg patch 20-30 kg 100 mcg patch >30 kg	25 mcg patch	q3-4 days Onset of effect 12-24 hrs after application
	3-10 mcg/kg/hr IV CRI after 5 mcg/kg bolus	1-5 mcg/kg/hr IV CRI after 3 mcg/kg bolus	
Sedatives			
Diazepam	0.2-0.6 mg/kg IV	0.5-1.0 mg/kg IV	
Midazolam	0.25-0.5 mg/kg IV 0.25-0.5 mg/kg/hr CRI	0.25-0.5 mg/kg IV 0.25-0.5 mg/kg/hr CRI	
Local Anesthetics			
Bupivacaine (0.5%)	2 ml/kg ID*	2 ml/kg ID*	
	1.5-2.5 mg/kg (6ml total) EPI	1.5-2.5 mg/kg (1.5ml total) EPI	Mix with opioid EPI
Lidocaine (2%)	2-4 mg/kg ID*	2 mg/kg ID*	
	3-5 mg/kg (6ml total) EPI	3-5 mg/kg (1.5ml total) EPI	Mix with opioid EPI
Combination			
Morphine Lidocaine Ketamine	20 mg/L 300 mg/L 60 mg/L	Rapidly infuse 2 ml/kg, then set rate at 2 ml/kg/hr	
Fentanyl Lidocaine Ketamine	1.5 mg/L 300 mg/L 60 mg/L		
Bupivacaine (0.5%) Sodium bicarbonate	1 mg/kg 0.01 mEq/kg Diluted with 0.9% saline to 3, 6, 12 mls	Administered intrapleural or intraperitoneal	

SC: Subcutaneous, IM: Intramuscular, IV: Intravascular, SL: Sublingual, EPI: Epidural, ID: Intradermal, CRI: Constant rate infusion (a bolus infusion of drug is required prior to initiating therapy)

*can be diluted by 50% with warm saline to reduce the pain of injection and promote dispersal

Table 2: Differential diagnosis list for acute abdominal pain

GI tract lesions

Gastric ulceration, pyloric outflow obstruction, gastric dilatation-volvulus, perforation

Intestinal obstruction, torsion, ulceration, perforation, infection, intussusception

Mesenteric thrombosis, volvulus, avulsion of mesenteric vessels

Lymph node neoplasia or infection

Inflammation, abscess, or infarction of the pancreas

Reproductive tract

Uterine infection, torsion, rupture
 Intra-abdominal testicular torsion, infarct, abscess
 Prostatic infection, cyst, infarct, abscess
 Reticuloendothelial system (liver and spleen)
 Splenic tumors, torsion, fracture, infection, thrombosis, or enlargement secondary to systemic disease
 Liver abscess, infection, tumor, torsion, enlargement as seen with extramedullary hematopoiesis or hepatopathy, biliary obstruction or rupture
 Urinary tract
 Kidney swelling, renal calculi, infarct, infection, fracture, hematoma, or avulsion
 Urethral /Ureteral obstruction, rupture, passage of calculi
 Urinary bladder obstruction, inflammation, trauma, rupture
 Peritoneal space and surfaces
 Hemoperitoneum Pneumoperitoneum Bile peritonitis Septic peritonitis
 External abdominal structures
 Steatitis Myositis Fasciitis
 Nerve root irritation by disc disease or neoplasia
 Hernias with strangulated viscera: Umbilical, Inguinal, Perineal, Abdominal wall

Table 3: Indications for exploration in the animal with acute abdominal pain

Lack of diagnosis in a deteriorating patient	Free abdominal air
Bacteria seen within WBC in abdominal or DPL fluid	Torsion/volvulus of an abdominal organ
Plant fibers or bacteria seen in abdominal or DPL fluid	Penetrating foreign body
Significant or persistent abdominal hemorrhage	Evidence of pyometra
Total or partial gastric or intestinal obstruction	Evidence of organ ischemia
Failure to respond to medical treatment for pancreatitis	Evidence of mass lesion or abscess
Debridement of infected wounds, muscle, fascia	Rupture of a major abdominal organ
Need for feeding tube placement that bypasses stomach	Obtain biopsies