Case Report

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Gastrointestinal tract perforations caused by ingestion of multiple magnets in a dog

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Abstract

Objective – To describe a case of gastrointestinal tract perforation, septic peritonitis and coagulopathy caused by ingestion of multiple magnets in a dog.

Case Summary – An 8-month-old castrated male Rottweiler, weighing 30.5 kg was presented for evaluation of vomiting and weakness. Abdominal radiography and abdominal ultrasonographic examination identified a metallic foreign object within the gastric lumen, presence of free peritoneal gas, and peritoneal effusion. Septic peritonitis was diagnosed by abdominal fluid analysis. Exploratory celiotomy revealed the presence of an omental abscess, and gastric and colonic perforations. Four magnetic foreign objects were found within the lumen of the perforated stomach. Surgical management including removal of the magnets, abscess debridement and excision, perforation repair, and abdominal drainage combined with intensive medical therapy resulted in complete recovery of this dog.

New or Unique Information Provided – This report describes in detail the case management of a dog that developed both gastric and colonic perforations and severe morbidity secondary to ingesting multiple magnets.

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Introduction

Ingestion of multiple magnets leading to gastrointestinal (GI) injuries is well documented in the human medical literature,[1–7] but has only recently been reported in veterinary literature.[8] In people, magnet ingestion has been associated with intestinal volvulus,[1] perforation,[1,3,4,5] intestinal fistula formation,[6,7] adhesions,[2] abscess formation,[2,5] obstruction,[2] and peritonitis.[2,3,5] A recent report describes 2 cases of multiple magnet ingestion in dogs, with morbidity limited to gastric and intestinal wall necrosis with early perforation.[8]

Extrapolating from the experience in human medicine, one would expect similar morbidity associated with multiple magnet ingestion in dogs, including GI perforation and peritonitis. GI perforation in dogs is associated with gastric and small intestinal ulceration,[9–11] neoplasia,[10] gastric dilatation and volvulus,[11] trauma,[10,11] and ingestion of foreign bodies.[11] Ingested foreign bodies commonly occur in veterinary medicine, and often cause GI tract obstruction and subsequent perforation. Foreign objects typically cause perforation by pressure necrosis at the site of obstruction or through plication of the intestines resulting in multiple sites of perforation.

Serial perorally administered magnets have been experimentally investigated in dogs as a means to produce a functional gastroenteric anastomosis with interventional radiology techniques, thus avoiding celiotomy in people.[12] By administering the magnets several hours apart, the magnets were attracted to each other across intestinal sections, and the strong magnetic attraction between the intestinal walls led to pressure necrosis and communication between the intestinal lumens.[12] Gastric perforation was identified as a complication.[12]

In the current case report, we describe the presentation, surgical repair and medical management of a dog with multiple magnet ingestion that led to gastric and colonic perforation, abscess formation, septic peritonitis, and coagulopathy.

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Case Summary

An 8-month-old, castrated male Rottweiler, weighing 30.5 kg, presented for evaluation of lethargy and vomiting. On presentation, the dog vomited clear fluid, was anorexic, and lethargic. The dog's rectal temperature was 39.2°C, heart rate was 170/min, and respiratory rate was 40/min. The dog had depressed mentation, injected and dry mucous membranes with a capillary refill time between 2 and 3 seconds, sunken eyes, and decreased skin turgor. The dog was weak and unsteady while standing, and exhibited pain on cranial abdominal palpation. Dehydration was assessed between 8% and 10%. Systolic blood pressure[a] was 122 mm Hg, diastolic blood pressure was 73 mm Hg with a mean arterial pressure (MAP) of 94 mm Hg. A peripheral IV catheter was placed, and the dog was administered a 600 mL bolus of a balance crystalloid fluid[b] and 0.1 mg/kg of hydromorphone[c] IV for analgesia.

Initial diagnostic tests included a complete blood cell count, serum biochemical profile, and abdominal radiographs. Complete blood cell count demonstrated a moderate leukocytosis (21.2×10^9/L [21.2×10^3/μL]; reference interval 5.5–16.9×10^9/L [5.5–16.9×10^3/μL] with a neutrophilia (17.9×10^9/L [17.9×10^3/μL]; reference interval 2.0–12.0×10^9/L [2.0–12.0×10^3/μL]), with other values within reference interval. Abnormal biochemistry results included an increased alkaline phosphatase (305 U/L; reference interval 23–212 U/L), and hypoglobulinemia (24 g/L [2.4 g/dL]; reference interval 25–45 g/L [2.5–4.5 g/dL]). Electrolytes were within reference intervals. Urine could not be collected. Abdominal radiographs identified a cylindrical metallic density within the gastric lumen. Amorphous mineralized material was present in the stomach and colon. Mildly decreased abdominal detail was evident. Cursory sonography was performed and identified a small amount of peritoneal fluid. Abdominocentesis yielded a single drop of fluid for cytology and revealed primarily red blood cells, mature neutrophils and an occasional extracellular rod. The dog was transferred to the ICU for continued stabilization and diagnostic investigation. The dog was administered dolasetron[d] 0.6 mg/kg, IV, every 24 hours, famotidine[e] 0.5 mg/kg, IV, every 24 hours, continued on hydromorphone 0.1 mg/kg, IV, every 6 hours, and isotonic crystalloid fluids[b] at 6.5 mL/kg/h. Over the next few hours, the dog's demeanor, vital signs, and hydration improved. Repeat cursory ultrasound and abdominocentesis performed 1.5 hours after the initial scan did not yield additional fluid.

Upon review of the radiographs by a board-certified radiologist, several small gas opacities in the cranial abdomen were noted on a lateral projection, suggesting pneumoperitoneum (Figure 1). A full-abdominal ultrasound was performed, identifying a thickened, irregular pancreas, multiple fluid pockets at the caudal aspect of the stomach, multiple foci of peritoneal gas (Figure 2) and an atonic GI tract. Abdominocentesis was repeated and fluid cytology revealed degenerate neutrophils and occasional extracellular cocci with rare short rods. Fluid total protein was 34 g/L [3.4 g/dL], and fluid glucose concentration <1.1 mmol/L [20 mg/dL]. Peripheral blood glucose concentration was 4.7 mmol/L [85 mg/dL]. Metronidazole[f] 10 mg/kg, IV, every 12 hours and ampicillin sulbactim[g] 20 mg/kg, IV, every 8 hours were started. Based on the ultrasonographic examination, perforation of the GI tract was suspected, with infectious pancreatitis or pancreatic abscess also considered. The patient was prepared for exploratory celiotomy.

Abnormal values identified on pre-operative laboratory testing included hypoalbuminemia (21 g/L [2.1 g/dL]; reference interval 23–40 g/L [2.3–4.0 g/dL]) and hypoproteinemia (40 g/L [4.0 g/dL]; reference interval 52–82 g/L, [5.2–8.2 g/dL]). Hydroxyethyl starch[h] therapy was initiated at 20 mL/kg/d for oncotic support. A 4-lumen central venous catheter[i] was placed. The dog was blood typed as DEA 1.1 positive. Anesthesia was induced with fentanyl[j] IV (2.95 μg/kg), midazolam[k] IV (0.1 mg/kg), and propofol[l] IV (1.7 mg/kg), and maintained with isoflurane[m] in 100% oxygen and a constant rate infusion (CRI) of fentanyl (3–8 μg/kg/h), lidocaine[n] (0.6 mg/kg/h), and ketamine[o] (0.15 mg/kg/h in 0.9% NaCl). Crystalloid and synthetic colloid fluids were administered at 270 and 25 mL/h, respectively.

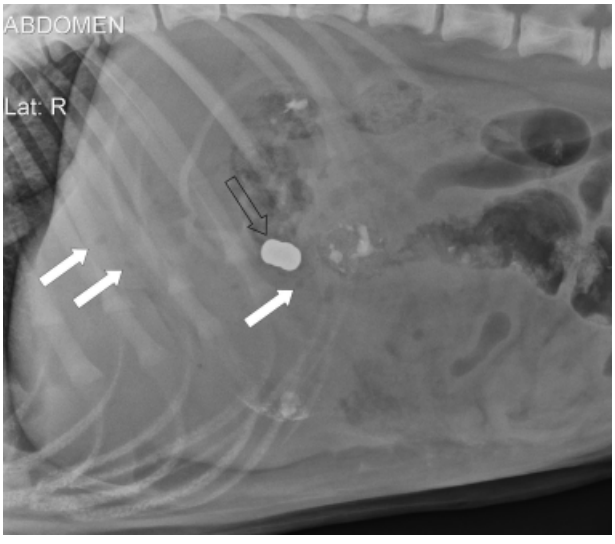

Figure 1: Right lateral abdominal radiograph of the patient demonstrating gas opacities or gas bubbles (white arrows) within the abdomen consistent with pneumoperitoneum. The open arrow indicates the metallic foreign object in the gastric lumen that was the magnetic foreign objects.

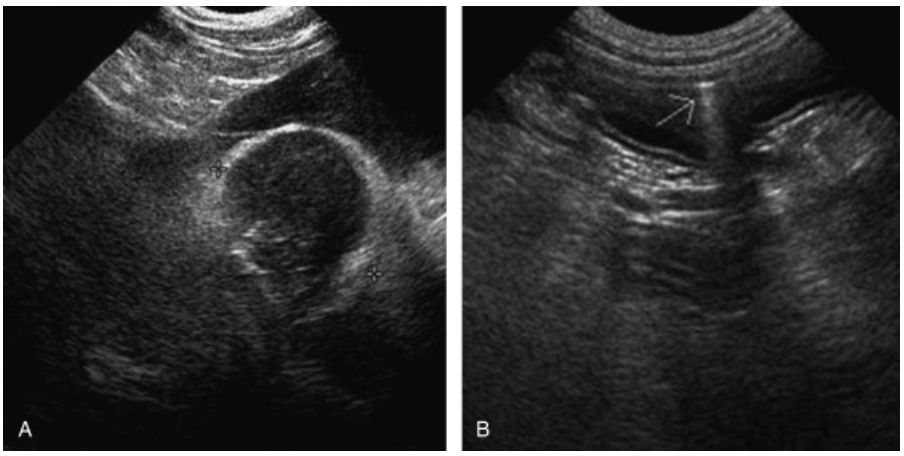

Figure 2: (A) The fluid filled structure caudal to the stomach that was the omental abscess adhered to gastric and colonic perforations. Abscess enclosed by the two cross hatches. (B) The appearance of free peritoneal gas on ultrasound is indicated by the arrow and seen as a hyperechoic point with distal reverberation.

Exploration of the abdomen revealed an extensive omental abscess involving mesenteric and intestinal adhesions in the left cranial abdominal quadrant in addition to a moderate amount of abdominal effusion. The abscess cavity was explored, debrided, and adhesions gently broken down. The colon was adherent to the ventral aspect of the abscess, and a 5 mm antimesenteric colonic perforation was evident. At the dorsal aspect, the abscess cavity communicated with a 7–10 mm gastric perforation in the dorsal gastric wall 3 cm from the greater curvature. Four small (5 mm × 7 mm) cylindrical magnets covered with cloth were found wedged in the perforation. The magnets were firmly adhered to each other, and were separated with difficulty. The colonic and gastric perforations were debrided back to vital tissue, then closed routinely. An omental patch was sutured over the site of the colonic repair. The abscess was intimately associated with the splenic vasculature, so a splenectomy was required for adequate debridement of the abscessed tissues. Aerobic and anaerobic bacterial cultures of the abscess were obtained. The abdomen was lavaged with 10 L warmed 0.9% NaCl.[p] Two Jackson-Pratt (JP) drains[q] attached to a closed collection system were placed in the abdomen to provide continuous drainage, to allow daily cytologic monitoring and quantification of abdominal effusion. A nasogastric tube[r] (NG) was placed to the level of the gastric fundus for postoperative removal of gastric residual volume, as well as to allow enteral feeding in the postoperative period.

While in surgery, the patient developed hypotension (systolic blood pressure 75 mm Hg, diastolic blood pressure 25 mm Hg, with MAP of 50 mm Hg) which was not responsive to crystalloid and colloid fluid boluses. Hypoglycemia (glucose 1.22 mmol/L [22 mg/dL]; reference interval 4.1–7.9 mmol/L [74–143 mg/dL]) and hypoproteinemia (total plasma protein 30 g/L [3.0 g/dL]; reference interval 52–82 g/L [5.2–8.2 g/dL] via refractometer) were identified. A bolus of 50 mL of 25% dextrose was administered IV and 2.5% dextrose was added to the crystalloid fluid bag. Blood pressure improved to a systolic blood pressure of 90 mm Hg, diastolic 45 mm Hg with a MAP of 72 mm Hg. The remaining anesthesia was uneventful. Enrofloxacin[s] (10 mg/kg, IV, q 24 h) was added for expanded microbial coverage. The dog was initiated on parenteral nutrition (PN) immediately postoperatively and fed at 66% resting energy requirements, calculated to deliver 6 g protein/100 kcal of fed calories,[t] lipids comprised 31% of total kcal delivered,[u] and dextrose comprised 19.8% of total kcal delivered.[v] The fentanyl, lidocaine, and ketamine CRI was continued for analgesia. Fluid therapy included isotonic crystalloids[b] with 2.5% dextrose and hydroxyethyl starch solution.[h] An indwelling Foley urinary catheter[w] was placed to monitor urine output. Continuous ECG, temperature, heart rate, respiratory rate and effort, noninvasive blood pressure, quantification of drain production, NG suctioning, and peripheral blood glucose concentrations were monitored. Fluid collected from drains were cytologically examined every 24 hours.

Postoperative laboratory testing identified hypokalemia (3.2 mmol/L; reference interval 3.5–5.8 mmol/L), hypoalbuminemia (12 g/L [1.2 g/dL]; reference interval 23–40 g/L [2.3–7.0 g/dL]), and prolonged prothrombin time (14 s; reference interval 9–12 s) and activated partial thromboplastin time (aPTT) (129 s; reference interval 59–87 s). Testing for fibrin degradation products was not performed, but the dog's septic state, in addition to prolongation of coagulation times, led to

a supposition of the development of disseminated intravascular coagulopathy, although coagulopathy secondary to synthetic colloid administration could not be ruled out. Potassium chloride,[x] 30 mEq/L, was added to the crystalloid fluid[b] with 2.5% dextrose. Transfusion of fresh frozen plasma (FFP) 7.8 mL/kg was commenced to address the coagulopathy. The lidocaine/ketamine CRI was discontinued 5 hours postoperatively, and the fentanyl CRI was continued for analgesia. Despite gastric suctioning yielding small quantities of fluid (0–22 mL q 4 h) and prior administration of a central antiemetic, the dog continued to vomit moderate quantities of bile numerous times in the first 8 hours after surgery. Because of the continued vomiting, maropitant[y] (1 mg/kg, IV, q 24 h) therapy was initiated. Blood glucose concentrations remained stable (4.9–5.7 mmol/L [88–103 mg/dL]; reference interval 4.1–7.9 mmol/L [74–143 mg/dL]), and dextrose supplementation was discontinued. Fluid production from JP drain ranged from 6 to 11 mL/k/h.

On Day 1 after surgery, 2 additional units of FFP (15.3 mL/kg in total) were administered due to continued prolongation of prothrombin time (14 s) and aPTT (141 s) and continued hypoalbinemia (14 g/L [1.4 g/dL]). Platelet estimate from a blood smear was 130.5×10^9/L [130,500/μL] with an average of 8.7 platelets per high-power field. Because of recurrent hypoglycemia, 2.5% dextrose was added to the IV crystalloids. Initial abdominal fluid cytology from the JP drains yielded moderate numbers of degenerate neutrophils with intracellular rods in numbers much higher than normally expected 24 hours after surgery, in the surgeon's clinical experience. Enrofloxacin was discontinued and amikacin[z] (20 mg/kg, IV, q 24 h) was added to the antimicrobial regime for increased spectrum against enteric pathogens and improved penetration into abscessed tissues and abdominal fluid. Because of the persistence of bacteria on subsequent samples of abdominal fluid, peritoneal lavage (1 L 0.9% NaCl[aa] with 15,000 U heparin[bb] once, then 1 L Lactated Ringer's solution[cc] with 5000 U heparin for each remaining lavage) was instituted every 8 hours aseptically via the JP drains to dilute and remove bacteria and inflammatory mediators, reduce fibrin deposition and potential adhesion formation, decrease clogging of the drain fenestrations, and maximize fluid recovery. PN was continued and increased to meet the dog's full resting energy requirements, delivering protein at 6 g/100 kcal of fed calories, with lipids comprising 45% of total kcal delivered, and dextrose comprising 30% of total kcal delivered. Laboratory testing identified static hypoalbuminemia (13 g/L [1.3 g/dL]) and improved aPTT (97 s).

On the second day after surgery, the dog was ambulatory and began to eat small amounts of food. Blood glucose concentration remained within reference interval (3.9–5.4 mmol/L [70–98 mg/dL) and dextrose supplementation was discontinued. Hetastarch was continued for oncotic support attributable to continued hypoalbuminemia (14 g/L [1.4 g/dL]). Drain fluid production ranged from 1.4 to 8 mL/kg/h, and abdominal fluid cytology was deemed to have improved, with only occasional extracellular cocci and degenerate neutrophils with no intracellular bacteria noted. On the third day after surgery, the dog was bright and alert. He continued to eat well. Blood glucose concentrations remained within reference interval. The fentanyl CRI was discontinued and buprenorphine[dd] was commenced at 10 μg/kg, IV, every 8 hours. Drain fluid production markedly decreased, and abdominal fluid cytology revealed no evidence of extracellular or intracellular bacteria. The dog's hypoalbuminemia had improved (16 g/L [1.6 g/dL]). Both drains and the NG tube were removed and the dog was weaned off PN.

The dog was discharged home 4 days after surgery and prescribed amoxicillin with clavulanic acid[ee] 500 mg, PO, every 12 hours, cefpodoxime[ff] 100 mg, PO, every 24 hours, metronidazole 250 mg, PO, every 12 hours, famotidine 20 mg, PO, every 24 hours, and tramadol[gg] 75 mg, PO, every 8–12 hours. Bacterial culture results were reported 2 days later. The anaerobic culture yielded *Proteus mirabilis*, while the aerobic culture yielded 2 strains of *Escheria coli*, *Proteus mirabilis*, and an *Enterococcus* species. Because all bacterial species were susceptible to amoxicillin/clavulanic acid, this antimicrobial was continued for 6 weeks. The source of the magnets was subsequently identified to be from a toy with magnets that was chewed by the dog.

Discussion

In people, multiple magnet ingestion can lead to serious GI complications.[1–7] Interestingly, a recent case series reported gastric and intestinal wall necrosis in 2 dogs following ingestion of multiple magnets with no evidence of peritonitis and uncomplicated recoveries.[8] The present case report describes a dog that developed septic peritonitis secondary to gastric and colonic perforations from ingestion of multiple magnets. Based on lesions noted at surgery, it is postulated that the magnets had been ingested at separate times, allowing one set of the magnets to reach the colon unrestricted until the subsequent magnets were ingested. Once in the stomach, it is presumed the second set of magnets were attracted to the colonic set, resulting in compression of the gastric and colonic walls between the magnets, necrosis, and perforation with resultant abscess formation and septic peritonitis.

Pneumoperitoneum can be challenging to identify on survey radiographs when only a small amount of free air present.[10] Horizontal beam radiography or abdominal ultrasound are additional modalities that can aid in the detection of free abdominal air. In this case, radiologist review identified several questionable gas opacities in the cranial abdomen on the lateral projection, but not on the ventrodorsal projection. These opacities were not identified when the dog was first hospitalized, which contributed to a delay in pursuing exploratory surgery. In addition, diagnostic peritoneal lavage may have aided in confirming the presence of a septic exudate earlier in the course of treatment.

Septic peritonitis can be treated with intraoperative lavage and debridement alone,[13] debridement and lavage coupled with open abdominal drainage,[14] with closed suction drainage,[14] or vacuum-assisted closure systems.[15] Closed suction drainage via fenestrated silicone drains was chosen for this dog for a number of reasons including the ability to close the abdomen while removing accumulating fluid, bacteria and inflammatory by-products from the abdomen continuously, for possible reduction in nosocomial infection risk, for ease of postoperative wound and bandage care, and to eliminate the need for repeated sedations or anesthesia.

The ease of abdominal fluid collection via the drain allowed for frequent cytological analyses, which was used to gauge the dog's progress in resolving the infection. However, care must be exercised in interpreting cytology results obtained in this manner because bacterial colonization within the drain system may falsely lead to conclusion of continued septic peritonitis. The cytological examination performed 24 hours after surgery was deemed to represent continued septic peritonitis as suggested by the presence of intracellular bacteria. In retrospect, direct abdominocentesis could have been used to confirm this suspicion.

Abdominal lavage with crystalloids and heparin was instituted to dilute residual bacteria and inflammatory mediators. Heparin was added to the lavage solution to help improve fluid recovery by decreasing intra-abdominal accumulations of fibrin that potentially block drain fenestrations, and to prevent further abdominal adhesion formation,[16–20] decreasing fluid loculation that could harbor continued bacterial growth. Abdominal lavage with heparin has been described in equine medicine[16] and is an accepted therapy for patients with abdominal contamination or septic peritonitis.[18] Use of heparin in lavage solution for treatment of pyothorax in small animals is commonly performed,[20] but has only been discussed in experimental canine models designed to prevent adhesion formation.[hh] Intraperitoneal or subcutaneous heparin administration without abdominal lavage has been demonstrated to be effective in reducing the experimental formation of abdominal adhesions in both rats[19] and dogs.[21] While aseptic technique was used, introducing bacteria from the drains into the abdomen is possible. Using in situ drains as the vehicle through which to instill lavage is not without risk, but is used commonly when treating pyothorax in dogs,[20] or in treating peritonitis in horses.[16–18]

Aggressive postoperative management was believed to be integral to a timely and successful recovery for the dog described in this case report. The daily removal of protein-rich fluid through the closed suction drains likely contributed to the persistent hypoalbuminemia. Hypoalbuminemia has been identified as a significant risk factor associated with leakage following intestinal surgery.[22] Patients with significant albumin deficits can benefit from aggressive nutritional support and may be helped in the short term with human serum albumin (HSA) or canine serum albumin. Concerns for reported complications[23] associated with administration of HSA in dogs was the reason this treatment option was not used, although 1 study postulated that ill dogs may respond differently to HSA than healthy dogs.[24] Although a commercially available canine serum albumin solution has been marketed, it was not available at the authors' hospital. Plasma and hetastarch were used to increase oncotic pressure, which was presumed to be decreased due to the continued hypoalbuminemia.

The dog's coagulopathy prompted therapy with FFP. Given the severe peritonitis identified during surgery, the abnormalities in hemostasis were deemed to be reflective of disseminated intravascular coagulopathy secondary to sepsis. However, acquired platelet dysfunction associated with hetastarch administration has been reported. In addition, decreases in factor VIII and von Willebrand factor concentrations have also been associated with synthetic colloid use and cannot be fully discounted in this case.[25] Assaying coagulation parameters before administration of hetastarch could have identified a preexisting coagulopathy, potentially prompting the use of FFP earlier in the course of therapy. Measuring fibrin degredation products and platelet numbers, in addition to evaluating red blood cell morphology would have aided in determining the nature of the coagulopathy, but would not have significantly changed the therapy postoperatively.

A recent report[8] discusses 2 cases of GI disease secondary to multiple magnet ingestion. In 1 case GI necrosis without perforation was identified, and in the second case there was intestinal perforation without evidence of peritonitis reported.[8] In contrast to the progression of disease in the previously reported cases, the

current case highlights serious sequelae to ingestion of magnets. Interestingly, the metallic density in the radiographs from 1 case closely mirrors the radiographs obtained for the current case reported. In human medicine, there is a great reliance on a history of witnessed or suspected ingestion of magnets to make a presumptive diagnosis of multiple magnet ingestion[1–7] because radiographic appearance cannot differentiate between magnets or other metallic material. Unless magnet ingestion is witnessed in an animal, the initial course of action to the identification of a metallic foreign body on radiographs may be to presume that the foreign material could pass without difficulty, especially if the material appears small in size. Therefore, animals exhibiting GI signs with confirmed small metallic foreign bodies apparent on radiographs should have magnet foreign bodies as a consideration or possible differential. In these cases, or in patients known to have ingested magnets, the immediate removal of the foreign material is strongly recommended. As this case illustrates, significant morbidity can result from multiple magnet ingestion, and veterinarians should be aware of this type of foreign body and its potential for causing GI necrosis and perforations.

Footnotes

[a] Surgivet, Waukesha, WA.
[b] Plasmalyte A, Baxter Healthcare Corporation, Syracuse, NY.
[c] Hydromorphone (Dilaudid), Abbott Laboratories, North Chicago, IL.
[d] Dolasetron (Anzemet), Aventis Pharmaceuticals Inc, Bridgewater, NJ.
[e] Famotidine (Pepcid), Baxter Healthcare Corporation.
[f] Metronidazole (Flagyl), Hospira Inc, Lake Forest, IL.
[g] Ampicillin sulbactam (Unasyn), Baxter Healthcare Corporation.
[h] 6% Hetastarch, Hospira Inc.
[i] Arrow International Inc, Reading, PA.
[j] Fentanyl citrate, Baxter Healthcare Corporation, Deerfield, IL.
[k] Midazolam HCl, Hospira Inc.
[l] Propofol (PropoFlo), Abbott Laboratories.
[m] Isoflurane (Isoflo), Abbott Laboratories.
[n] Lidocaine HCl, Agri Laboratories Ltd, St Joseph, MO.
[o] Ketamine HCl, Vedco Inc, St Joseph, MO.
[p] 0.9% NaCl, Hospira Inc.
[q] Jackson-Pratt drain, Cardinal Health, McGaw Park, IL.
[r] MILA International, Erlanger, KY.
[s] Enrofloxacin (Baytril), Bayer Healthcare, Shawnee Mission, KA.
[t] Freamine III 10%, B. Braun Medical, Irvine, CA.
[u] Liposyn 20%, Hospira Inc.
[v] Dextrose 50%, Vedco Inc.
[w] SurgiVet.
[x] Potassium chloride, Hospira Inc.
[y] Maropitant citrate (Cerenia), Pfizer Animal Health, New York, NY.
[z] Amikacin sulfate, Bedford Laboratories, Bedford, OH.
[aa] 0.9% NaCl, Hospira Inc.
[bb] Heparin, APP Pharmaceuticals LLC, Schaumburg, IL.
[cc] Lactated Ringer's solution, Hospira Inc.
[dd] Buprenorphine (Buprenex), Bedford Laboratories.
[ee] Amoxicillin/Clavulanic acid (Clavamox), Pfizer Inc, New York, NY.
[ff] Cefpodoxime proxetil (Simplicef), Pfizer Inc.
[gg] Tramadol (Ultram), Amneal Pharmaceuticals, Hauppauge, NY.
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