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CASE REPORT

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Gastrointestinal obstruction secondary to activated charcoal granule impaction in a dog

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Abstract

Objective: To describe a serious adverse event of gastrointestinal obstruction requiring surgery following routine administration of multiple doses of activated charcoal (AC) granules, which were prescribed for carprofen toxicosis.

Case Summary: A 2-year-old female neutered Airedale Terrier presented for ingestion of 207 mg/kg of carprofen. Decontamination was initiated with apomorphine to induce emesis. Along with additional supportive care, the dog received an initial dose of 75 mL of AC suspension containing sorbitol by mouth (15.6 g of AC, or 0.6 g/kg), followed by 50 g of AC granules every 8 hours for 4 additional doses. While hospitalized, the dog experienced clinical signs, including vomiting and black diarrhea, as well as bloodwork changes including mild to moderate elevations in kidney and liver enzymes. Given clinical improvement after 72 hours of hospitalization, the patient was discharged for monitoring and ongoing care at home. Two days later, the patient presented again for nausea, dark diarrhea with frank blood, and panting. Abdominal ultrasound showed findings suspicious for partially obstructive foreign material or atypical impacted fecal material partially occluding the distal ileum. Despite medical management overnight, recheck ultrasound the following day demonstrated persistent obstruction with ileal foreign material. Exploratory laparotomy and enterotomy revealed moderate distension and obstruction of the distal ileum with black granular foreign material consistent with charcoal granules. The patient remained in hospital for supportive care for 4 days following the procedure, and all clinical signs were resolved at the time of discharge.

New or Unique Information Provided: This report documents a serious adverse event of gastrointestinal obstruction associated with routine multidose AC administration, which has been occasionally reported in people but not in dogs. The potential for this complication should be taken into account when prescribing multiple doses of AC granules.

KEYWORDS
canine, carprofen, decontamination, nasogastric, obstruction, toxicant

Abbreviations: AC, activated charcoal; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; COX, cyclooxygenase; GI, gastrointestinal; NG, nasogastric; NSAID, nonsteroidal anti-inflammatory drug; RI, reference interval

1 | INTRODUCTION

Activated charcoal (AC) is administered commonly to veterinary patients and occasionally to people following recent ingestion of a potentially toxic substance. The surface of AC is composed of carbon moieties that provide a large surface area for adsorption of toxicants.[1] Many AC products also contain a cathartic used to decrease gastrointestinal (GI) transit time and promote fecal expulsion. While single dose AC is more common, particular toxicants and circumstances may warrant multidose administration of AC. These scenarios include ingestion of drugs that undergo enterohepatic or enteroenteric recirculation (eg, carprofen, ibuprofen, ivermectin), drugs with a long half-life (eg, naproxen), or delayed-release products.[2-5]

Despite widespread use of AC in veterinary medicine and intermittent use in people, there are relatively infrequent reports of associated adverse effects in the human or veterinary literature. In people, the majority of adverse events reported are related to vomiting or regurgitation with subsequent aspiration of charcoal into the lungs or accidental direct administration of charcoal into the lungs via a misplaced nasogastric (NG) tube.[1,2,6] While constipation is reported in people, it is typically mild and does not generally require treatment.[1,2] There is one report of GI obstruction in a person after single-dose AC therapy, while GI obstruction requiring manual evacuation or surgery has been reported rarely in people following treatment with multidose AC.[2,7-14]

GI obstruction associated with administration of AC has not been reported in a dog to the authors' knowledge. The purpose of this case report is to document evidence of GI obstruction requiring surgery following routine administration of multidose AC therapy prescribed for a dog for carprofen intoxication.

2 | CASE REPORT

A 2-year-old female neutered Airedale Terrier weighing 25.2 kg was presented to the emergency service of a veterinary teaching hospital 1.5 hours after ingestion of 55 tablets of 75 mg carprofen[a] and 11 tablets of 100 mg carprofen[*] (total of 5,225 mg, or 207 mg/kg). The chewable carprofen tablets were prescribed during a visit to the same hospital's general practice earlier that day, where the dog had been evaluated for hip pain. A kidney panel at the general practice visit was unremarkable, with a BUN of 7.1 mmol/L (20 mg/dL; reference interval [RI] 3.9–11.8 mmol/L [11–33 mg/dL]) and creatinine of 79.6 μmol/L (0.9 mg/dL; RI 70.7–132.6 μmol/L [0.8–1.5 mg/dL]).

On presentation to the emergency service following carprofen ingestion, the patient was bright, hydrated, and had normal vital parameters. The dog had a tense abdomen, formed feces on rectal examination, and no other significant findings on physical examination. Immediately following presentation, the dog was administered apomorphine[†] 1 mg (0.04 mg/kg, IV) and vomited a small volume of brown material containing several carprofen tablets. The dog was admitted to the hospital, where it received intralipid 20% fat emulsion[‡] (0.25 mg/kg/min, IV) for 1 hour (375 mL total) and was administered lactated Ringer's solution[§] (60 mL/h [2.4 mL/kg/h, IV). The dog initially received 75 mL of AC suspension containing sorbitol[**] by mouth (15.6 g of AC, or 0.6 g/kg) and was subsequently treated in hospital with IV maropitant,[††] IV ondansetron,[‡‡] IV pantoprazole,[§§] PO sucralfate,[***] and PO misoprostol,[†††] as well as PO trazodone[‡‡‡] for anxiety. The dog was given an additional 50 g (2.0 g/kg) of AC granules[§§§] syringe-fed with water by mouth every 8 hours for 4 additional doses.

The patient was clinically normal over the first 24 hours of hospitalization, aside from marked anxiety. Recheck BUN was 7.9 mmol/L (22 mg/dL), and creatinine was 79.6 μmol/L (0.9 mg/dL). The dog vomited following discontinuation of antiemetic therapy after 24 hours, and ondansetron was restarted. During the second day of hospitalization, the dog's appetite declined, and the dog developed a scant volume of malodorous black diarrhea. Due to mild interstitial edema, the fluid rate was decreased to 35 mL/h (1.4 mL/kg/h, IV). Recheck BUN was 8.9 mmol/L (25 mg/dL) and creatinine was 70.7 μmol/L (0.8 mg/dL). The serum biochemistry panel also revealed a mild mixed hepatopathy with an alanine aminotransferase (ALT) of 155 U/L (RI 21–72 U/L), aspartate aminotransferase (AST) of 232 U/L (RI 20–49 U/L), and alkaline phosphatase (ALP) of 227 U/L (RI 14–91 U/L). The dog vomited again and was restarted on maropitant. On the third day of hospitalization, the patient remained inappetent, reluctant to drink available water, and anxious but was bright, alert, and responsive and had no further vomiting or progressive diarrhea. The dog's abdomen remained tense but not overtly painful. Compared to overhydration noted the day prior, the dog appeared appropriately hydrated. The BUN increased to 10 mmol/L (28 mg/dL) and creatinine to 114.9 μmol/L (1.3 mg/dL). There was also mild progression of the mixed hepatopathy with ALT 317 U/L, AST 334 U/L, ALP 243 U/L, and total bilirubin 5.1 μmol/L (0.3 mg/dL; RI 0.0–3.4 μmol/L [0.0–0.2 mg/dL]).

Given clinical improvement after 72 hours of hospitalization and the persistence of marked anxiety in hospital, the patient was discharged home for ongoing supportive care and feeding in a less stressful environment for the patient. Home care included maropitant[****] (60 mg, PO) once daily for 3 days, sucralfate (1 g slurry, PO) every 8 hours for 7 days, omeprazole[††††] (20 mg, PO) every 12 hours for 7 days, and a home-cooked bland diet of boiled chicken, cottage cheese, and boiled white rice. It was recommended that the dog return in 1 week for a recheck serum biochemistry panel and urine specific gravity measurement or earlier if clinically unwell. During a follow-up phone call the day after discharge, the client reported that the dog was lethargic, uninterested in chicken soup, vomiting, having diarrhea, and polydipsic. The client suspected that the patient did not receive maropitant due to vomiting soon after administration. The client declined a recheck examination and elected to add famotidine[‡‡‡‡] and ondansetron[§§§§] to the medication regimen at home. The client was strongly advised to have the patient reassessed if vomiting and anorexia persisted.

Two days following discharge from the hospital, the patient presented to the emergency service for nausea, dark diarrhea with frank blood, and panting. On physical examination, the dog was quiet, panting, and 5% dehydrated. The rectal temperature was normal at 38.2°C (100.8°F), and the heart rate was 120/min with strong pulse quality. The dog's abdomen was moderately tense and apparently painful on palpation, and it had a marked volume of diarrhea with hematochezia.

An IV catheter was placed, and the dog was treated with fentanyl[*****] for analgesia. Initial CBC revealed a hematocrit of 0.50 L/L (50%; RI 0.40–0.55 L/L [40–55%]), WBC 12.2 × 10^9/L (12.2 × 10^3/μL; RI 6.0–13.0 × 10^9/L [6.0–13.0 × 10^3/μL]), neutrophils 4.4 × 10^9/L (4.4 × 10^3/μL; RI 3.0–10.5 × 10^9/L [3.0–10.5 × 10^3/μL]) with moderate toxicity, and bands 2.9 × 10^9/L (2.9 × 10^3/μL) with moderate toxicity. Initial chemistry panel values included BUN 21.8 mmol/L (61 mg/dL) and creatinine 176.8 μmol/L (2.0 mg/dL). Liver values were ALT 190 U/L, AST 35 U/L, ALP 198 U/L, and total bilirubin 8.6 μmol/L (0.5 mg/dL).

An abdominal ultrasound[††††††] performed shortly after presentation showed marked fluid dilation of the distal ileum proximal to lobular and tubular shadowing material that extended from the distal ileum to the transverse colon. Findings were suspicious for partially obstructive foreign material or atypical impacted fecal material partially occluding the distal ileum. There was no evidence of overt gastric ulceration, no hyperechoic mesentery, and no free peritoneal fluid or gas.

The options for medical management with recheck ultrasound versus exploratory laparotomy were discussed with the client. Given anesthetic concerns regarding recent kidney and liver value abnormalities, as well as surgical concerns for existing GI injury, medical management was elected. The dog was admitted to the hospital for overnight monitoring and supportive care to determine whether the partial obstruction could be relieved through rehydration and other medical means. The dog received IV fluid therapy, fentanyl, ondansetron, pantoprazole, and sucralfate. Given concern for the inflammatory and left-shifted CBC potentially representing bacterial GI translocation, the dog was also started on ampicillin-sulbactam[‡‡‡‡‡] (50 mg/kg, IV, q 8 h). During the first 24 hours of hospitalization, the dog had a persistent moderate volume of diarrhea with hematochezia. No further vomiting was noted, but there continued to be moderate discomfort on abdominal palpation. Twenty-four hours after hospitalization, the dog was assessed to be appropriately hydrated. A recheck serum kidney panel on the second day of hospitalization showed improved BUN of 15.7 mmol/L (44 mg/dL) and creatinine of 123.8 μmol/L (1.4 mg/dL).

A focused recheck ultrasound of the GI tract was performed on the second day of hospitalization. While the distal jejunum and ileum were no longer fluid distended, the shadowing material previously identified remained within the ileum, which was comparably distended to the prior ultrasound. No peritoneal gas was present, as previously noted, but a new scant volume of abdominal effusion and a focal region of peritoneal hyperechogenicity were present adjacent to the distal ileum. Ultrasound findings were consistent with unchanged ileal foreign material with focal mesenteric reactivity representing a chronic partial obstruction.

Surgical intervention was elected given the persistent obstructive foreign material within the distal ileum, with no change despite rehydration and supportive care, along with ongoing patient abdominal discomfort. The patient was anesthetized, and a standard ventral midline celiotomy was performed to explore the abdomen. Exploratory celiotomy revealed a site of impaction, most similar to sand or other granular material on palpation, located at the distal ileum approximately 10 cm proximal to the ileocecocolic junction. The ileocecocolic junction and distal ileum were markedly edematous and dilated, although there was no evidence of perforation of the affected bowel segment, and orad ileum and jejunum were distended and gas filled. Remaining abdominal organs were grossly unremarkable. An attempt to digitally manipulate the foreign material through the ileocecocolic junction into the colon was unsuccessful. An approximately 2 cm enterotomy was performed in the proximal ileum orad to the impaction, and a large amount of black granular material consistent with charcoal granules was retrieved from the lumen using a surgical gall bladder spoon (Figure 1). The intestinal lumen was copiously lavaged with warmed sterile saline[§§§§§] until no further granules were identified. The enterotomy site was apposed using 3-0 polydioxanone[******] in a simple interrupted pattern. The ileal lumen was distended with saline, and no leak was detected along the enterotomy site. The abdomen was lavaged with sterile saline and closed in a standard 3-layer fashion.

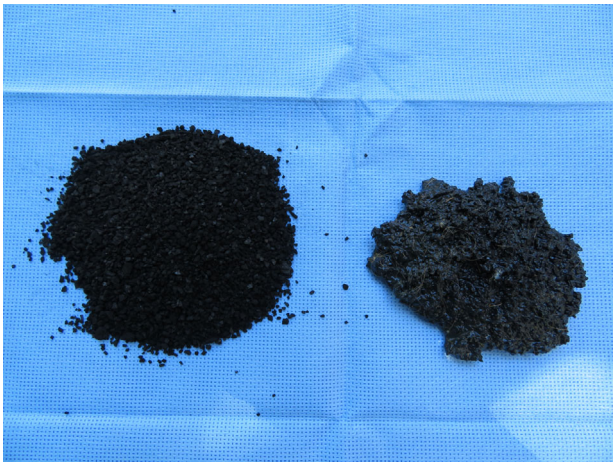

FIGURE 1 Comparison of normal charcoal granules (left) versus impacted charcoal foreign material recovered from the site of obstruction (right)

The patient recovered from anesthesia uneventfully and remained in hospital for 4 additional days for ongoing supportive care. An NG tube was placed to help alleviate accumulation of excess gastric fluid and to initiate enteral feeding. Feedings initiated the second day after surgery were tolerated well, and the patient was eating voluntarily prior to discharge. Diarrhea diminished and hematochezia resolved. No further vomiting or regurgitation was noted. A kidney panel on the day prior to discharge showed normalization of kidney values: BUN 5 mmol/L (14 mg/dL) and creatinine 61.9 μmol/L (0.7 mg/dL). The dog was discharged from the hospital with standard instructions for postoperative monitoring and care, with prescribed medications including maropitant, omeprazole, sucralfate, amoxicillin-clavulanic acid,[†††††††] and tramadol.[‡‡‡‡‡‡]

Six days following discharge, the patient visited for a recheck and was doing well. The client reported return to an excellent appetite and normal energy level, with improved stool quality. The dog's ventral abdominal incision site was healing well. Recheck kidney values showed BUN of 3.9 mmol/L (11 mg/dL) and creatinine 97.2 μmol/L

(1.1 mg/dL). Five months following final recheck, the owners verbally confirmed that the dog had complete resolution of clinical signs and was doing well at home.

3 | DISCUSSION

This report describes a serious adverse event of GI obstruction with AC that necessitated enterotomy after routine administration of multiple doses of AC granules for massive carprofen overdose. While GI obstruction with AC has been reported occasionally in people following AC therapy, this complication has not been previously documented in dogs.[2,7-14]

In the human literature, GI obstruction requiring medical therapy or surgical intervention is discussed as an uncommon complication associated with multidose AC therapy, and there is only 1 report of GI obstruction following treatment with single-dose AC.[2,7-14] One study evaluating the frequency of complications associated with multidose AC therapy reviewed 878 patients and found that none experienced GI obstruction.[6] Watson et al first reported a case of GI obstruction suspected to be secondary to the use of multiple doses of AC following carbamazepine intoxication.[7] The patient was managed medically with magnesium citrate and multiple saline enemas.

Additional reports exist in the human literature detailing cases requiring surgical intervention for GI obstruction. Ray et al described a man who received multidose AC following amitriptyline overdose and subsequently required a laparotomy to remove a charcoal bezoar in the distal ileum.[9] Atkinson et al reported a man intoxicated with barbiturates and benzodiazepines who received AC via NG tube over 18 hours.[10] The patient ultimately required a hemicolectomy due to a large bolus of inspissated charcoal in the cecum. Goulbourne and Cisek described the case of a woman treated for theophylline toxicity with multidose AC who required an ileotransverse colostomy for a large charcoal aggregate causing obstruction at the distal ileum.[11] The patient also had previously asymptomatic adhesions at the ileocecal valve from a prior hysterectomy. Gomez et al reported a woman who was receiving chronic methadone and ingested an overdose of amitriptyline, for which multidose AC was prescribed.[12] A subsequent exploratory laparotomy found a colonic perforation with an obstructing charcoal mass. Merriman and Stokes described a boy treated with 2 doses of AC without a cathartic agent following overdose of a tricyclic antidepressant, opioid, and benzodiazepine.[13] A small intestinal obstruction with a large charcoal bezoar was identified subsequently at surgery.

Green and McCauley detail the sole case report of bowel obstruction and perforation following single-dose AC.[14] A woman presented subsequent to a drug overdose, including opioids and benzodiazepines, and was administered AC without sorbitol. A laparotomy 3 days later revealed fecal and charcoal peritonitis secondary to perforation of the sigmoid colon. Histopathology demonstrated previously undiagnosed diverticular disease, with perforation near a diverticulum.

While definitive underlying causes of charcoal obstruction have not been established, some similarities exist in these cases that may have predisposed patients to GI obstruction. Several of the cases involved intoxication with drugs that may have reduced GI peristalsis or motility.[7,9,10,12-14] Many critically ill patients are prone to ileus, may be taking medications that slow the GI tract, and can have underlying comorbidities that contribute to decreased GI motility. Under the circumstances of reduced intestinal motility, especially compounded by dehydration, large volumes of normally innocuous medications or material may become inspissated and result in obstruction. Given that the majority of cases described in the literature report obstructions in the distal small intestine or colon, and none are described as gastric outflow or duodenal obstructions, fluid absorption from the GI and inspissation of the charcoal appear likely to play a role. The dog in this case report follows the pattern of a distal GI obstruction. This has been demonstrated with other granular material in dogs, as 2 reports of sand impactions also described obstructions at the terminal small intestine requiring intensive medical management or surgical management.[15,16] Additionally, anatomical abnormalities may predispose patients to obstruction with charcoal or any other foreign material such as those observed in patients with prior intestinal adhesions or diverticular disease.[11,14]

In terms of predisposing factors for the dog described in this report, the dog was not known to have any underlying GI disease and was hydrated while in hospital. However, after discharge from the first emergency hospitalization, the dog continued to have GI fluid losses at home and represented to the emergency service clinically dehydrated, which may have inhibited hydration of the intestinal tract and movement of the charcoal ingesta. The patient did receive medications while hospitalized, including trazodone and fentanyl, which potentially could have contributed to constipation; however, the patient exhibited ongoing diarrhea while hospitalized and at home. Though therapy was initiated early and optimized in hospital to support the GI tract, the dog did ingest a 207 mg/kg dose of carprofen, which is well above the reported GI toxic dose range of 20–22 mg/kg.[17,18] As a nonsteroidal anti-inflammatory drug (NSAID), carprofen inhibits cyclooxygenase (COX) enzymes and reduces formation of prostaglandins. While carprofen is considered more selective for the COX-2 isoform in dogs and thus may spare some of the GI and renal effects of nonselective COX inhibitors, all NSAIDs alter both COX-1 and COX-2 in cases of excessive exposure.[17,18] Since prostaglandins normally mediate many aspects of GI health including stimulation of mucus and bicarbonate production, inhibition of gastrin and hydrochloric acid secretion, regulation of blood flow, epithelial cell turnover, and mucosal leukocyte function, inhibition of prostaglandin synthesis can result in GI ulceration, mucosal barrier compromise, hemorrhage, and perforation.[18-21] Although it is unknown to what degree the carprofen overdose played a role in predisposing this patient to obstruction, it is plausible that carprofen intoxication could have altered GI motility and contributed to GI hydration deficits.

Although a specific dose of AC that may predispose a patient to charcoal obstruction is unknown, obstruction has mainly been reported in multidose treatment regimens in people. The case reports involving GI obstruction in people are quite varied in the doses received.[7-14] The recommended doses for AC for dogs are in the range of 1–5 g/kg for

the initial dose.[3-5] When powdered AC is to be combined with water, the recommended dose is approximately 1 g AC per 5 mL water.[3] When prescribing repeated doses, which is commonly recommended for NSAIDs given their enterohepatic recirculation and variably long half-lives, suggested protocols include administration of AC every 4 to 8 hours for 1–3 days.[3-5] Cathartics have been advocated for use in conjunction with AC on the first dose to speed transit time of toxicants through the GI tract, promote fecal excretion, and thus, decrease time for toxicant absorption from the GI.[3-5] As the patient in this case was administered 75 mL of AC plus sorbitol for the first dose (15.6 g of AC alone, or 0.6 g/kg), followed by 2 g/kg of AC granules PO every 8 hours for 4 additional doses, this protocol fell well within the recommended dose and frequency ranges.

In general, adverse effects of AC administration are relatively uncommon. In people and dogs, vomiting or regurgitation has been reported most commonly, with increased risk associated with concurrent administration of sorbitol cathartics.[1-6,22] While the unpalatable taste and texture of charcoal or its rate of administration may contribute to emesis, vomiting is also thought to occur subsequent to recent administration of emetic agents used for GI decontamination. Pulmonary complications can include aspiration of charcoal into the lungs or accidental direct administration of charcoal into the lungs via a misplaced feeding tube.[1-4,6,22] Additional GI complications may include diarrhea (particularly secondary to cathartic use), black stools (which may make identification of melena more difficult), and constipation (though typically not severe enough to require treatment).[1-4] Hypernatremia is also a rare complication reported in both people and dogs, particularly in cases of multiple doses of AC and sorbitol combinations resulting in osmotic water losses.[2-6] Hypernatremia appears to be more commonly reported in small dogs receiving multiple doses of AC but has been described in large dogs, patients that received only a single dose, and patients that received no cathartics.[3,4] Finally, a study in 6 healthy volunteer dogs found that an AC suspension containing propylene glycol and glycerol administered once at 4 g/kg increased mean serum osmolality, osmolal gap, and plasma lactate concentrations, thus, potentially complicating assessment of an intoxication.[23] These dogs also experienced vomiting, lethargy, and polydipsia. Given the findings in this case report, as well as evidence described in the human literature, it is apparent that GI obstruction should also be considered as a potential adverse event of multidose AC charcoal administration in dogs, though it may be rare.

A recent study compared the efficacy of single-dose AC, single-dose AC with sorbitol, and multidose AC for the reduction of plasma carprofen concentrations following experimental overdose of 120 mg/kg of carprofen in dogs.[24] The single doses of AC with or without sorbitol were administered at 2 g/kg AC at 1 hour following carprofen ingestion, while this dose was repeated every 6 hours for a total of 4 doses in the multidose protocol. Measurement of plasma carprofen concentrations over 36 hours demonstrated that a single dose of AC or AC with sorbitol was as effective as multidose AC in reducing carprofen concentrations in experimental dogs. Additionally, the multidose protocol resulted in significantly more vomiting compared to the single dose protocols. Results of this study may suggest that multidose AC is not required for treatment of otherwise healthy dogs with moderate carprofen overdose, although further studies are likely needed to compare the efficacy of different treatment regimens in clinical patients. As multidose charcoal is not without risks, including the potential for GI obstruction, it would be ideal to reduce unnecessary administration of AC when possible. It is unknown in people or dogs whether liquid charcoal formulations may decrease the likelihood of obstruction compared to dry powdered or granule formulations, and this may be a source of further investigation.

The outcome of this case, in conjunction with adverse events reported in the human literature, support a recommendation to monitor patients for hydration status and signs of obstruction while administering charcoal. Careful consideration of the need for multiple doses of AC in individual circumstances is also warranted. This may be particularly relevant for patients with underlying comorbidities or exposure to medications that may decrease normal GI motility.

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ENDNOTES

* Carprofen (Rimadyl), Zoetis Inc, Kalamazoo, MI.
† Apomorphine HCl, Medisca, Plattsburgh, NY.
‡ Intralipid 20%, Baxter Healthcare, Deerfield, IL.
§ Lactated Ringer's solution, Abbott Laboratories, North Chicago, IL.
** Actidose with sorbitol, Paddock Laboratories Inc, Minneapolis, MN.
†† Maropitant citrate injectable (Cerenia), Zoetis Inc, Kalamazoo, MI.
‡‡ Ondansetron injectable, West-Ward Pharmaceuticals, Eatontown, NJ.
§§ Pantoprazole (Protonix), Wyeth Pharmaceuticals Inc, Philadelphia, PA.
*** Sucralfate, Nostrum Laboratories, Kansas City, MO.
††† Misoprostol, Greenstone Ltd, Peapack, NJ.
‡‡‡ Trazodone hydrochloride, Qualitest Pharmaceuticals, Huntsville, AL.
§§§ Activated charcoal granules, Norit Americas Inc, Marshall, TX.
**** Maropitant oral (Cerenia), Zoetis Inc, Kalamazoo, MI.
†††† Omeprazole, Sunmark, San Francisco, CA.
‡‡‡‡ Famotidine oral, West-Ward Pharmaceuticals, Eatontown, NJ.
§§§§ Ondansetron PO, GlaxoSmithKline, Durham, NC.
***** Fentanyl citrate, West-Ward Pharmaceuticals, Eatontown, NJ.
††††† Philips iE33 ultrasound, Philips Ultrasound, Bothell, WA.
‡‡‡‡‡ Ampicillin-sulbactam (Unasyn), Pfizer Animal Health, Kalamazoo, MI.
§§§§§ 0.9% sodium chloride irrigation, Baxter Healthcare Corporation, Deerfield, IL.
****** 3-0 PDS Ethicon, Novartis Animal Health, Greensboro, NC.
†††††† Amoxicillin-clavulanic acid (Clavamox), Pfizer Animal Health, Kalamazoo, MI.
‡‡‡‡‡‡ Tramadol, Amneal Pharmaceuticals, Paterson, NJ.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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