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Case Report

Particularly intriguing cases

Gastroesophageal Intussusception in a German Shepherd

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A 4-year-old, 37-kg (81.6-lb), neutered male German shepherd was referred to the University of Georgia Veterinary Teaching Hospital with a 2-day history of hematemesis and lethargy. The dog had been hospitalized at an emergency clinic and was referred when the vomiting and lethargy worsened despite antiemetic and fluid therapy. The dog had ingested a small, unquantifiable amount of phenylbutazone within 7 days of the onset of clinical signs.

Case Presentation

On presentation, the dog was depressed but responsive and was assessed as 5% dehydrated. He was mildly hyperthermic (103°F [39.4°C]), tachycardic (140 bpm), and panting. His heart sounds were muffled and lung sounds were clear on thoracic auscultation. He was moderately lame (grade 3/5) in both pelvic limbs and had cranial drawer motion consistent with bilateral cranial cruciate ligament rupture. Doppler ultrasonography revealed a systolic blood pressure of 110 mm Hg. During a brief thoracic ultrasonographic examination, the heart was difficult to visualize, but no pericardial effusion was seen.

Diagnostics

Blood gas analysis revealed respiratory alkalosis (P_{CO_2}: 27.9 mm Hg) with compensatory metabolic acidosis and hyperlactatemia (**TABLE 1**). Abnormalities on the complete blood count included a mildly decreased platelet count and leukocytosis characterized by a mature neutrophilia and monocytosis. Blood chemistry abnormalities included hypoproteinemia, hyperglycemia, hypernatremia, hypocalcemia, and hyperbilirubinemia. The urine specific gravity was 1.057 (reference range: 1.030–1.050), but urinalysis revealed no other abnormalities. A coagulation profile was within normal limits.

Diagnostic imaging of the thorax and abdomen was performed. Thoracic radiography revealed a severely dilated thoracic esophagus involving a soft tissue opacity that superimposed the caudal and middle mediastina (**FIGURES 1** and **2**). The heart was ventrally displaced by the dilated esophagus (**FIGURE 1**). On abdominal radiographs, the stomach was not visible, and the liver and spleen were mildly dorsally displaced (**FIGURE 3**). On abdominal ultrasonography, the stomach was still not visualized. Except for mild mesenteric lymph node enlargement, no other ultrasonographic abnormalities were identified. The imaging findings supported a diagnosis of gastroesophageal intussusception (GEI).[1,2]

Treatment

Surgery was performed the same day. The dog was premedicated with diazepam (0.2 mg/kg [0.1 mg/lb] IV), and anesthesia was induced with fentanyl (5 µg/kg [2.3 µg/lb] IV) and lidocaine (2 mg/kg [0.9 mg/lb] IV). Anesthesia was maintained with sevoflurane in oxygen after endotracheal intubation. Exploration of the abdomen found that the esophageal hiatus was enlarged and that a large portion of the stomach had telescoped into the thoracic esophagus. The pylorus and approximately 10 cm (4 inches) of the distal stomach remained in the abdomen. Gentle traction failed to reduce the GEI. The esophageal hiatus was further enlarged by incising the diaphragm ventrally to the xiphoid process, and the stomach was slowly reduced to its normal position using gentle traction at the pylorus and compression of the esophagus cranial to the intussusception. After reduction, the stomach was congested, with dark purple discoloration of the fundus and body. A 20-Fr thoracostomy tube was placed, and the diaphragm was closed with 0 Prolene suture (Ethicon) in a simple continuous pattern from dorsal to ventral. The rest of the abdomen was explored, and no other abnormalities were found. Because the mesenteric lymph nodes appeared large on abdominal ultrasonography, wedge biopsy samples were taken from a jejunal lymph node. Reassessment of the stomach revealed improvement in color to dark pink. A left-sided incisional gastropexy was performed using 0 Prolene suture in a sim-

TABLE 1 Test Results

Test	Value	Reference Range
pH	7.45	7.35–7.45
Pco_2	27.9 mm Hg	29–42 mm Hg
HCO_3^-	19.6 mEq/L	17–24 mEq/L
Lactate	3.3 mmol/L	0–2.5 mmol/L
Leukocytes	28.2 × 10^3/µL	5.5 × 10^3/µL–13.9 × 10^3/µL
Neutrophils	23.688 × 10^3/µL	2.9 × 10^3/µL–12 × 10^3/µL
Monocytes	2.538 × 10^3/µL	0.1 × 10^3/µL–1.4 × 10^3/µL
Platelets	206 × 10^3/µL	211 × 10^3/µL–261 × 10^3/µL
Hematocrit	51.5%	36%–59%
Total protein	5.1 g/dL	5.2–7.3 g/dL
Sodium	158 mmol/L	146–154 mmol/L
Calcium	9.2 mg/dL	9.3–11.4 mg/dL
Total bilirubin	0.40 mg/dL	0–0.2 mg/dL
Prothrombin time	7.6 sec	5.8–9.8 sec
Activated partial thromboplastin time	13.6 sec	9.4–15.1 sec
Thrombin time	6.7 sec	3.7–10 sec
Acetylcholine receptor antibody	0.08 nmol/L	<0.6 nmol/L
Thyroxine	2.0 µg/dL	0.73–2.9 µg/dL
Thyrotropin	0.31 ng/mL	0.02–0.32 ng/mL
Cortisol (baseline)	1.4 µg/dL	0.5–3 µg/dL
Cortisol (1 h after corticotropin stimulation test)	15.5 µg/dL	—

ple continuous pattern, and a 20-Fr de Pezzer gastrostomy tube was placed ventral to the gastropexy. An additional right-sided incisional gastropexy was similarly performed to prevent recurrence of the intussusception. The stomach was reexamined and appeared viable; partial gastrectomy was not performed. The abdomen was thoroughly lavaged and closed routinely.

Postoperative Care

Postoperatively, the dog was monitored in the intensive care unit. Laboratory evaluation revealed a hematocrit of 35%, mature neutrophilia (29 × 10^3 cells/µL), and leukocytosis (31 × 10^3 cells/µL). The total protein concentration had decreased to 3.3 g/dL (reference range: 5.6–7.3 g/dL), and the albumin concentration was 1.5 g/dL (reference range: 3.1–4.1 g/dL). The calcium concentration had decreased to 8.1 mg/dL (reference range: 9.3–11.4 mg/dL), and the sodium and chloride concentrations were high at 158 mmol/L (reference range: 146–154 mmol/L) and 127 mmol/L (reference range: 107–117 mmol/L), respectively. The dog received two 240-mL transfusions of fresh frozen plasma. Clindamycin (10 mg/kg [4.5 mg/lb] IV q8h) and enrofloxacin (7.5 mg/kg [3.4 mg/lb] IV q12h) were administered because of significant gastric compromise. Maropitant citrate (1 mg/kg [0.45 mg/lb] SC q24h) and a constant-rate infusion of metoclopramide (1 mg/kg/d [0.45 mg/lb/d]) were instituted to prevent vomiting. Pantoprazole (1 mg/kg [0.45 mg/lb] IV q24h), famotidine (0.5 mg/kg [0.22 mg/lb] IV q12h), and sucralfate (1 g PO q8h) were administered to treat and prevent esophagitis and gastritis. For analgesia, lidocaine (50 µL/kg/h [22.7 µL/lb/h]) and fentanyl (3 µL/kg/h [1.36 µL/lb/h]) were administered as constant-rate infusions. Intravenous fluids with potassium supplementation (Normosol-R [Abbott Animal Health] with 16 mEq/L of potassium chloride) were administered at 90 mL/kg/d (41 mL/lb/d) IV. After surgery, the patient recovered slowly and did not vomit or regurgitate for 5 days, after which the patient regurgitated once daily.

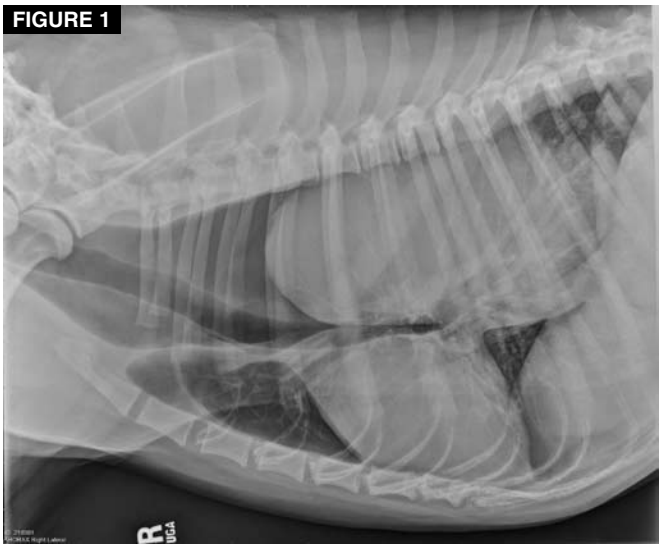


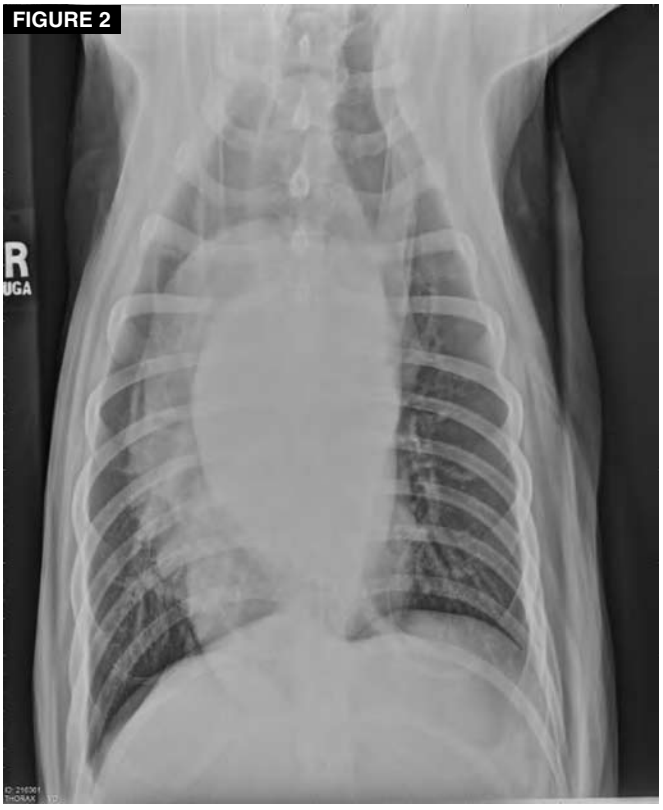
FIGURE 1

Lateral thoracic view.

Two days after surgery, thoracic radiography showed pleural fissure lines and a less severely dilated thoracic esophagus. A mild alveolar pattern suggestive of aspiration pneumonia was seen in the right middle lung lobe. The dog was fed via a gastrostomy tube for 4 days after surgery; on day 5, he was fed meatballs of canned food with his front end elevated and did not vomit or regurgitate.

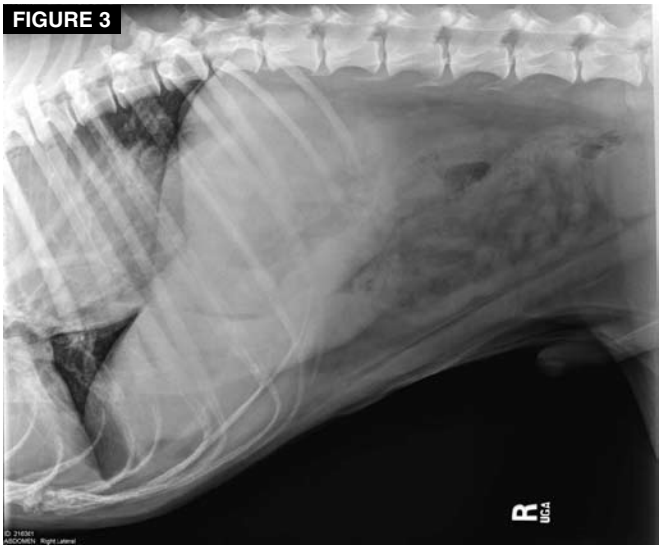
The dog was discharged 7 days after surgery. Enteral medications were administered via the gastrostomy tube. Medical management included tramadol (2.7 mg/kg [1.2 mg/lb] q12h), omeprazole (1 mg/kg [0.45 mg/lb] q24h), sucralfate (1 g slurry q8h), and metoclopramide (0.25 mg/kg [0.11 mg/lb] q8h before feeding). Broad-spectrum antibiotic therapy was continued with ciprofloxacin (6.75 mg/kg [3 mg/lb] q12h) and clindamycin (12 mg/kg [5.4 mg/lb] q8h).

FIGURE 2



Ventrodorsal thoracic view.

FIGURE 3



Lateral abdominal view.

Recommendations included elevated feedings and hand-feeding meatballs. The owner was instructed to feed the dog via the gastrostomy tube if the dog consistently regurgitated after oral feedings.

Outcome

After discharge, the patient removed the gastrostomy tube once. For 2 weeks after surgery, regurgitation and vomiting persisted despite elevated feedings and treatment with maropitant citrate and cisapride. Thoracic radiography and endoscopy of the upper gastrointestinal tract were conducted 15 days after initial presentation and surgery. Radiography confirmed persistent megaesophagus and resolution of aspiration pneumonia. Endoscopy revealed a distended esophageal lumen and an accumulation of saliva and undigested food proximal to the lower esophageal sphincter. Contraction of the lower esophageal sphincter appeared normal. The gastric mucosa also appeared normal, and the stomach was fixed in position because of the gastropexies. Several biopsies of the gastric mucosa were performed, and histopathologic analysis showed no abnormalities except mild to moderate fibrosis. Endoscopy revealed no overt cause of vomiting, and the only noted cause of regurgitation was megaesophagus.

The dog was readmitted to the hospital 2 weeks later due to significant weight loss and an inability to tolerate food or water by mouth. At this time, the dog weighed 29.5 kg (65 lb), which was 7.5 kg (16.5 lb) less than at initial presentation. To rule out other potential causes of megaesophagus, the dog was tested for hypothyroidism, hypoadrenocorticism, and myasthenia gravis. The results of these tests were all within normal limits. A new gastrostomy tube was placed. Fluids and food were administered solely via the tube, and minimal regurgitation of saliva was noted. The patient was discharged but returned 1 week later after pulling out the gastrostomy tube. The owners elected euthanasia, as it was apparent that return to normal esophageal function was unlikely and the dog would not tolerate a gastrostomy tube.

Discussion

GEI is rare, has an unknown etiology, and most often occurs in dogs younger than 1 year; the prevalence is increased in dogs younger than 3 months. Male German shepherds are at increased risk.[1–6] GEI has a guarded to poor prognosis, with 95% of dogs dying or being euthanized before surgery can be performed.[1,7] This case was unusual because of the dog's age, the duration of clinical signs before surgery, and the dog's survival well beyond initial treatment.

GEI occurs when the gastric cardia and body invaginate into the distal esophagus. Other abdominal organs, such as the spleen, liver, and intestines, can also be involved.[2,4] Young animals with megaesophagus may be predisposed to GEI. Invagination of the stomach and other organs into the esophagus compresses the pulmonary parenchyma and vasculature, leading to respiratory distress and shock due to decreased venous return. Respiratory distress may also result from aspiration pneumonia secondary to megaesophagus. Other complications include vascular compromise and necrosis of the gastric body and other involved organs. In these cases, death is usually attributed to either hypovolemic

shock caused by obstruction of the caudal vena cava or endotoxic shock due to gastric ischemia and necrosis.[3,5]

Animals with GEI typically present with acute onset of clinical signs (e.g., vomiting, regurgitation, lethargy, depression, shock) lasting 1 or 2 days.[2] In approximately 50% of cases, the patient has a history of signs consistent with esophageal disease, such as regurgitation or vomiting.[1–3] In the case presented here, there was no history of signs related to esophageal disease before the week of presentation.

GEI is most often diagnosed using thoracic radiography.[1–4] In this case and in previously documented cases, a soft tissue mass associated with the esophagus was seen in the caudal mediastinum. The heart and trachea were displaced ventrally, and the esophagus cranial to the soft tissue mass was large and filled with gas.[1,5] Aspiration pneumonia is frequently seen on radiographs.

Emergency surgery is required in cases of GEI. Before anesthesia, shock should be treated with fluids and broad-spectrum antibiotics.[2] Surgery involves reducing the stomach to its normal position and performing right- and left-sided gastropexies to prevent recurrence.[1,2] Gastric viability should be assessed, and resection of devitalized tissue is recommended. Immediately after reduction, the stomach may be congested; thus, its viability should not be assessed until sufficient time for reperfusion has been allowed.[2] Persistent megaesophagus is a common complication after surgical correction of GEI; therefore, it is beneficial to place a gastrotomy tube for postoperative feeding.[4,5] Withholding oral medications and food also encourages resolution of esophagitis, which is typically present.[2,3] Postoperative care should include intravenous fluid therapy to maintain hydration, broad-spectrum antibiotic therapy to treat aspiration pneumonia and prevent endotoxemia associated with gastric necrosis, and administration of proton pump inhibitors to treat and prevent esophagitis. Metoclopramide therapy can be important in preventing gastroesophageal reflux because it promotes gastric emptying and increases esophageal tone.[8] Megaesophagus is likely to persist in dogs that survive treatment of GEI, so occasional regurgitation is likely throughout an affected dog's life.[5]

It is unclear in this case, as in most cases of GEI, why intussusception occurred. It has been suggested that megaesophagus is heritable in German shepherds, predisposing them to the condition.[5] The heritability of megaesophagus and resulting GEI fits with the young age at presentation of most affected animals. In this case, the dog was 4 years of age, so congenital megaesophagus was unlikely.

Conclusion

GEI is a rare, often fatal disease of young dogs. The diagnosis and imaging results in this case were consistent with those of other documented cases. This case differs from other cases in that the patient was older and survived initial treatment of the disease. This demonstrates that GEI should be a diagnostic differential in dogs older than 1 year and in young dogs presenting in shock or in respiratory distress. Clinicians should remember that affected patients have a chance of survival despite the guarded to poor prognosis.

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