Gastric dilatation-volvulus after splenic torsion in two dogs

Darryl L. Millis, MS, DVM; Jean Nemzek, DVM, MS; Craig Riggs, DVM; Richard Walshaw, BVMS

- Anatomic changes that develop with splenic torsion, including stretching of gastric ligaments, may predispose dogs to the development of gastric dilatation-volvulus because of increased mobility of the stomach.
- Prophylactic gastropexy at the time of splenectomy may reduce the chance of future gastric dilatation-volvulus in dogs.

A 9-year-old male Great Dane (dog 1), weighing 52 kg and having a history of acute collapse, was initially evaluated by the emergency service. Physical examination findings included 5% dehydration, weakness, inability to stand, and mild abdominal distention. Examination of hemogram revealed mild nonregenerative anemia (PCV, 34%), leukocytosis (31,270 WBC/μl, with moderately toxic neutrophils), and thrombocytopenia (148,000 cells/μl). Urinalysis revealed specific gravity of 1.037, with 3+ proteinuria, a large amount of bilirubin, and 1+ urobilinogen. Serum alkaline phosphatase and alanine aminotransferase activities were high (920 and 378 IU/L, respectively).

Abdominal radiography revealed generalized loss of abdominal detail, consistent with free abdominal fluid; cranial displacement of the stomach; and a mass with mottled radiolucency in the cranial left quadrant, which was suspected to be the spleen. Splenic neoplasia, torsion, or abscess and generalized splenomegaly were considered in the differential diagnoses. Thoracic radiographic findings were within normal limits.

Lactated Ringer's solution was administered IV (5 ml/kg of body weight/h) and urine output was monitored, using a closed collection system. Twenty-four hours after admission, PCV was 22% and the dog developed melena. Disseminated intravascular coagulation was diagnosed, on the basis of prolonged activated partial thromboplastin time, high concentration of fibrin/fibrinogen degradation products, thrombocytopenia, and low antithrombin III activity. Fluid obtained by abdominocentesis had a PCV of 4.5% and nucleated cell count of 43,600/μl (99% neutrophils and 1% mononuclear cells). Cytologic evaluation was consistent with a septic exudate. *Staphylococcus intermedius*, sensitive to cephalothin, was isolated on microbial culture of peritoneal fluid samples. Heparin (75 U/kg, SC, q 6 h), sucralfate (1 g, PO, q 6 h), cimetidine (5.5 mg/kg, IV, q 6 h), and cephalothin (22 mg/kg, IV, q 6 h) were administered.

Dog 1 was anesthetized for abdominal exploration 36 hours after admission. At that time, the dog was hemodynamically stable. Surgery revealed mild peritoneal inflammation and a 360° splenic torsion, with complete thrombosis of the splenic veins and arteries. Complete splenectomy was performed. The dog recovered without complications and was discharged from the hospital 6 days after surgery. Histologic evaluation of the spleen did not reveal evidence of neoplasia, and was consistent with splenic torsion.

Dog 1 was readmitted 55 days after discharge, with signs of shock, depression, abdominal distention, and retching. Gastric dilatation-volvulus (GDV) was diagnosed on the basis of clinical signs. Treatment, consisting of gastric decompression with an orogastric tube and IV administration of lactated Ringer's solution (90 ml/kg in the first hour, then 5 ml/kg/h), dexamethasone sodium phosphate (8 mg/kg), and cephalothin (22 mg/kg), was initiated. Abdominal radiographic findings were consistent with the clinical diagnosis of GDV.

Dog 1 was anesthetized for exploratory surgery after a 2-hour stabilization period. At surgery, the stomach was distended and had a 180° clockwise volvulus. Several omental adhesions associated with the previous splenectomy also were noticed. Following decompression, the stomach was returned to its normal position and circumcostal gastropexy was performed. Recovery from surgery was uncomplicated and the dog was discharged from the hospital 4 days after admission.

Dog 2, a 6-year-old male German Shepherd Dog, was initially evaluated because of a history of acute collapse and lethargy. This dog had vomited 3 to 4 times weekly for the past 2 years. Three months prior to this admission, gastroscopic examination and evaluation of biopsy specimens from the stomach and duodenum were within normal limits.

Physical examination findings included pale mucous membranes, abdominal distention, weak femoral pulses, lethargy, and splenomegaly. Dog 2 had mild nonregenerative anemia (PCV, 28%), leukocytosis (24,790 WBC/μl), and high serum alkaline phosphatase activity (186 IU/L). Splenomegaly, loss of abdominal detail indicating free abdominal fluid, and cranial displacement of the stomach were evident on abdominal radiography, and splenic torsion was suspected.

Lactated Ringer's solution (90 ml/kg in the first hour, then 5 ml/kg/h), dexamethasone sodium phosphate (8 mg/kg), and cephalothin (22 mg/kg) were administered, and dog 2 was anesthetized for exploratory surgery 4 hours after admission. A 450° splenic torsion, with complete thrombosis of splenic veins and arteries, was found during surgery. Other abnormalities were not apparent, and complete splenectomy was performed. The dog recovered without complications and was discharged from the hospital 3 days later.

Dog 2 was readmitted because of retching 17

From the Department of Small Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824-1314. Dr. Millis' present address is Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, PO Box 1071, Knoxville, TN 37901-1071. Dr. Rigg's present address is Oakland Veterinary Referral Services, 1843 Orchard Lake Rd, Sylvan Lake, MI 48320.

months after splenectomy. Treatment by the referring veterinarian had included gastric decompression with an orogastric tube and IV administration of lactated Ringer's solution. Physical examination findings at our hospital included lethargy, mild abdominal distention, and 5% dehydration. Clinical and radiographic findings were consistent with a diagnosis of GDV.

Treatment was similar to that in dog 1. At surgery, the stomach was dilated and had a 90° clockwise volvulus. Other abnormalities were not noticed on abdominal exploration and a circumcostal gastropexy was performed. Dog 2 recovered from surgery and was discharged from the hospital 5 days after admission.

Splenic torsion and splenectomy preceded the development of GDV in these dogs by 2 and 17 months. Little is known about the relationship between GDV and splenic torsion. Splenic torsion often accompanies GDV, but either condition may be observed alone.[1-4] In 1 theory concerning the development of splenic torsion, the spleen is suggested to become displaced after gastric distention develops.[2,5] After gastric dilatation abates, the spleen is left in an abnormal position and the dog may develop acute splenic torsion, vascular thrombosis, and splenic infarction. Dog 2 had a history of chronic vomiting, which may have been associated with intermittent gastric dilatation; the intermittent gastric dilatation may have contributed to the development of splenic torsion.

Alternatively, splenic displacement and torsion may contribute to the development of GDV.[6] Splenic torsion increases tension on the gastrosplenic ligament,[6,7] and may increase tension on the hepatoduodenal and hepatogastric ligaments. The increased tension stretches these ligaments, and the resultant laxity may increase the mobility of the stomach and predispose the dog to GDV.[8]

Splenectomy has been described as an adjunct procedure in the management of GDV in dogs, although it alone does not prevent recurrence of GDV,[2,7,9] as was evident in the dogs of this report. In fact, splenic torsion and splenectomy may have contributed to the later development of GDV because of stretching of the hepatoduodenal and hepatogastric ligaments and creation of an anatomic void in the cranioventral part of the abdomen, all of which may allow increased mobility of the stomach.[10]

The cases reported here do not imply a cause-and-effect relationship; the development of GDV following splenectomy for the treatment of splenic torsion may have been coincidental. Both conditions are observed in dogs with similar signalments (ie, large and giant deep-chested breeds). The cases reported here represent 2 of the 6 surgically treated cases of isolated splenic torsion evaluated at our hospital during a 10-year period. Of the other 4, 1 dog had a prophylactic gastropexy performed at the time of splenectomy, 1 dog has not had evidence of GDV, 1 dog was euthanized several years after splenectomy for "a twisted gastrointestinal tract," and 1 dog was lost to follow-up.

Because of the possible relationship of splenic torsion and intermittent gastric dilatation, and the possibility of increased stomach mobility from stretching of gastric ligaments associated with splenic torsion, we believe prophylactic gastropexy should be considered in any large- or giant-breed dog undergoing splenectomy for the treatment of splenic torsion. Although the efficacy of prophylactic gastropexy in preventing GDV has not been documented, gastropexy in dogs with GDV is an effective method of preventing recurrence of the condition.[11,12] In most cases of splenic torsion, a gastropexy may be performed without undue risk if the dog is hemodynamically stable. In cases of acute, life-threatening splenic torsion, however, the additional time needed to perform a prophylactic gastropexy in a hemodynamically unstable dog entails unnecessary risk and is not advisable.

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