Primary Splenic Torsion in a Boston Terrier

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ABSTRACT. A 7-year-old female Boston terrier was referred to Hokkaido University Veterinary Teaching Hospital with a history of hemoglobinuria and anemia for several days. Abdominal radiographs showed splenomegaly, and ultrasonography revealed a hypoechoic splenic parenchyma with interspersed linear echoes consistent with the ultrasonographic appearance of splenic torsion. Ultrasonography and computed tomography (CT) indicated a C-shaped spleen. Exploratory laparotomy confirmed the diagnosis of splenic torsion. A splenectomy was performed, and the dog recovered well without complications. This is the first report of splenic torsion in Boston terriers, and the usefulness of ultrasonographic and CT findings of the splenic torsion was also confirmed.

KEY WORDS: Boston terrier, computed tomography, splenic torsion, ultrasonography.

Primary splenic torsion is a relatively uncommon occurrence, and it is usually considered to be secondary to the gastric dilatation and volvulus (GDV) complex [1]. Primary splenic torsion without concurrent gastric disease is highly unusual, representing 0.5% of all splenic disease in 1,480 dogs and 3.4% of all splenic conditions requiring surgery in 87 dogs [2, 10], and it is most common in large- or giant-breed, deep-chested dogs, especially Great Danes and German Shepherds [6, 8]. The etiology of primary splenic torsion is poorly understood, and it may have either an acute or a chronic clinical course. Acute splenic torsion may cause signs of cardiovascular collapse and shock. The chronic form of splenic torsion is difficult to diagnose because its clinical signs, physical examination findings, clinical pathology results and radiographic abnormalities are nonspecific [3, 11]. The preoperative diagnosis of splenic torsion is based mainly on ultrasonography [8], and computed tomography (CT) is also considered to be a useful diagnostic method [7]. We report a case of isolated splenic torsion in a non-large- or giant-breed dog diagnosed with ultrasonographic and CT findings.

A seven-year-old, intact female Boston terrier weighing 7.6 kg was referred to Hokkaido University Veterinary Teaching Hospital for evaluation of hemoglobinuria and moderate anemia. Symptomatic therapy including cephalixin and ofloxacin had been administered by the referring veterinarian.

At presentation, the dog was mildly lethargic, and an abdominal mass was palpable on physical examination. The mucous membranes exhibited slight pallor with capillary refill time within 1 second. The complete blood count (CBC) revealed moderate, normocytic, normochromic, regenerative anemia (packed cell volume [PCV] 22.7%, reticulocytes 335,160/µl) and leucocytosis (white blood cells [WBC], 21,200/µl) attributable to neutrophilia (17,278/µl) without a left shift. Platelet count was within the normal range (567,000/µl). Both slide agglutination test and direct Coombs test results were negative. There was no evidence of spherocytosis in the blood smear. Serum biochemistry abnormalities included slight elevations of aspartate aminotransferase (111 IU/l), alkaline phosphatase (268 IU/l) and creatine kinase (243 IU/l), and mild hyperbilirubinemia (1 mg/dl). The C-reactive protein concentration was moderately elevated (6.2 mg/dl). Prothrombin time (8.6 sec) and activated partial thromboplastin time (16.4 sec) were within the normal range. Urinalysis revealed severe hemoglobinuria with hemoglobin cast formation. Thoracic radiographs were unremarkable, and abdominal radiographs revealed marked splenomegaly.

Ultrasonography revealed a markedly enlarged, diffusely hypoechoic spleen with prominent intraparenchymal veins (lacy parenchyma) (Fig. 1A). The cranial extremity and the caudal extremity of the enlarged spleen could be observed in the same image, creating C-shaped spleen [4], an unusual ultrasonographic finding in dogs (Fig. 1B). The adjacent mesentery was markedly hyperechoic, and small amount of peritoneal effusion was observed. Color-flow Doppler failed to detect any blood flow into the spleen vein. CT examination was performed with general anesthesia. There was enlargement of the spleen and a small amount of fluid in the left ventrolateral aspect of the peritoneal cavity. The descending and ascending parts of the enlarged spleen came close together (Fig. 2). Contrast CT imaging was not performed because we could not detect any blood flow into the spleen and in the splenic parenchyma by ultrasonography. Spleen fine-needle aspirates were unremarkable.

Exploratory laparotomy and total splenectomy were performed without untwisting the splenic pedicle on the day of presentation, and intraoperative whole blood transfusion was performed. The enlarged spleen was curled around...
with its hilus to the inside) and the cranial extremity and caudal extremity of the spleen adhered to each other (Fig. 3). The degree of torsion in the spleen was unclear, but it appeared to be twisted over 180°. The surface of the spleen was dark red, and the cut surface was black. The rest of the abdominal organs appeared normal. Histopathology of the spleen revealed severe hemorrhage, but there was no evidence of malignancy. The dog recovered well from surgery and was discharged without complications 3 days later.

Primary or isolated splenic torsion is an uncommon splenic disease, generally occurring in large- or giant-breed, deep-chested dogs [6, 8]. To the author’s knowledge, there has been no report of splenic torsion in small- or medium-size dogs, including Boston terriers.

The etiology of primary splenic torsion is unknown. It has been hypothesized that it occurs in conjunction with spontaneously resolving gastric dilatation/volvulus (GDV) or partial gastric torsion, which leaves the spleen in a rotated position when the GDV resolves [4]. Another theory is that stretching of the suspensory gastrosplenic, splenocolic, and phrenicosplenic ligaments from GDV can cause a loosening of the splenic attachments and may predispose the spleen to torsion. These 2 hypotheses are supported by reports of prior GDV episodes in the histories of some dogs with isolated splenic torsion [6]. However, Boston terriers are not predisposed to GDV, and the present case did not have a previous history of GDV. Therefore, the cause of the splenic torsion in this case is unclear.

Clinical signs of chronic splenic torsion are non-specific and vague, and most dogs with splenic torsion are presented for some combination of vomiting, weakness or depression, icterus, hematuria or hemoglobinuria, abdominal pain, and/or diarrhea of several days’ duration. Anemia, leukocytosis, and hemoglobinemia are usually found in cases of chronic splenic torsion. The dog presented here exhibited lethargy;
and an abdominal mass, and had laboratory findings including regenerative anemia, hyperbilirubinemia, and hemoglobinuria. These signs are also observed in patients with immune-mediated hemolytic anemia (IMHA), a common cause of hemolytic anemia in dogs. Thus, it is thought to be difficult to distinguish the splenic torsion from IMHA based on clinical signs and laboratory findings, especially in small- or medium size dogs because of the extremely rare occurrence of splenic torsion in these dog breeds. However, the negative results of slide agglutination test and direct Coombs test, and the absence of spherocytosis may be important findings to distinguish splenic torsion from IMHA, because positive results of these tests are common in IMHA.

Ultrasonography is an accurate, widely available, and noninvasive technique for the diagnosis of primary splenic torsion in dogs. Ultrasonographic abnormalities, including splenomegaly, a diffusely hypoechoic splenic parenchyma with linear echoes (lacy parenchyma) and splenic vein enlargement at the splenic hilus have been reported in dogs with splenic torsion [5, 8]. However, these ultrasonographic findings are also seen with splenic necrosis secondary to infarction without splenic torsion [9]. Therefore, in addition to B-mode evaluation of the splenic parenchyma, spectral or color Doppler evaluation for absent blood flow is necessary for accurate diagnosis of splenic torsion. Additionally, in the case presented here, another ultrasonographic sign was observed: the cranial extremity and the caudal extremity of the enlarged spleen could be observed in the same image because the spleen was C-shaped due to greater than 180º torsion of the splenic pedicle. CT findings also indicated the C-shaped spleen, and this finding was highly suggestive of splenic torsion. Thus, ultrasonographic and CT findings indicating the C-shaped spleen appeared to be useful for the diagnosis of splenic torsion.

The recommended treatment for splenic torsion is splenectomy after stabilization of the patient, although there is one case report of spontaneously resolved acute splenic torsion in a St. Bernard [12]. The splenic pedicle should not be untwisted in most cases, especially if the clinical signs are chronic, because this can cause release of toxins, microemboli, or bacteria into the bloodstream [4]. The prognosis for surgical treatment of splenic torsion is generally good if the diagnosis is made early in the disease course [4, 6]. Complications of untreated splenic torsion include cardiovascular collapse, ventricular arrhythmias, hemorrhage, coagulation disorders, disseminated intravascular coagulation, thrombosis, pancreatitis, GDV, and hemoglobinemia-associated nephrosis [13]. Early total splenectomy and supportive care reduce the risk of these complications. In the present case, splenectomy was performed on the day of presentation, and the dog recovered from surgery without any complications.

In summary, splenic torsion should be considered in the differential diagnosis for splenomegaly even in small breed dogs, and ultrasonographic and CT findings indicating a C-shaped spleen may be useful for the diagnosis of splenic torsion.

REFERENCES