**The Spleen Be Trippin**

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Ryan K Poling

Advisor: Allison Kenzig, DVM

## Introduction

A splenic torsion is a condition which is commonly associated with a gastric dilation and volvulus (GDV) or as a rare primary condition. [23] Primary splenic torsion seldom occurs and its pathophysiology is poorly understood; however, it is more commonly seen in deep-chested, large breed dogs, much like those affected by GDV. [7, 23] There are multiple reports published describing the same disease occurring in smaller dogs, such as the Boston Terrier [11], an Alpaca [19], and in human beings. [1,5,22] A primary splenic torsion may be difficult to diagnosis due to the vague clinical signs and its presentation as an acute or chronic form. Advanced diagnostics, such as ultrasound and computed tomography (CT), are very helpful in making a diagnosis.

Acute primary splenic torsions can present as a patient experiencing abdominal pain and/or cardiovascular collapse from the sequestration of blood within the spleen. [23] Retching, drooling, tachycardia, hypotension, poor peripheral perfusion, and weakness may also be evident on presentation. [24] The presentation is very similar to a patient experiencing a GDV. The prognosis for acute primary splenic torsion is guarded-to-poor due to conjunctive problems associated with cardiovascular shock and toxemia. [23]

Chronic primary splenic torsions can present with a variety of vague signs such as, lethargy, depression, vomiting, anorexia, and weakness. [23] Elevated serum alkaline phosphatase (ALP), elevated serum alanine aminotransferase (ALT), anemia, leukocytosis, thrombocytopenia, and hemoglobinemia may be found several days after a chronic splenic torsion has occurred. [23, 24] Pancreatic enzymes may be elevated due to the compromised communal blood supply from the splenic artery. Clinical signs may often imitate immune mediated hemolytic anemia (IMHA) or immune-mediated thrombocytopenia (ITP). [14] The

prognosis for dogs with chronic splenic torsion is good because these cases have a lower incidence of toxemia and cardiovascular shock. [23, 24]

This paper focuses on primary splenic torsion, describing its typical clinical features, pathophysiology, diagnosis, treatment, and prognosis. An emphasis will be made on the canine patient with primary splenic torsion not associated with gastric dilatation and volvulus.

**History and Presentation**

A 7-year-old intact, male German Shepherd presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) on February 24, 2016 with a 4-day history of vomiting and lethargy. On February 22, 2016, he was seen by his primary veterinarian who performed bloodwork and abdominal radiographs, which did not reveal any significant abnormalities. He was discharged and sent home with metronidazole, metoclopramide, and flunixin meglumine. He continued to experience clinical signs despite medical management and re-presented to the primary veterinarian the following day. During the second presentation, the primary veterinarian diagnosed the patient with a mild anemia of 27% (35%-55%). He was given oral barium and abdominal radiographs revealed a mass effect. He was referred to MSU-CVM for further diagnostics.

Upon presentation to Mississippi State University College of Veterinary Medicine's Internal Medicine service, he was quiet, alert, and responsive. He weighed 46.7 kilograms (102.7 pounds). His vitals parameters were within normal limits (Temperature: 101.8F, Pulse: 100 beats per minute, Respiration: 24 breaths per minute). His mucous membranes were pink and tacky with a capillary refill time of 2 seconds. He was estimated to be approximately 5-7% dehydrated. His body condition was ideal with a score of 5/9. His was guarding his abdomen,

and pain was elicited upon abdominal palpation. His spleen palpated as subjectively enlarged. A soft, freely moveable, well-circumscribed mass over the dorsal aspect of the left hock was also noted.

**Diagnostic Approach/Considerations**

On presentation, an abdominal focused assessment with sonography for trauma (aFAST) scan revealed free fluid in 3 out of 4 quadrants. A suspected large, cavitary mass was noted, which seemed to be originated from the tail of the spleen. His packed cell volume (PCV) was 37% (35-55), total protein was 7.8 g/dl (5.5-7.5), and lactate was 1.5 mmol/L (0.5–2.5). A complete blood count, serum chemistry and coagulation profile were performed and revealed a stress leukogram, but these tests were otherwise unremarkable. Thoracic radiographs revealed mild mineralization of the walls of the larger bronchi, which was contributed to age-related changes, and spondylosis deformans of multiple thoracic vertebrae. Abdominal radiographs revealed decreased serosal margin detail due to a large, ill-defined, ovoid, soft tissue opacity mass causing centrifugal displacement of the gastrointestinal tract to the abdominal periphery. The mass was causing border effacement of the liver margin, and the left kidney and spleen were not seen. A normal splenic head was not found caudal to the left margin of the stomach. Differential diagnoses of described mass included splenic torsion (as the head of the spleen was not seen), neoplasia, or hematoma. Upon abdominal ultrasound, the spleen was severely enlarged and extended along the entire length of the left abdomen from the liver to the urinary bladder. There was a perivenous hyperechoic triangle [9, 19] at the splenic hilus and the splenic veins revealed no flow on Doppler interrogation. The primary differential for the ultrasonographical changes described in the spleen was splenic torsion, with a splenic infarct considered less likely.

A ventral midline celiotomy was performed under general anesthesia. The spleen was grossly enlarged, filling about 75% of the body cavity. The edges were rounded, and it was dark red-to-purple in color. The spleen was removed from the abdomen with a LigaSure which was used to cauterize the torsed pedicle of the spleen. It was determined to have an approximately 180 to 270-degree torsion at the base of the spleen.

A liver biopsy was taken from the quadrate lobe using a guillotine method. This biopsy was sectioned, and submitted for aerobic/anaerobic culture and sensitivity and histopathologic evaluation. The spleen was also submitted for histopathology. An incisional gastropexy was performed prophylactically prior to closing the abdominal cavity to prevent a future GDV. The liver culture did not reveal any growth within 48 hours. The splenic histopathology revealed tissue that was heavily filled with blood and did not contain visible neoplastic change or erythrophagocytosis. Apoptotic splenic lymphoid cells were appreciated which is often noted in cases of severe stress. The liver histopathology did not reveal any significant abnormalities. A primary splenic torsion was suspected.

**Pathophysiology**

The exact pathophysiology for a primary splenic torsion is poorly understood; however, large breed, deep-chested dogs appear to have an increased risk with males being affected more frequently than females. [7, 14] A report which evaluated 1,480 cases of splenic disease in dogs showed that primary splenic torsions occurred in approximately 0.5% of those cases. [16] The possible underlying etiology resulting in a splenic torsion may include congenital abnormalities of the splenocolic or gastrosplenic ligaments, concurrent rotation in association with a GDV, or traumatic disruption associated with a GDV. [16]

A primary splenic torsion occurs when the tail of the spleen is able to rotate around the pedicle in the absence of other inciting causes, such as a GDV. The pedicle is weakly attached by the gastrosplenic and splenocolic ligaments making the normal spleen relatively mobile. [20] Torsions initially result in compression of the splenic veins, causing vascular congestion and thrombosis of those vessels. As the spleen continues to rotate, the splenic arteries become occluded, resulting in formation of thromboemboli and infarction. [14] Congenital abnormalities, such as hypoplasia and aplasia of the ligaments, may predispose the spleen to torsion; however, the exact pathogenesis is not well-documented.

Approximately 20% of secondary splenic torsions occur concurrently with a gastric dilatation and volvulus. [8] The spleen may rotate in conjunction with the gastric dilatation and volvulus allowing for hypermotility and/or congestion. [8] Another theory proposes that if the animal experiences multiple partial gastric torsions the splenocolic and gastrosplenic ligaments will loosen and allow the spleen to become hypermotile. [14] The hypermotility allows the spleen to move more freely on its axis and predisposes the spleen to torsion. Adhesions may develop after the partial episodes, keeping the spleen in a state of constant torsion. [23]

**Treatment and Management**

Treatment of splenic torsion includes supportive care and splenectomy. [16] During surgery, the spleen must not be unrotated, as this may result in intravascular release of free radicals, thrombi, and other vasoactive compounds. [17] Ventricular arrhythmias are a relatively common finding in dogs that have undergone a splenectomy; therefore, electrocardiogram (ECG) monitoring is strongly recommended. [3] The exact pathogenesis of ventricular arrhythmias associated with a splenectomy is poorly understood. [14] In a retrospective study where 60 dogs presenting for hemoabdomen were evaluated, approximately 36.8% of dogs with splenic torsion

had ventricular arrhythmias intraoperatively or postoperatively. [3] A case report from 2005 discussed a successful correction of splenic torsion by derotation in a racing greyhound; however, this case presented to the hospital immediately, splenic infarction was not severe, and the torsion was less than 90º.[12]

During surgery, it is strongly recommended to perform a prophylactic gastropexy following splenectomy to prevent any future gastric dilatation and volvulus from occurring. [17]

**Case Outcome**

Surgery was successful, and the patient recovered in the intensive care unit where he was monitored for abnormalities with continuous ECG and blood pressure. No ECG or blood pressure abnormalities occurred in this case. He continued to improve and was sent home two days postoperatively with medications to control pain and instructions for exercise restriction. Based on the nature of his clinical signs and acute onset, he was diagnosed with an acute splenic torsion.

Despite occurring less than 0.5% of the time, primary splenic torsions should always be considered as a differential diagnosis in large breed, deep-chested dogs when faced with a mass in the abdomen which may be associated with the spleen. With surgical correction, a 79% survival rate has been reported for splenic torsions, both primary and those associated with GDV.[8]

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