

Diagnosis and Management of Splenic Torsion in a Cane Corso Dog

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ABSTRACT

Splenic torsion is a life-threatening acute abdomen disorder in dogs. A four-year-old female Cane Corso dog was presented to the Madras Veterinary College Teaching Hospital with a history of abdominal pain, vomiting, inappetence and lethargy in the last three days. Physical examination revealed splenomegaly, and abdominal ultrasonography showed marked splenic enlargement with a lacy parenchymal pattern, suggestive of splenic torsion. Molecular technique revealed the presence of the Babesia species. The animal was stabilised with emergency medical management with blood transfusion and treated for babesiosis. Subsequently, an exploratory laparotomy was performed for splenectomy. The animal recovered eventually.

Keywords: Cane Corso, Splenic torsion, Ultrasonography, Splenectomy

INTRODUCTION

Primary splenic torsion is a rare clinical disorder in dogs and occurs as a secondary complication in combination with gastric dilatation and volvulus. It is an acute life-threatening condition, as splenic torsion may lead to splenic hypertension, infarction and bleeding. Early surgical intervention is the need of the hour to save the life of the patient (Sodhio *et al.*, 2003). Splenic torsion may be acute or chronic, and abdominal ultrasonography is the suggested method to diagnose splenic torsion in dogs (Saunders *et al.*, 1998). Splenomegaly, variable and decreased echogenicity of the parenchyma of the spleen and hypoechoic appearance or

“lace” are suggestive sonographic findings of splenic torsion, although the definitive diagnosis is through exploratory laparotomy (Konde *et al.*, 1989). This paper reports a case of splenic torsion, mainly focusing on the ultrasonographic diagnosis, emphasising the importance of the ultrasonographic examination.

CASE HISTORY AND OBSERVATION

A four-year-old female Cane Corso dog weighing 40 kg was presented with the history of lethargy, vomiting and inappetence for the past 3 days and treated by a nearby veterinarian with fluid therapy, antibiotics and antiemetics. Clinical examination revealed hyperthermia, tachycardia, pale mucous membrane, capillary refill time of 3 seconds. Abdominal palpation revealed splenomegaly. A blood sample was collected for haematology and serum biochemistry (Table I). These investigations revealed anaemia and leukocytosis, and there was no detectable change in serum biochemistry. The blood sample was further screened for any hemoprotozoan diseases by the polymerase chain reaction technique, which detected *Babesia* sp. Arterial Blood Gas analysis (Table II) indicated marked respiratory alkalosis due to pain and hyperlactemia, indicative of hyperventilation and hypoperfusion. Ultrasonography revealed a severe splenic enlargement with a lacy pattern of parenchyma, and colour flow Doppler studies revealed no blood flow at the splenic hilus, indicating splenic torsion (Figure 1).

TREATMENT AND DISCUSSION

The dog was initially stabilized intensively with fluid and oxygen therapy, opioid analgesic butorphanol @ 0.2mg/kg I/V for the management of respiratory alkalosis. Whole blood (350ml) was collected from a healthy dog and transfused and the patient was treated for babesiosis with Imidocarb at 6.6mg/kg s/c at weekly intervals for two

weeks, and azithromycin @10mg/kg PO and atovaquone at 13.3mg/kg PO for 10 days. Subsequently exploratory laparotomy was performed (Figure 2). Splenectomy was done as splenic torsion with necrotic splenic parenchyma was noticed. Post operative care included fluid therapy, amoxicillin, metronidazole and hematinics. The dog recovered after 15 days of post-operative care and was followed up for a period of 4 months.

Table I: Haematology Profile and Serum Biochemistry

Haematology Profile			Serum Biochemistry		
Parameters	Before Treatment	10 days After Treatment	Parameters	Before Treatment	10 days After Treatment
Hb (g/dl)	8.50	10.60	Glucose	66 mg/dl	128 mg/dl
PCV (%)	24.90	32.00	Total protein	8.2 g/dl	7.4 g/dl
RBC (m/cmm)	3.25	4.93	Albumin	3.2 g/dl	2.7 g/dl
WBC/cmm	75000	23,000	BUN	25 mg/dl	29 mg/dl
Platelet/cmm	7,25,000	5,94,000	Creatinine	0.5 mg/dl	0.7 mg/dl
			ALT	110 U/L	126 U/L
			ALP	214 U/L	134 U/L

Table II: Arterial Blood Gas Analysis

Parameters	Before treatment	After Treatment
pH	7.463	7.431
PCO2	28.7 mmHg	25.2 mmHg
PO2	82.7 mmHg	87.1 mmHg
CHCO3	20.5 mmol/L	24.1 mmol/L
Glucose	127 mg/dl	116 mg/dl
Urea	8.5 mmol/L	7.3 mmol/L
Lactate	4.19 mmol/L	1.32 mmol/L
Creatinine	0.9 mg/dl	0.7 mg/dl
Cl-	125 mmol/L	132 mmol/L
Na+	143 mmol/L	137 mmol/L
K+	4.2 mmol/L	4.8 mmol/L



Figure 1: Ultrasonography - Lacy pattern



Figure 2: Exploratory Laparotomy - Splenectomy

The major predisposing factor for splenic torsion is the breed predisposition, which is common in German Shepherd and Great Dane dogs. The male dogs (castrated and sexually intact) are more frequently affected by splenic torsion than female dogs (Vazquez *et al.*, 2004). Increased thoracic depth-to-width ratio, as in deep chested breed, causes primary splenic torsion due to the additional space in the abdominal cavity, which allows organs to rotate abnormally. Torsion commonly occurs when the spleen is motile because of congenital absence or weakness of one or more of the ligaments that hold the spleen in its normal position in the left cranial abdomen (De groot *et al.*, 2016). Mild to moderate torsion causes chronic abdominal pain associated with splenic congestion, whereas severe torsion leads to acute abdominal pain caused by infarction and adhesion with adjacent organs (Reinhart *et al.*, 2015). The clinical signs encountered include nausea, vomiting, fever, and palpable abdominal mass, which represent splenomegaly (Steinberg *et al.*, 2002). Abdominal radiography would show splenomegaly with loss of serosal detail, and gastrointestinal displacement might suggest the presence of splenic torsion. However, abdominal ultrasonography is still an accurate, widely used, and non-invasive technique for the diagnosis of primary splenic torsion in dogs. Ultrasonographic findings of splenic

torsion include splenomegaly, diffusely hypoechoic parenchyma with linear echoes, lacy parenchyma and engorged splenic vein at the splenic hilus. The recommended treatment for splenic torsion associated with infarction is splenectomy (Steinberg *et al.*, 2002), supportive therapy and post-operative monitoring with continuous electrocardiography to assess the frequency and extent of tachyarrhythmias (Marino *et al.*, 1994). The prognosis of splenic torsion is fairly good when the condition is diagnosed earlier, along with medical and surgical management, with a 79 per cent survival rate (Jaeger *et al.*, 2006).

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