



NOTE

Internal Medicine

A case of acute splenic vein thrombosis in a dog

Jaehwan KIM^{1)*}¹⁾Helix Animal Medical Center, 162, Sinbanpo-ro, Seocho-gu, Seoul, Republic of Korea

ABSTRACT. An 11-year-old, castrated male, Yorkshire Terrier was presented with acute vomiting after chicken bone ingestion. The dog had been diagnosed with hyperadrenocorticism previously and showed acute splenomegaly and signs of systemic inflammatory response syndrome during hospitalization. On diagnostic imaging, acute splenic vein thrombosis was found, concurrent with pancreatitis and gastritis. The spleen showed marked enlargement and hypoechoic lacy appearances on ultrasonography, mimicking splenic torsion. On the histopathologic report, only splenic hemorrhage and congestion with large splenic vein thrombosis were identified. After splenectomy, the dog completely recovered and was discharged.

KEY WORDS: dog, splenic vein, splenic vein thrombosis, systemic inflammatory response syndrome, thrombosis

J. Vet. Med. Sci.

81(10): 1492–1495, 2019

doi: 10.1292/jvms.19-0258

Received: 13 May 2019

Accepted: 30 July 2019

Advanced Epub:

12 August 2019

The splenic vein is a branch of the portal vein that is clinically significant because it is relatively easy to access during ultrasound examination and is affected in several diseases. Splenic vein thrombosis (SVT) is the most common disease that occurs in the splenic vein in both humans and dogs of a small breed. In humans, the most common cause of SVT is chronic pancreatitis, and other causes, such as neoplasia, pancreatic abscess, trauma, and inflammatory disorders, have also been reported [11]. Although the reported underlying causes of SVT in dogs are limited, they are similar to those in humans. For instance, neoplasia, corticosteroid injection, sepsis, diabetes mellitus, and hyperadrenocorticism have been reported in veterinary medicine [6]. Owing to the nature of the disease, SVT often progresses in a chronic rather than acute manner, and is most commonly discovered incidentally in daily veterinary practice [7, 10]. This case report describes the unusual presentation of splenic thrombosis with acute clinical symptoms.

An 11-year-old, neutered, male Yorkshire Terrier, weighing 4.3 kg, was presented with vomiting after eating chicken bones. The dog had been diagnosed with and treated for hyperadrenocorticism for the past 2 years and appeared healthy without any other clinical signs. On physical examination, only abdominal distension and panting (respiratory rate, 60/min) were observed. Complete blood counts and serum biochemistry profile revealed mild leukocytosis (19.5; reference range, 6–17 × 10⁹/l), as well as increased triglyceride (375; reference range, 21–110 mg/dl), amylase (1,822; reference range, 388–1,007 U/l), and lipase levels (3,226; reference range, 0–1,800 U/l). Elevated CRP (97.8; reference range, 0–20 mg/l) and cPL levels (923; reference range, 0–200 ng/ml) were also noted. The differential counts of the white blood cells were as follows: segmented neutrophils, 74%; lymphocytes, 11%; monocytes, 8%; eosinophils, 5%; and basophils, 2%.

Radiographs were taken in a routine manner (Titan 2000M; Comed Medical Systems, Seoul, Korea). On the lateral radiograph, hyperechoic linear bone opacities were seen in the pyloric region, which was consistent with the chicken bone ingested. Diffuse hepatomegaly with a blunted liver margin and caudodorsal deviation of gastric axis were noted. There was no remarkable evidence of gastric perforation, or mechanical or functional ileus (Fig. 1A). Ultrasonographic examination was performed (Aplio 500, Toshiba Medical System, Tokyo, Japan) with linear-array (10–13 MHz) and curvilinear array (6–8 MHz) probes. Abdominal ultrasound examination revealed no remarkable findings of the gastrointestinal tract including the stomach and spleen. Only hypoechoic pancreatic parenchyma with peripancreatic fat edema and intraluminal hyperechoic foreign bodies with distal acoustic shadowing artifacts, which were consistent with the ingested chicken bones that were identified. The dog was hospitalized for endoscopy for chicken bone removal the next day because of uncontrolled vomiting.

The dog had sudden abdominal distension, pain, and lethargy after 8 hr of hospitalization. On the physical and hematologic exam, hyperthermia (39.7°C), tachycardia (140 beats/min), and leukocytosis (32; reference range, 6–17 × 10⁹/l) were noted. In addition, the band-shaped immature neutrophils were found on the blood smear, suggestive of systemic inflammatory response syndrome (SIRS). A re-test of the differential cell counts revealed the following: band neutrophils, 11%; segmented neutrophils, 69%; lymphocytes, 11%; monocytes, 5%; eosinophils, 3%; and basophils, 1%. Increased serum lactate levels (10.8; reference

*Correspondence to: Kim, J.: ultradiovet@gmail.com

©2019 The Japanese Society of Veterinary Science



This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial No Derivatives (by-nc-nd) License. (CC-BY-NC-ND 4.0: <https://creativecommons.org/licenses/by-nc-nd/4.0/>)

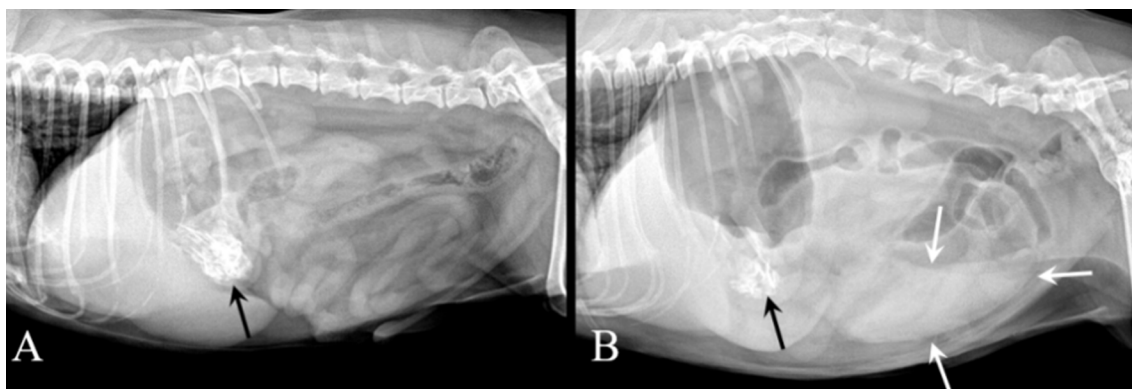


Fig. 1. Lateral radiographs of the dog at the time of admission (A), and 8 hr from the hospitalization (B). Hyperechoic bone opacities are seen in the pyloric region, and this finding is consistent with ingested chicken bones (black arrows). Note the markedly enlarged and elongated splenic tail with a round margin on B (white arrows).

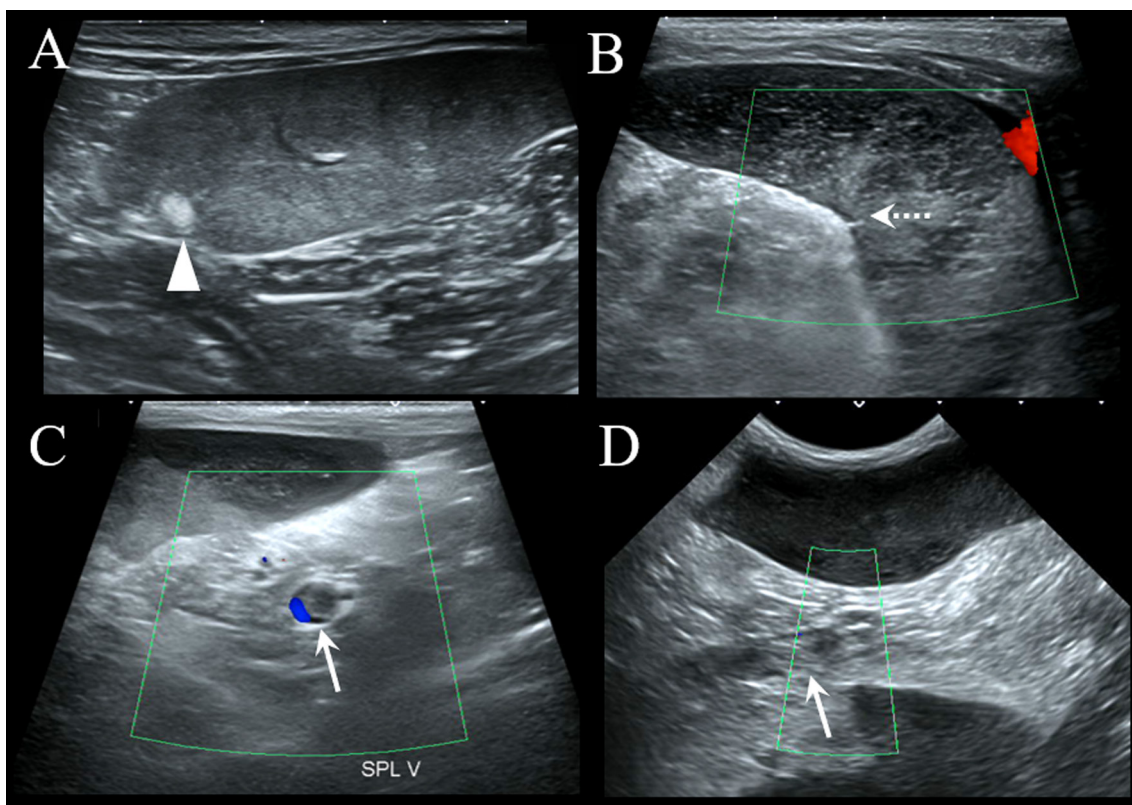


Fig. 2. Abdominal ultrasonography of the splenic parenchyma and vasculatures. (A) The spleen shows a hyperechoic nodule consistent with splenic myelolipoma (arrowhead), without parenchymal abnormalities. (B) The spleen shows marked enlargement and hypoechoic parenchyma with a lacy appearance, after 8 hr. Small amount of peritoneal effusion is found around the spleen. Note the lack of vascular response in the splenic hilum (dashed arrow) and the large thrombus (arrows) in the splenic vein (C and D). A and B, Images of the splenic parenchyma; C, Transverse image of the splenic vein; D, sagittal image of the splenic vein.

range, 0.5–2.5 mg/dl) and CRP levels (132.9; reference range, 0–20 mg/l) were noted. Coagulation tests were not performed.

Serial abdominal radiographs showed a markedly enlarged and elongated spleen with rounded margins (Fig. 1B). On the abdominal ultrasound, the spleen was markedly enlarged with a coarse echotexture and showed a diffuse hypoechoic, lacy parenchymal pattern (Fig. 2). Lack of blood flow at the splenic hilum on the color Doppler examination and an echogenic thrombus in the lumen of the splenic vein were noted. A small amount of peritoneal effusion was identified around the spleen.

Medical treatment and laparotomy were performed to relieve the acute splenic inflammation and infarction. Splenectomy was

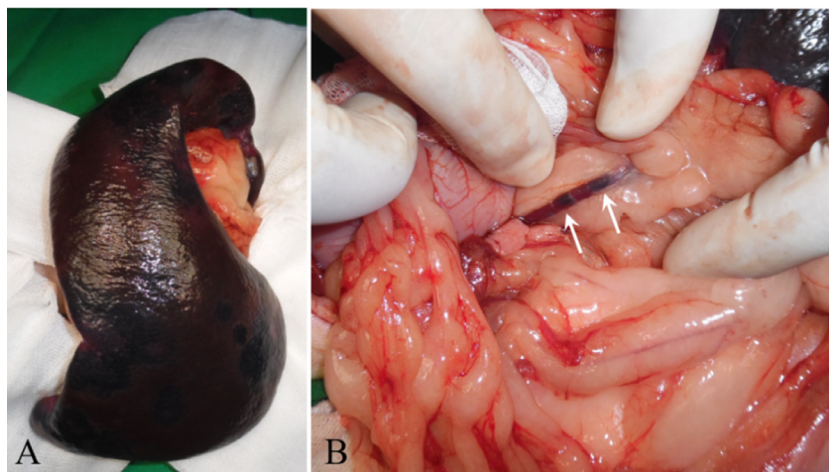


Fig. 3. Intraoperative images of the dog. (A) Markedly enlarged, hemorrhagic, and congested splenic parenchyma is seen. (B) A large thrombosis is distinctively seen, which completely obstructs the lumen of the splenic vein (arrows).

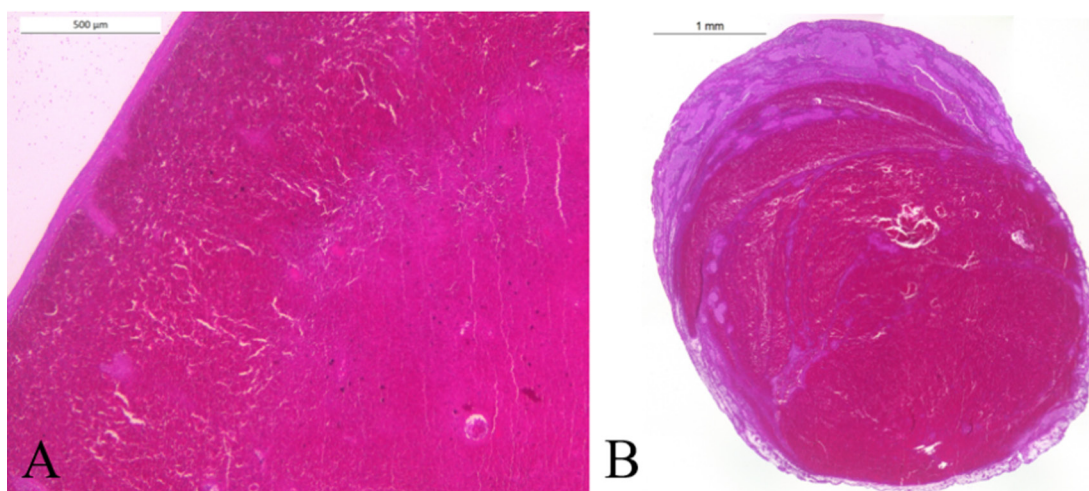


Fig. 4. Histopathological results of the spleen (A) and splenic vein (B). A, Severely hemorrhagic, congested splenic parenchyma without distinction of red and white pulps. B, On the transverse section of the splenic vein, a huge thrombus is filling the lumen. (A, hematoxylin and eosin [H&E] staining, 400× magnification; B, H&E staining, 100× magnification)

performed as a routine procedure. During the surgery, markedly enlarged, congested splenic parenchyma, and distinct thrombosis were found in the splenic vein (Fig. 3). Chicken bones identified in the stomach were removed through gastrotomy, and edematous mucosal ulceration by sharp ends of the bones were identified. Severely hemorrhagic, congested splenic parenchyma with a large thrombus filling the entire lumen of the splenic vein was identified on the histopathologic examination (Fig. 4). No evidence of neoplastic and inflammatory changes was found on the splenic parenchyma. No remarkable thrombosis of the portal branches such as the gastric and mesenteric veins was observed on surgical exploration. The dog recovered without incidence after the surgery. After a week, all parameters of blood investigation and imaging examination were normal.

Splenic vein thrombosis is frequently observed on routine abdominal ultrasonography in dogs. Most cases are discovered incidentally, and clinical symptoms are rare [7]. Therefore, the clinical implication of SVT is to investigate the underlying causes such as neoplasia and endocrine disorders. According to a study of SVT and concurrent diseases, neoplasia is the most common concurrent condition, with lymphoma being the most common neoplasm [6]. Exogenous corticosteroid administration, SIRS, disseminated intravascular coagulation, immune-mediated disorders, and pancreatitis have been also reported [1, 8, 12]. As most cases of SVT are caused by chronic underlying causes with slow progression, clinical symptoms are rare. In this case, hyperadrenocorticism was the underlying factor and acute pancreatitis and SIRS due to chicken bone ingestion were thought to cause acute SVT, even though the dog appeared otherwise healthy. Interestingly, most cases of SVT are discovered incidentally, while this case presented with severe acute clinical symptoms such as abdominal distention and lethargy, despite the absence of

portal vein thrombosis. Although further studies are needed, it can be concluded that acute SVT can have clinical manifestations.

The development of thrombus has been classically explained through Virchow's triad; endothelial injury, blood stasis, and the presence of a hypercoagulable state. Endothelial injury and blood stasis occur mainly in patients with heart disease and occur mainly by mitral or aortic valve degeneration. Thoracic radiography and echocardiography did not reveal cardiac abnormalities in this case. The hypercoagulable state follows a more complex mechanism and occurs in various conditions, such as malignancy, trauma, inflammatory, endocrine disorders, sepsis, and SIRS [2]. Hyperadrenocorticism is associated with an increased risk of thromboembolism [5] and in veterinary medicine, evidence of hypercoagulability has been identified in dogs with hyperadrenocorticism including several reports of pulmonary thromboembolism associated with increased serum glucocorticoid level [6]. SIRS is also known to be the primary cause of SVT in humans, and it was reported that evidence of SIRS was found in 25% of SVT dogs [6]. Cytokines of systemic inflammation activate the coagulation cascade and most likely contribute to the formation of venous thrombosis. In humans, pancreatic disease particularly chronic pancreatitis is associated with SVT due to its anatomic relationship [3]. Since vascular supply to the left limb of pancreas arises from splenic vasculatures, pancreatitis with perivascular inflammation may induce SVT frequently. As a result, in dogs with underlying diseases such as hyperadrenocorticism and pancreatitis, the possibility of thrombus formation in the splenic vein, and the induction of acute clinical symptoms should be considered.

On abdominal radiographs, the spleen showed peracute enlargement. In addition, hypoechoic splenic parenchyma with lacy, interspersed linear echoes due to congestion were seen on abdominal ultrasonography. The color Doppler flow of the splenic hilus was not identified and these imaging characteristics are typical findings of splenic torsion. If we had overlooked the fact that the dog had SIRS or failed to scan the splenic vein carefully to find the thrombus, it was likely that the dog would have been misdiagnosed with splenic torsion. Nevertheless, what distinguishes it from typical splenic torsion are firstly there was no 'C-shape', which is typical of splenic torsion in radiographs. Secondly, there was no distinct dilation of the hilus or typical parallel echogenic lines on abdominal ultrasound.

A lacy appearance in abdominal ultrasonography is typical of splenic infarction. Whatever the cause, splenic infarction may occur if an abnormality occurs in the blood supply to the spleen, which is known to occur in less than 2% of dogs [4, 9]. The lacy appearance with splenomegaly has been known to be a characteristic feature that could easily help diagnose splenic torsion on ultrasonography [7]. However, in the present case, acute SVT showed complete consistency with these findings as well as peritoneal effusion and regional peritonitis of omentum around the spleen on ultrasonography. Therefore, even if splenic torsion with multiple infarction is strongly suspected on ultrasonography, acute SVT should be considered as a differential diagnosis, and imaging of splenic vein is recommended.

Although SVT is a relatively common disease in dogs, it is generally regarded as a chronic condition, with subclinical signs and focus on the underlying disorders. However, in this case, acute SVT manifested with clinical symptoms. In addition, when a torsion or acute infarction of the spleen is suspected on the diagnostic imaging, splenic vein should be closely examined and underlying diseases, such as a pancreatitis, or thrombosis should be considered. In conclusion, acute SVT should be considered as a differential diagnosis in cases with acute clinical manifestations and splenomegaly.

REFERENCES

1. Carr, A. P., Panciera, D. L. and Kidd, L. 2002. Prognostic factors for mortality and thromboembolism in canine immune-mediated hemolytic anemia: a retrospective study of 72 dogs. *J. Vet. Intern. Med.* **16**: 504–509. [[Medline](#)] [[CrossRef](#)]
2. Ettinger, S. J. and Feldman, E. C. 2010. Textbook of Veterinary Internal Medicine, 7th ed., Elsevier Health Sciences, London.
3. Ghelfi, J., Thony, F., Frandon, J., Rodiere, M., Leroy, V. and Vendrell, A. 2016. Gastrointestinal bleeding due to pancreatitis-induced splenic vein thrombosis: Treatment with percutaneous splenic vein recanalization. *Diagn. Interv. Imaging* **97**: 677–679. [[Medline](#)] [[CrossRef](#)]
4. Hardie, E. M., Vaden, S. L., Spaulding, K. and Malarkey, D. E. 1995. Splenic infarction in 16 dogs: a retrospective study. *J. Vet. Intern. Med.* **9**: 141–148. [[Medline](#)] [[CrossRef](#)]
5. Jacoby, R. C., Owings, J. T., Ortega, T., Gosselin, R. and Feldman, E. C. 2001. Biochemical basis for the hypercoagulable state seen in Cushing syndrome; discussion 1006–7. *Arch. Surg.* **136**: 1003–1006. [[Medline](#)] [[CrossRef](#)]
6. Laurenson, M. P., Hopper, K., Herrera, M. A. and Johnson, E. G. 2010. Concurrent diseases and conditions in dogs with splenic vein thrombosis. *J. Vet. Intern. Med.* **24**: 1298–1304. [[Medline](#)] [[CrossRef](#)]
7. Mattoon, J. S. and Nyland, T. G. 2014. Small animal diagnostic ultrasound, 3rd ed., Saunders, Philadelphia.
8. Respass, M., O'Toole, T. E., Taeymans, O., Rogers, C. L., Johnston, A. and Webster, C. R. L. 2012. Portal vein thrombosis in 33 dogs: 1998–2011. *J. Vet. Intern. Med.* **26**: 230–237. [[Medline](#)] [[CrossRef](#)]
9. Spangler, W. L. and Culbertson, M. R. 1992. Prevalence, type, and importance of splenic diseases in dogs: 1,480 cases (1985–1989). *J. Am. Vet. Med. Assoc.* **200**: 829–834. [[Medline](#)]
10. Thrall, D. E. 2013. Textbook of Veterinary Diagnostic Radiology, 6th ed. Elsevier Health Sciences, London.
11. Uy, P. P. D., Francisco, D. M., Trivedi, A., O'Loughlin, M. and Wu, G. Y. 2017. Vascular diseases of the spleen: a review. *J. Clin. Transl. Hepatol.* **5**: 152–164. [[Medline](#)]
12. Van Winkle, T. J. and Bruce, E. 1993. Thrombosis of the portal vein in eleven dogs. *Vet. Pathol.* **30**: 28–35. [[Medline](#)] [[CrossRef](#)]