Surgical correction of a splenophrenic shunt in a dog: a case report

H. Yoon[1], M. Roh[2], S. Jeong[1]

[1]College of Veterinary Medicine, Konkuk University, Seoul, South Korea
[2]Duckso Animal Hospital, Wabu, Namyangju, Kyunggido, South Korea

ABSTRACT: A 4.3 kg, nine-year-old, spayed female Shih Tzu was presented for a two-month history of seizures, ataxia, and hyper-salivation. A diagnosis of a splenophrenic shunt was made by use of computed tomography angiography with volume-rendered imaging. A cellophane band was placed around the shunt after its isolation from the central tendon of the diaphragm. Clinical signs continued to wax and wane. Preprandial and postprandial bile acids levels were still elevated 10 months after surgery. An ameroid ring constrictor was placed around the shunt vessel before the vessel entered the diaphragm from its caudal aspect. At three months after the second surgery, the dog was near the normal ranges of preprandial and postprandial bile acids. Although a study of the anatomy of different types of extrahepatic portosystemic shunts has been reported in dogs, to the authors' knowledge, there is a lack of information on clinical presentation, treatment, and postoperative results in a specific type of extrahepatic portosystemic shunt, such as a splenophrenic shunt. Cellophane banding should be avoided for occlusion of a splenophrenic shunt passing along the central tendon of the diaphragm.

Keywords: ameroid ring constrictor; cellophane band; splenophrenic shunt; dog

Extrahepatic portosystemic shunts are anomalous vessels that join the portal circulation and systemic venous circulation, resulting in neurological, gastrointestinal, or urological manifestations (Windsor 2007; Doran et al. 2008; Mertens et al. 2010). Extrahepatic portosystemic shunts are mostly portocaval, originating from the portal vein and draining into the caudal vena cava, followed by portoazygos, branching from the portal vein to the azygos or left hemiazygos vein (Broome et al. 2004; Windsor 2007). Extrahepatic portosystemic shunts can be characterised into different types such as splenocaval, splenophrenic, splenoazygos, double right gastric azygos, right gastric caval, and double right gastric caval, depending on exact shunt location or morphology (Nelson and Nelson 2011). Treatments usually include medical therapy to stabilise the patients, followed by surgical attenuation of the anomalous vessel (Broome et al. 2004; Doran et al. 2008; Worley and Holt 2008). Although a study of the anatomy of different types of extrahepatic portosystemic shunts has been reported in dogs, to the authors' knowledge, there is a lack of information on clinical presentation, treatment, and postoperative results in a specific type of extrahepatic portosystemic shunt, such as a splenophrenic shunt (Nelson and Nelson 2011). The purpose of this case report was to describe the clinical presentation, diagnosis, and successful surgical management of a splenophrenic shunt in a dog.

Case description

A 4.3 kg, nine-year-old, spayed female Shih Tzu was presented for a two-month history of seizures, ataxia, and hyper-salivation. The owner reported that the dog showed lethargy, progressive inappetence, weight loss, vomiting, and diarrhoea for two years. Preoperatively, the patient's status was evaluated clinically and using laboratory testing. Abdominal radiographs and computed tomography angiography (CTA) with volume-rendered imaging (VRI) were performed to evaluate the size of the liver and accurately identify the origin and termination of any shunt vessels. Preoperative therapy

included low-protein diet, lactulose (1.5 ml, *p.o.*, tid; Duphalac®, Choongwae Pharm, Korea) and metronidazole (10 mg/kg, *p.o.*, tid; Flasinyl CJ tab®, CJ Cheil Jedang, Korea) for seven days. The dog was premedicated for surgery with atropine sulphate (0.02 mg/kg *s.c.*; Atropine sulfate inj®, Je Il Pharm. Co., Ltd, Korea), followed by anaesthetic induction with propofol (6 mg/kg *i.v.*; Provive 1%®, Myungmoon Pharm. Co., Ltd, Korea). The dog was intubated and anaesthesia was maintained with isoflurane (Isoflurane®, Choongwae. Co., Ltd, Korea) and oxygen. Lactated Ringer's solution was administered intravenously at a rate of 5 ml/kg/h until completion of the surgical procedure. The dog received cefazolin (20 mg/kg *i.v.*; Safdin®, Daehan Newpharm. Co., Ltd, Korea) at the time of anaesthetic induction. Surgical correction using a cellophane band was performed with informed consent. Low-protein diet, lactulose (1.5 ml, *p.o.*, tid), and metronidazole (10 mg/kg, *p.o.*, tid) were prescribed postoperatively. At 10 months after surgery, the owner reported that the dog still showed lethargy, progressive inappetence, weight loss, vomiting, and diarrhoea. The patient's status was re-evaluated clinically and using laboratory testing. The CTA with VRI procedures were performed once again to evaluate the success or failure of shunt occlusion 10 months postoperatively. The second surgical correction using an ameroid ring constrictor was performed on the week following the second CTA. Postoperative care was the same as that implemented following the first surgery. At the third and tenth month postoperative checkups, clinical exams and laboratory testing including of preprandial and postprandial bile acids were performed to evaluate the success or failure of shunt occlusion.

RESULTS

Physical abnormalities on presentation included depression. Neurologic examination revealed mild ataxia of gait. Low alkaline phosphatase activity (22 IU/l, reference range; 23–212 IU/l), low serum total protein (4.3 g/dl, reference range; 5.2–8.2 g/dl), low serum albumin (1.8 g/dl, reference range; 2.2–3.9 g/dl), and low cholesterol levels (63 mg/dl, reference range; 110–320 mg/dl) were identified on serum biochemical profiling. Measurement of bile acid concentrations revealed elevated levels of preprandial and postprandial bile acids (Table 1). Abdominal radiographs revealed microhepatica. Neurologic dysfunction was attributed to hepatic encephalopathy, based on biochemical abnor-

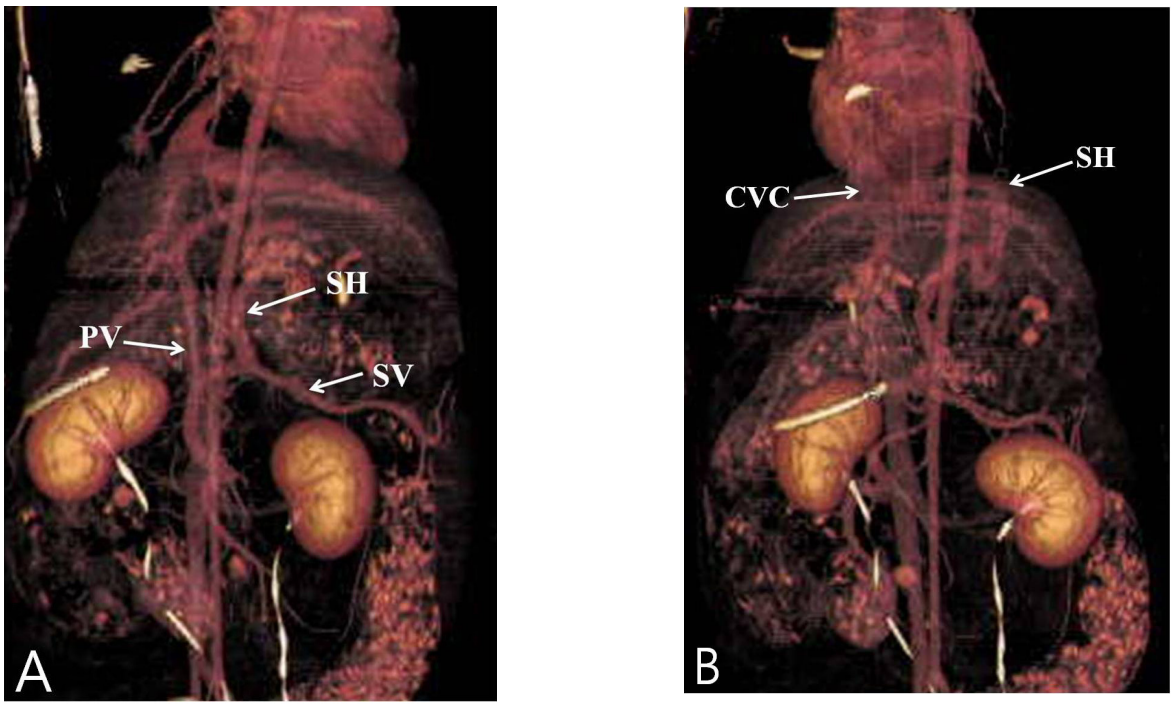

Figure 1. Preoperative volume-rendered imaging: the patient's right is on the left in all images. A portocaval shunt originates from the splenic vein (**A**) and terminates in the caudal vena cava cranial to the liver (**B**). PV = portal vein; SH = shunt; SV = splenic vein; CVC = caudal vena cava

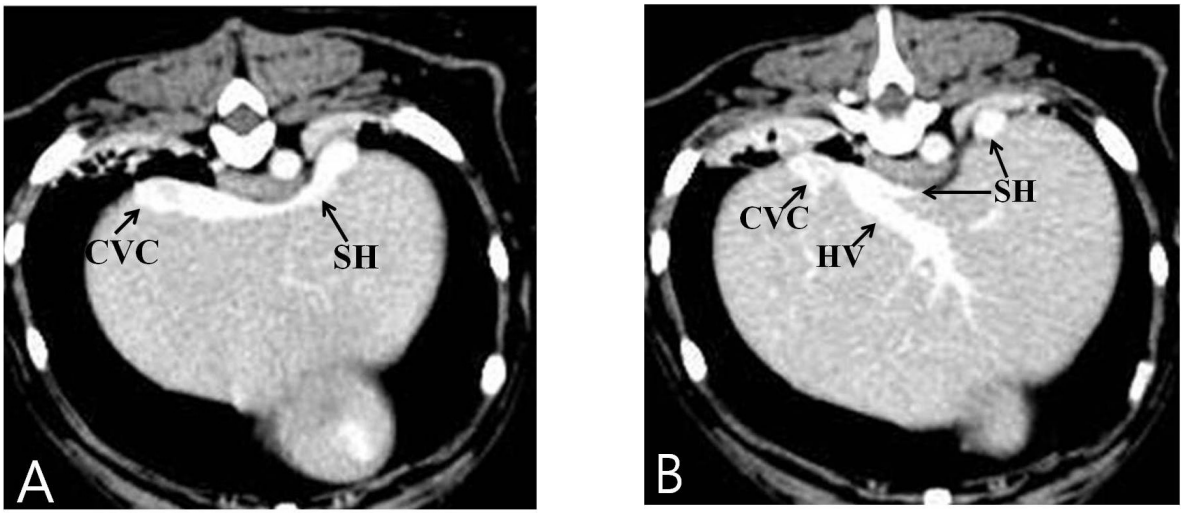

Figure 2. Preoperative transverse computed tomography angiography image after *i.v.* injection of iohexol: The patient's right is on the left in all images. The large shunting vessel passes cranial to the liver along the diaphragm and inserts into the caudal vena cava from the left side (**A**). The large hepatic vein enters this shunt from its ventral aspect, near where the shunt inserts on the caudal vena cava (**B**). CVC = caudal vena cava; SH = shunt; HV = hepatic vein

malities and identification of microhepatica on radiographs. CTA with VRI demonstrated that a portocaval shunt originated from the splenic vein and terminated in the caudal vena cava cranial to the liver (Figure 1). A transverse CTA image revealed that the large shunting vessel passed cranial to the liver along the diaphragm and inserted into the caudal vena cava from the left side (Figure 2). Large hepatic veins entered this shunt from its ventral aspect, near where the shunt inserted into the caudal vena cava (Figure 2). A diagnosis of a splenophrenic shunt was made.

Abdominal exploration was unremarkable except for the splenophrenic shunt. The liver was retracted to the right and caudally to approach the left part of the diaphragm. The shunt, originating from the splenic vein, passing cranial to the liver along the diaphragm with a small tributary shunt vessel, and inserting into the caudal vena cava from the left side, was identified (Figure 3). Concordance between CTA imaging with VRI and surgical findings was found. The shunt was temporarily occluded to verify that the correct vessel was identified and was isolated from the central tendon of the diaphragm by means of blunt dissection for placement of a cellophane band. An ethylene oxide-sterilised cellophane band formed by folding 1.5 cm-wide strip in thirds longitudinally was passed around the shunt. The cellophane band was secured using a haemoclip and 3-0 nylon (Nylon®, Namhae Chemical, Korea) without attenuation (Figure 3). No changes in heart rate, intestinal colour and motility, and pancreatic colour were identified. The linea alba, subcutaneous tissues, and skin were closed routinely. On physical examination 10 months after surgery, the neurological deficits and clinical signs including lethargy, inappetence, weight loss, vomiting, and diarrhoea continued to wax and wane. Preprandial and postprandial bile acids levels were still elevated 10 months after surgery (Table 1). The second

Table 1. Results of specialised biochemical test for liver function before 1st surgical shunt attenuation, 10 months after 1st surgical shunt attenuation, and three months after 2nd surgical shunt attenuation

Parameter	Before 1st surgical shunt attenuation	Ten months after 1st surgical shunt attenuation	Three months after 2nd surgical shunt attenuation
Preprandial bile acid[a] (μmol/l)	36.1	60.6	15.6
Postprandial bile acid[b] (μmol/l)	434.1	202.4	77.7

[a]reference range 0–10 μmol/l; [b]reference range 0–20 μmol/l

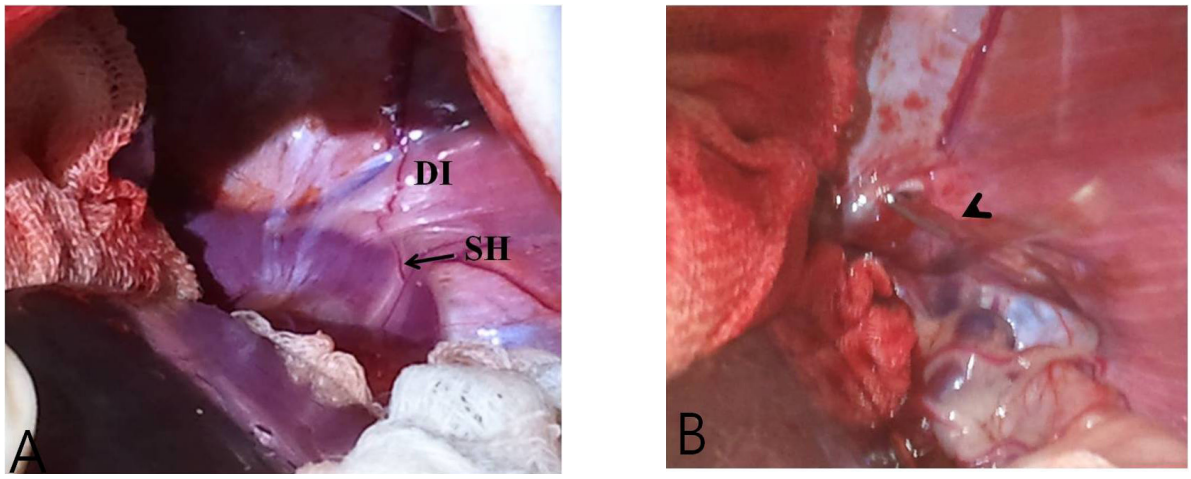

Figure 3. Intraoperative photographs of cellophane banding: the patient's right is on the left in all images. The shunt, originating from the splenic vein, passing cranial to the liver along the diaphragm, and inserting into the caudal vena cava from the left side, can be observed (**A**). The cellophane band (arrow head) is secured using a haemoclip without attenuation (**B**). DI = diaphragm; SH = shunt

CTA with VRI revealed that there was evidence of portosystemic communication and no attenuation of the shunt on the level of the cellophane band with the haemoclip (Figure 4). During the second surgery, there was no evidence of fibrosis formation between the cellophane band and shunt wall. No tributaries of the shunt were identified proximal to the site of constrictor placement. The constrictor with 5 mm internal diameters was placed around the shunt vessel before the vessel entered the diaphragm from its caudal aspect (Figure 5). Postoperative radiographs revealed that the constrictor was located caudal to a haemoclip used to secure the cellophane band on the first surgery (Figure 6). Serum biochemical profiling revealed nothing remarkable three months postoperatively. Bile acid concentrations were near the normal range of preprandial and postprandial bile acids (Table 1). On physical examination 10 months after surgery, there was no evidence of seizures, ataxia, hyper-salivation, lethargy, inappetence, weight loss, vomiting, and diarrhoea.

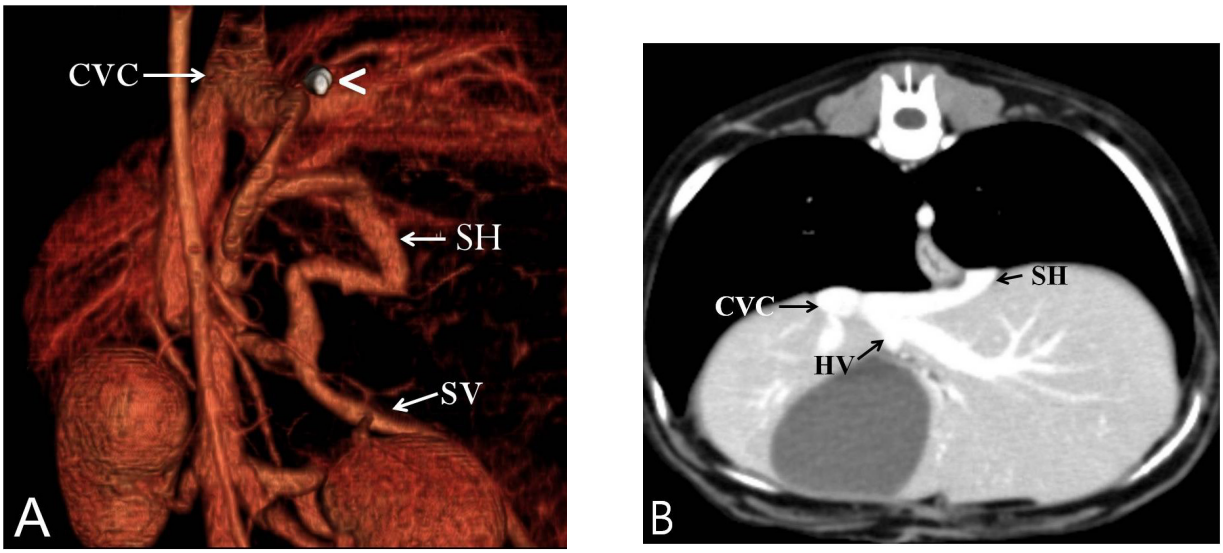

Figure 4. Postoperative volume-rendered imaging (VRI, **A**) and transverse computed tomography angiography image (CTA, **B**) 10 months after surgery: A haemoclip (arrow head) used to secure the cellophane band is identified on VRI. There is evidence of portosystemic communication and no attenuation of the shunt on the level of the cellophane band on CTA and VRI. CVC = caudal vena cava; SH = shunt; SV = splenic vein; HV = hepatic vein

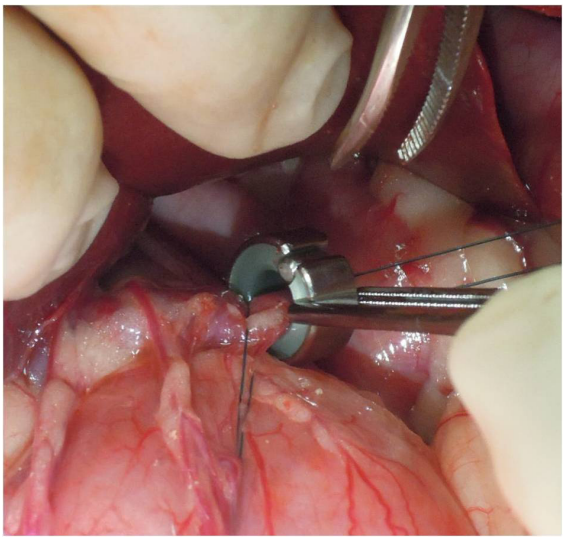

Figure 5. Intraoperative photograph of placement of an ameroid ring constrictor: an ameroid ring constrictor with 5 mm internal diameter is placed around the shunt vessel before the vessel enters the diaphragm from its caudal aspect

DISCUSSION AND CONCLUSIONS

Congenial portosystemic shunts are typically classified as intrahepatic or extrahepatic, based on their location (Fossum 2007; Berent and Tobias 2012). Extrahepatic shunts are typically single vessels and arise from the portal vein or a major tributary including left gastric, gastroduodenal, splenic, cranial mesenteric, and caudal mesenteric veins, and drain into the caudal vena cava cranial to the phrenicoabdominal vein (Tobias 2003; Mehl et al. 2005). In a report from 2011, six general conformations of an extrahepatic portosystemic shunt were identified using CTA: a splenocaval shunt arising from the splenic vein and terminating in the caudal vena cava caudal to the liver, a splenophrenic shunt arising from the splenic vein and terminating in the caudal vena cava cranial to the liver along the diaphragm, a splenoazygos shunt arising from the splenic vein and terminating in the azygos vein, a right gastric caval shunt extending along the lesser curvature of the stomach and inserting on the caudal vena cava, a right gastric azygos shunt with a caudal loop arising from the gastroduodenal vein and terminating in the azygos vein, and a right gastric caval shunt with a caudal loop extending from the gastroduodenal vein and terminating in the caudal vena cava (Nelson and Nelson 2011). In this earlier study, an extrahepatic portosystemic shunt, draining into the caudal vena cava cranial to the phrenicoabdominal vein and caudal to the liver was the most common type, occurring in approximately 48% of all extrahepatic portosystemic shunt cases, followed by the shunt type, which drained into the azygos vein (36%) (Nelson and Nelson 2011). The type, which drained into the caudal vena cava cranial to the liver along the diaphragm, occurred least often, in 16% of cases (Nelson and Nelson 2011). In embryology, congenial extrahepatic portosystemic shunts are believed to result from abnormal communications between the vitelline and cardinal venous systems (Broome et al. 2004). These two venous systems contribute to formation of the portal vein and its tributaries

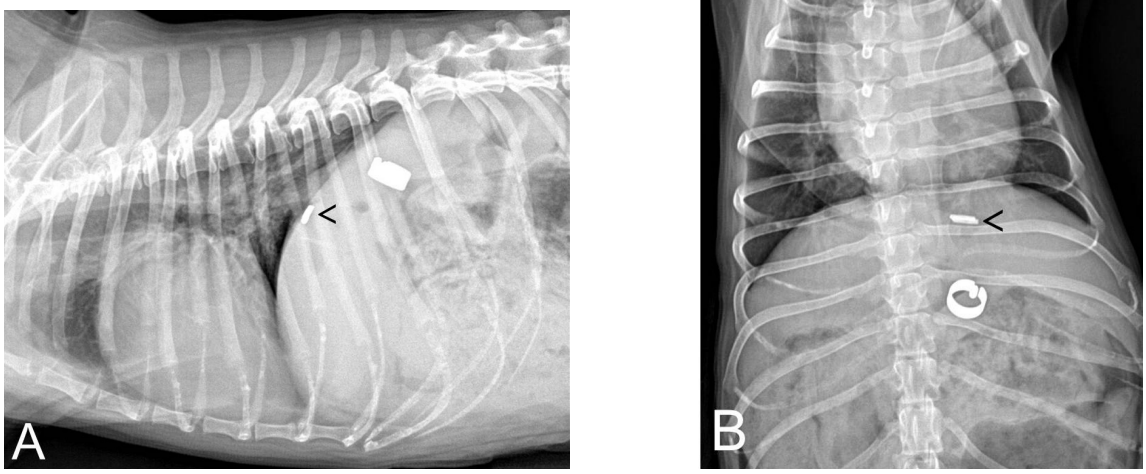

Figure 6. Postoperative lateral (**A**) and ventrodorsal (**B**) radiographs after constrictor placement: the constrictor is located caudal to a haemoclip (arrow head) used to secure the cellophane band on the first surgery

and the remaining intra-abdominal veins including the caudal vena cava, respectively, caudal to the liver. Therefore, if abnormal communication between these two systems occurs, there could be a greater possibility of the shunt type, which drains into the caudal vena cava caudal to the liver than cranial to the liver along the diaphragm (Broome et al. 2004). In the case reported here, the shunt type was a splenophrenic shunt, originating from the splenic vein, passing cranial to the liver along the diaphragm, and inserting into the caudal vena cava from the left side.

In the case reported here, CTA provided detailed anatomical information of the extrahepatic portosystemic shunt for a definitive diagnosis. A previous report has described considerable variation in the accuracy of ultrasonography for the diagnosis of portosystemic shunts, ranging between 67% and 100% (Haers et al. 2007). However, ultrasonography is suitable for identifying extrahepatic portosystemic shunts due to its availability and non-invasiveness without anaesthesia.

The diaphragm consists of the muscle that surrounds the central tendon on all sides, and central tendon where the caval foramen is located towards the right side (Evans 1993). The splenophrenic shunt vessels originate from the splenic vein, pass cranial to the liver along the diaphragm, and insert into the caudal vena cava from the left side. Therefore, the part of the shunt vessel passing along the diaphragm is mostly located in the central tendon of the diaphragm. Isolation of the shunt vessel from the tough central tendon of the diaphragm is more difficult, dangerous, and time-consuming than isolation of a shunt enclosed in soft tissue. In the case reported here, isolation of the shunt was the most time-consuming procedure during the first surgery since the shunt vessel was inserted into the central tendon fibres.

In a study from 1999, the femoral vein was dissected from the surrounding connective tissue without any tissue attachment and fibrosis could be easily stimulated by the cellophane resulting in vessel occlusion (Youmans and Hunt 1999). However, in the case reported here, the shunt vessel was inserted into the central tendon fibres and it was not possible to dissect the shunt without the tendon fibres. A tendon is a tough band of fibrous connective tissue (Evans 1993). Failure to occlude the shunt could be attributed to the tough tendon fibres attached to the shunt vessel although an inflammatory response was caused by the cellophane band.

A cellophane band and ameroid ring constrictor have been described to be effective for slow occlusion of portosystemic shunts (Hunt et al. 2004; Frankel et al. 2006; Leshem et al. 2008). The cellophane band and banding technique described in the case reported here were the same as in a previous case report where computed tomography angiography demonstrated no evidence of portosystemic communication on the level of the cellophane band and caudal to the cellophane band (Yoon et al. 2011). In the case reported here, there was a small tributary shunt vessel along the diaphragm, inserting into the main shunt vessel. At the first surgical correction, a cellophane band was placed proximal to the small tributary shunt vessel to occlude both the main and small tributary shunt vessels; however, CTA demonstrated no attenuation of the shunt on the level of the cellophane band. Failure or success of shunt occlusion do not depend on the technique or equipment used, such as a cellophane band and ameroid ring constrictor used for stimulation of fibrosis, rather on the site where the equipment are placed. Portosystemic shunt ligation might be a surgical option together with intensive assessment of postocclusion portal pressure in cases where the shunt vessel is inserted into the central tendon fibres and it is not possible to dissect the shunt without the tendon fibres.

When portal blood bypasses the hepatic sinusoids, the liver is deprived of hepatotrophic substances including insulin and glucagon, and the systemic circulation and brain are exposed to non-metabolised intestinal toxins (Youmans and Hunt 1999). Medical management may control hepatic encephalopathy in the short term; however, a progressive deterioration in liver function occurs until shunt occlusion (Worley and Holt 2008). In the case reported here, pre- and post-operative therapy including low-protein diet, lactulose, and metronidazole was effective in controlling neurological deficits and clinical signs in the short time despite the failure in shunt occlusion. However, the neurological deficits and clinical signs continued to wax and wane until placement of an ameroid ring constrictor.

Choosing an appropriate technique to treat a splenophrenic shunt can be difficult. The following surgical considerations may increase the likelihood of successful surgery: cellophane banding should be avoided for the occlusion of a splenophrenic shunt passing along the central tendon of the diaphragm; isolation of a splenophrenic shunt vessel before the

vessel enters the diaphragm from its caudal aspect is recommended for placement of a cellophane band or ameroid ring constrictor.

REFERENCES

Berent AC, Tobias KM (2012): Hepatic vascular anomalies. In: Tobias KM, Johnston SA (eds.): Veterinary Surgery Small Animal. Elsevier Saunders, St Louis, Missouri. 1624–1658.

Broome CJ, Walsh VP, Braddock JA (2004): Congenital portosystemic shunts in dogs and cats. New Zealand Veterinary Journal 52, 154–162.

Doran IP, Barr FJ, Moore AH, Knowles TG, Holt PE (2008): Liver size, bodyweight, and tolerance to acute complete occlusion of congenital extrahepatic portosystemic shunts in dogs. Veterinary Surgery 37, 656–662.

Evans HE (1993): Diaphragm. In: Evans HE (ed.): Miller's Anatomy of the Dog. 3rd ed. WB Saunders, Philadelphia, Pennsylvania. 304–308.

Fossum TW (2007): Surgery of the liver. In: Fossum TW (ed.): Small Animal Surgery. 3rd ed. Mosby, St Louis, Missouri. 539–553.

Frankel D, Seim H, MacPhail C, Monnet E (2006): Evaluation of cellophane banding with and without intraoperative attenuation for treatment of congenital extrahepatic portosystemic shunts in dogs. Journal of the American Veterinary Medical Association 228, 1355–1360.

Haers H, Paepe H, Vermote K, Saunders JH, Daminet S, Risselada M (2007): Portosystemic shunts in dogs and cats: imaging portosystemic shunts in small animals-ultrasonography, nuclear scintigraphy, computed tomography, magnetic resonance imaging. Vlaams Diergeneeskundig Tijdschrift 76, 322–330.

Hunt GB, Kummeling A, Tisdall PL, Marchevsky AM, Liptak JM, Youmans KR, Goldsmid SE, Beck JA (2004): Outcomes of cellophane banding for congenital portosystemic shunts in 106 dogs and 5 cats. Veterinary Surgery 33, 25–31.

Leshem SS, Lotsikas PJ, Reimer B (2008): In vitro expansion patterns of ameroid ring constrictors. American Journal of Veterinary Research 69, 1520–1524.

Mehl ML, Kyles AE, Hardie EM, Kass PH, Adin CA, Flynn AK, De Cock HE, Gregory CR (2005): Evaluation of ameroid ring constrictors for treatment for single extrahepatic portosystemic shunts in dogs: 168 cases (1995–2001). Journal of the American Veterinary Medical Association 226, 2020–2030.

Mertens M, Fossum TW, Willard MD, Fosgate GT, de la Paz AG, Farmer R, Miller MW (2010): Diagnosis of congenital portosystemic shunt in miniature schnauzers 7 years of age of older (1997–2006). Journal of the American Animal Hospital Association 46, 235–240.

Nelson NC, Nelson LL (2011): Anatomy of extrahepatic portosystemic shunts in dogs as determined by computed tomography angiography. Veterinary Radiology and Ultrasound 52, 498–506.

Tobias KM (2003): Portosystemic shunts and other hepatic vascular anomalies. In: Slatter D (ed.): Textbook of Small Animal Surgery. 3rd ed. Saunders, Philadelphia, Pennsylvania. 727–752.

Windsor RC (2007): Congenital portosystemic shunts in five mature dogs with neurological signs. Journal of the American Animal Hospital Association 43, 322–331.

Worley DR, Holt DE (2008): Clinical outcome of congenital extrahepatic portosystemic shunt attenuation in dogs aged five years and older: 17 cases (1992–2005). Journal of the American Veterinary Medical Association 232, 722–727.

Yoon HY, Choi YD, Han HJ, Kim SW, Kim KH, Jeong SW (2011): Contrast-enhanced computed tomography angiography and volume-rendered imaging for evaluation of cellophane banding in a dog with extrahepatic portosystemic shunt. Journal of the South African Veterinary Association 82, 125–128.

Youmans KR, Hunt GB (1999): Experimental evaluation of four methods of progressive venous attenuation in dogs. Veterinary Surgery 28, 38–47.

Received: 2014–04–03
Accepted after corrections: 2014–09–11

Corresponding Author:

Soon-Wuk Jeong, Konkuk University, College of Veterinary Medicine, Department of Veterinary Surgery, Seoul 143-701, South Korea

Tel. +82 2 450 3670, E-mail: swjeong@konkuk.ac.kr