

Change in the Liver Size Following Cellophane Banding for the Gradual Attenuation of a Congenital Extrahepatic Portosystemic Shunt in a Dog

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KEY WORDS: bile; cellophane; liver; portal vein; portosystemic shunt

ABSTRACT

A 1.35-kg, 1-year-old, intact male poodle dog with a congenital extrahepatic portosystemic shunt was referred to our clinic. Abdominal radiographs revealed microhepatia. Abdominal ultrasonography revealed a large, tortuous shunt vessel arising from a portal vein branch that inserted into the caudal vena cava. Laparotomy was performed, and a single, extrahepatic shunt was identified. Cellophane banding was performed on the shunt vessel, with no attenuation. Serum chemical values recovered gradually, and abdominal radiographs revealed a normal-sized liver 10 days after surgery. To our knowledge, this is the first case report to demonstrate an increase in the liver size and diameter of the cranial portion of the portal vein within a short period after successful cellophane banding without intraoperative attenuation.

INTRODUCTION

The portal vein carries blood from the stomach, intestines, pancreas, and spleen to the liver, delivering 75–80% of the liver's afferent blood supply and 50% of its oxygen supply.¹ A portosystemic shunt (PSS) is an abnormal vascular connection between the portal vein and the systemic venous system.^{2,3} Animals with a congenital PSS exhibit inadequate liver development because important hepatotropic factors from the pancreas and intestines do not flow into the liver, resulting in hepatic atrophy and microhepatia associated with hepatic encephalopathy.^{2,4,5,6}

There are many surgical treatment options for extrahepatic PSS, including acute occlusion with ligatures or gradual attenuation with ameroid rings, cellophane bands, or hydraulic occluders. After the use of surgical methods for gradual attenuation, the prognosis can be evaluated through liver function assessments based on laboratory

Figure 1. Preoperative radiographic images obtained in left lateral (A) and ventrodorsal (B) recumbency for a 1-year-old intact male poodle with a congenital extrahepatic portosystemic shunt

Microhepatica can be observed. The hepatic ratio (liver length/length of the second lumbar vertebral body, arrows in A) is 2.3.



parameters such as bile acid and fasting ammonia levels. However, it has been reported that normalization of serum bile acid levels may not be associated with the long-term outcome.^{7,8,9}

The liver size is correlated with the portal vein flow and liver function.¹⁰ When ligation is used for shunt occlusion, the greatest increase in the liver size occurs between 0 and 8 days in many dogs.¹¹ The correlation between the changes in the hepatic volume and the gradual occlusion of congenital extrahepatic PSS has been described for surgical treatment using an ameroid ring constrictor.⁶ However, there are no existing reports describing changes in the liver size following cellophane banding for complete shunt occlusion.

In this case report, we describe changes in the liver size following surgery using cellophane banding without intraoperative attenuation in a dog with a congenital extrahepatic PSS. To our knowledge, this is the first case report that demonstrates an increase in the liver size after a successful cellophane banding procedure without intraoperative attenuation.

CASE PRESENTATION

A 1-year-old, intact male poodle dog weighing 1.35 kg presented for evaluation of a congenital extrahepatic PSS. The owner

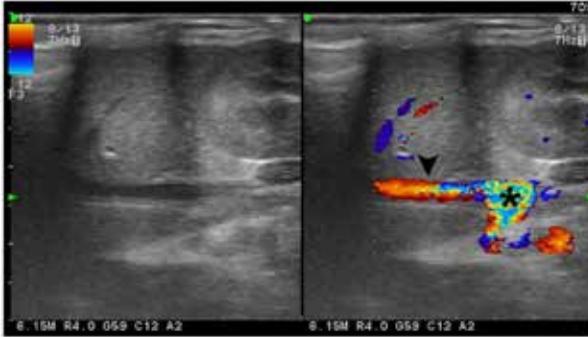
reported that the dog had been experiencing vomiting, diarrhea, and anorexia since 1 month. Laboratory tests conducted at a local animal hospital 2 weeks prior revealed hyperammonemia, hypoglycemia, and hypoalbuminemia. Physical examination revealed that the dog was emaciated (body condition score, 2/5) and exhibited cryptorchidism.

Laboratory testing revealed the following:

- mild normocytic anemia (mean cell volume, 60 fl; reference range, 60–74 fl)
- normochromic anemia (mean corpuscular hemoglobin concentration, 30 %; reference range, 31%–36 %)
- anemia (packed cell volume, 34.75 %; reference range, 37%–55%)
- mild prolongation of the clotting time (prothrombin time, 18.3 sec; reference range, 11–17 sec)
- high serum aspartate aminotransferase (48 μ l; reference range, 15–43 μ l)
- serum alkaline phosphatase (178 μ l; reference range, 15–127 μ l)
- serum gamma-glutamyl transpeptidase (11.6 mg/dl; reference range, 15–43 mg/dl), and
- fasting serum bile acid (144.3 μ mol; reference range, 0–10.0 μ mol) levels
- an elevated blood ammonia level (199 μ mol/l; reference range, 0.0–92 μ mol/l),

Figure 2. An ultrasonography image of the abdomen in a 1-year-old intact male poodle with a congenital extrahepatic portosystemic shunt

The image shows a large, tortuous shunt vessel originating from a branch of the portal vein and coursing into the caudal vena cava (arrowhead). Turbulent flow in the shunt vessel (*) and at its junction with the caudal vena cava (arrowhead) is evident in color Doppler mode.



and

- low serum total protein (4.0 g/dl; reference range, 5.4–7.4 g/dl)
- serum blood urea nitrogen (4 mg/dl; reference range, 8–31 mg/dl)
- serum cholesterol (119 mg/dl; reference range, 135–345 mg/dl), and
- serum albumin (2.0 g/dl; reference range, 2.9–4.2 g/dl) levels.

Abdominal radiographs revealed microhepatica (hepatic ratio, 2.3; reference range, 4.8–5.0; Figure 1). The hepatic ratio is a value obtained by dividing the liver length by the length of the second lumbar vertebral body. The liver length (cm) was measured as the length of the axis from most cranial point of the diaphragm to the apex of the right medial lobe. Abdominal ultrasonography showed a large, tortuous shunt vessel originating from a branch of the portal vein that was coursing into the caudal vena cava (CVC).

Turbulent flow was identified in the shunt vessel and at its junction with CVC in color Doppler mode (Figure 2). Computed tomography (CT) angiography revealed that the shunt vessel originated from the right gastric vein and terminated in CVC, caudal to the liver, and that the splenic vein inserted into the shunt vessel (Figure 3). The diameters of the pre- and post-shunt portal veins were 4 and 2 mm, respectively. The shunt diameter (just before entering CVC) was approximately 4 mm. An extrahepatic portocaval shunt was diagnosed.

The dog was fed a protein-restricted diet and received metronidazole (10 mg/kg, PO TID) and lactulose (0.5 ml/kg, PO BID) for 7 days before surgical intervention. It was

premedicated with butorphanol (0.2 mg/kg, IV) and cefazolin (20 mg/kg, IV), and

Figure 3. Preoperative computed tomography angiography image (CTA, A) and preoperative volume-rendered image (VRI, B) for a 1-year-old intact male poodle with a congenital extrahepatic portosystemic shunt

CTA and volume-rendered imaging reveal that the shunt vessel (arrowheads) originates from the right gastric vein and terminates in the caudal vena cava (*), caudal to the liver. The splenic vein inserts into the shunt vessel (arrowhead) between the point of origin and insertion. The post shunt portal vein (arrow) enters the liver with a smaller diameter than that of the pre shunt portal vein.

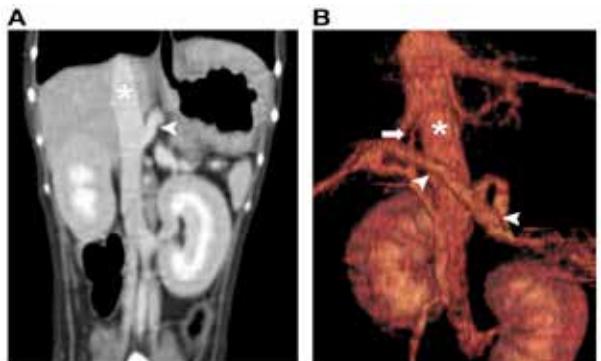


Figure 4. Photographs obtained during the cellophane banding procedure for a 1-year-old intact male poodle with a congenital extrahepatic portosystemic shunt

The small intestine is retracted to the left with the surgeon's fingers, exposing the epiploic foramen. The shunt, originating in the portal vein and flowing into the caudal vena cava, is identified at the epiploic foramen. A cellophane band (arrowhead) is inserted around the shunt without intraoperative shunt attenuation with a surgical clip.



anesthesia was induced with propofol (6 mg/kg, IV) and maintained with isoflurane in 100% oxygen.

A cellophane band was used for surgery with the owner's consent. Exploratory laparotomy using a ventral midline incision confirmed the small size of the liver. The mesoduodenum was retracted to the left to obtain access to the epiploic foramen. It was discovered that the shunt from the portal vein drained into CVC at the epiploic foramen. Blunt dissection was performed around the shunt to place the cellophane band. Surgical correction using the cellophane band was performed without intraoperative shunt attenuation with a surgical clip (Figure 4). At the time of surgery, intraabdominal cryptorchid castration

was performed, and liver biopsy samples were obtained for histological examination to determine the presence of intrahepatic microscopic vascular malformations.

Ten days after surgery, abdominal radiography and ultrasonography revealed that the hepatic ratio had increased from 2.3 to 4.6 (Figure 5), and a post-shunt portal vein diameter of 0.9–2.9 mm was noted. The preprandial bile acid level was within the reference range, while the postprandial level was higher than the reference range (Table 1).

Forty days after surgery, the hepatic ratio remained unchanged, and the post-shunt portal vein diameter had increased to 3.5 mm. The preprandial bile acid level remained within the reference range, while the postprandial bile acid level approached the reference range (Table 1). The post-shunt portal vein diameter had increased to 3.9 mm, and both the pre- and postprandial bile acid levels were within the reference range at the 6-month follow-up.

Thirteen months after surgery, the hepatic ratio and post-shunt portal vein diameter increased to 4.8 and 4 mm, respectively, which approximated the pre-shunt portal vein diameter of 4 mm. The preprandial bile acid level was within the reference range, but the postprandial level had increased. There was no evidence of clinical signs as-

Figure 5. Radiographs obtained after cellophane banding for a 1-year-old intact male poodle with a congenital extrahepatic portosystemic shunt

Postoperative radiographic images obtained in left lateral (A) and ventrodorsal (B) recumbency reveal a normal-sized liver. The hepatic ratio (liver length/the second lumbar vertebral body length, arrows in A) is 4.6, which confirms that the liver's size is approximately twice its preoperative size.



sociated with PSS.

DISCUSSION

Surgical techniques for the management of PSS involve acute occlusion with ligatures or gradual attenuation with ameroid constrictors, cellophane bands, or hydraulic occluders.¹² In the late 1990s, surgeons usually performed surgical ligation for single PSS occlusion, but up to 68% dogs that underwent shunt ligation reportedly developed postoperative portal hypertension.^{13,14} Subsequently, gradual attenuation of congenital PSS using cellophane bands or ameroid constrictors to prevent postoperative portal hypertension was described in many veterinary publications. Even though both devices result in gradual shunt attenuation, experimental studies have revealed that the time required for complete occlusion differs.

In a 1999 experimental study using the femoral vein of a dog, the femoral vein was completely occluded 3 weeks after surgery using an ameroid constrictor, but cellophane banding had not achieved complete occlusion of the vein 6 weeks after surgery.¹⁴ In many congenital PSS cases, ameroid constrictors induce complete occlusion within 2–5 weeks after placement.^{15,16} Cellophane bands with an internal diameter of 2.5 or 3 mm have been reported to induce complete shunt occlusion within approximately 8 weeks or <2.5 months, respectively.

Cellophane banding without intraoperative attenuation has been previously reported to completely occlude the shunt in >6 months.^{17,18} In a study using cellophane banding without attenuation, the size of a shunt with incomplete occlusion was larger than that of a shunt with complete occlusion. This suggests that large-diameter shunts might cause incomplete occlusion and may be more resistant to complete occlusion than small-diameter shunts.¹⁸ In the present case, cellophane banding was used without intraoperative attenuation, and the 4-mm-diameter shunt was nearly completely occluded 6 months after surgery.

With regard to the rate of shunt occlusion, the use of an ameroid constrictor or

partial attenuation of a shunt vessel to a diameter of <3 mm during cellophane banding may result in multiple acquired shunts because of the initial rapid occlusion.^{6,17} However, cellophane banding without intraoperative attenuation is not likely to cause multiple acquired shunts, because attenuation occurs more gradually with this procedure than with an ameroid constrictor or partial attenuation using cellophane bands.^{17,19,20} With the use of an ameroid constrictor, the shunt was reported to be rapidly occluded within 2 weeks after surgery, because the inflammatory reaction and luminal narrowing occurred within a short period.^{14,16,21,22} As observed with ameroid constrictors, cellophane banding with intraoperative attenuation also achieves rapid occlusion in a short time following surgery because of an added inflammatory reaction during shunt narrowing.¹⁴ However, in experiments conducted using the same internal implant diameter, the degree of rapid occlusion after surgery was more than three times greater in the group that received ameroid constrictors than in the group that received cellophane bands.¹⁴

No studies have investigated the rate of shunt occlusion using cellophane banding without intraoperative attenuation. In the present case report describing the use of cellophane banding without intraoperative attenuation, rapid shunt occlusion could be assumed to occur 10 days after surgery, taking into account the significant changes in the pre- and postprandial bile acid levels, hepatic ratio, and post-shunt portal vein diameter. Rapid occlusion probably occurred within a short time after surgery because of the development of a rapid acute inflammatory reaction. Therefore, when cellophane banding is performed, intraoperative attenuation is not necessary because the inflammatory reaction and subsequent shunt occlusion occur quickly. Evaluation of a larger number of cases is required to determine the timing of rapid attenuation in shunts treated without intraoperative attenuation.

The liver size varies according to multiple factors, including age and body weight,

and the weight of a normal liver equates to 3.38% of the total body weight.²³ In patients with chronic inflammation, fibrosis, cirrhosis, atrophy of the liver, or congenital PSS, the liver may be smaller than normal. In dogs with PSS, the supply of hepatotropic factors decreases and microhepatica occurs because most of the portal blood bypasses the liver and the portal blood flow into the liver is reduced.^{4,7} The portal vein pressure and flow play an important role in liver regeneration.^{4,23} With the use of ameroid constrictors and cellophane banding to attenuate PSS, the increase in the portal vein flow and pressure after surgery can increase liver regeneration and, consequently, the liver size.

In the present case, the post-shunt portal vein diameter was 2 mm before surgery and 2.9, 3.5, and 3.9 mm 10 days, 40 days, and 6 months after surgery, respectively. Because an increased liver size results in increased portal flow into the liver, measuring changes in the liver size following surgery for PSS is very important to determine the postoperative prognosis.^{6,11} Ligation of the shunt resulted in the maximum liver volume regeneration at 8 days after surgery, and there was no significant change in the liver volume at 1 and 2 months after surgery.¹¹

In a report investigating the use of ameroid constrictors, when the liver volume was measured 8 weeks after surgery, it was significantly larger.⁶ However, there are no existing reports regarding changes in the liver size according to the length of the postoperative period following cellophane banding. In the present case, similar to a report that investigated PSS ligation, the liver size increased rapidly until 10 days after surgery, with no significant increase at 40 days or 6 or 13 months after surgery. The increase in the liver size within a short period after surgery indicates that the portal flow into the liver had increased, even if the shunt was not completely occluded after cellophane banding. Moreover, the increase in the liver size suggests a lower possibility of portal

vascular resistance and portal hypertension.⁸

In veterinary medicine, the liver size is usually evaluated radiographically in the lateral and ventrodorsal positions and could be a very useful prognostic indicator for assessing the liver's functional capacity in animals.⁴ The hepatic ratio in dogs was first described by Cockett in 1986.²⁴ The liver length, measured using right lateral radiographs, has been correlated with the actual liver volume in dogs.²⁵ A few studies have measured the liver volume using CT angiography, but none have radiographically measured the liver size after PSS surgery.^{4,11} The ability to conduct follow-up studies by radiographic measurements of the liver size after surgery is essential because postoperative reimaging with CT may be costly and can subject the patient to further anesthetic burden. In the present case, we determined the hepatic ratio because plain radiography is an inexpensive, simple procedure that does not require anesthesia. A large number of case studies evaluated changes in the liver size following cellophane banding without intraoperative attenuation are needed.

In many patients, bile acid levels do not return to normal (preprandial, <10 $\mu\text{mol/l}$; postprandial, <25 $\mu\text{mol/l}$) after surgery, even after complete shunt occlusion.^{19,26} In some studies, the bile acid level decreased 2–4 months after ligation, ameroid constrictor placement, and cellophane banding, but thereafter, it gradually increased, albeit without related clinical signs.^{7,17,27} In one study involving the use of an ameroid constrictor, the mean bile acid level was the lowest at 30 days after surgery, and it gradually increased until 90 days.²⁷ The reason why bile acid levels and the degree of shunt occlusion are not associated remains unclear because of undefined liver pathologies such as congenital hepatic microvascular dysplasia and the variable hepatic mass regeneration ability of each individual.²⁷

In the present case, the preprandial bile acid level was normal at 10 days after surgery, while the postprandial level was

normal by 6 months. However, the latter increased again at 13 months, and the cause remains unknown. Further studies investigating the association between bile acid levels and shunt occlusion with cellophane banding without attenuation are needed. The bile acid test cannot be considered a reliable postoperative assessment tool for determining the time of treatment discontinuation or for confirming complete shunt occlusion; postoperative assessment factors should include the liver size and function and biochemical parameters such as blood urea nitrogen, albumin, and cholesterol levels.

To our knowledge, this is the first case report to demonstrate increased liver size and diameter of the cranial portion of the portal vein within a short period following a successful cellophane banding procedure without intraoperative attenuation. Cellophane banding without intraoperative attenuation results in the most gradual shunt occlusion. Therefore, the possibility of developing portal hypertension and acquired multiple shunts after surgery is lower than with other methods of gradual attenuation. Additionally, there is a small possibility that liver function is reduced by an incompletely occluded shunt. Since cellophane banding without intraoperative attenuation can result in increased liver size within a short period after surgery, even if complete occlusion does not occur, it is important to measure the change in liver size to determine the patient's postoperative prognosis.

ACKNOWLEDGEMENTS

None.

DISCLOSURE OF STATEMENT

Authors do not have any conflicts of interest.

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