Ultrasound guided Trans-hepatic Coil Embolization of an Intrahepatic Portosystemic Shunt in a Dog

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ABSTRACT

A 16-week-old, intact female Australian Shepherd puppy was diagnosed with a large congenital intrahepatic portosystemic shunt (PSS). A long needle loaded with a stainless steel coil which was coated by Dacron fibers was advanced, ultrasound guided, through the liver parenchyma into the lumen of the shunt. Once the needle tip was clearly visible within the lumen, the coil was pushed into the lumen of the shunt until approximately 70% occlusion was recorded using color Doppler. Then the needle was retracted and the rest of the coil was lodged within the liver parenchyma and surface. Within 8 months from the procedure there was complete occlusion of the shunt without any ill-effect. We describe a relative safe and effective method for a controlled, ultrasound guided, coil embolization of a large intrahepatic portosystemic shunt.

Keywords: Congenital Portosystemic Shunt (PSS); Dog; Coil embolization; Ultrasound guided

INTRODUCTION

Congenital portosystemic shunts (PSS) are abnormal vascular communications allowing some of the portal blood draining the abdominal organs to bypass the liver and directly enter the systemic circulation. Intrahepatic PSS are shunts within the liver parenchyma and are usually classified based on the supplying or draining vessels. Typically, right divisional shunts pass through either the caudate process of the caudate lobe or the right lateral lobe, central divisional shunts pass through either the right medial or the quadrate lobes, and left divisional shunts pass through the left medial, left lateral, or the papillary process of the caudate lobe before entering the vena cava (1-3). The aim of successful treatment is to attenuate the abnormal vessel in order to restore or improve hepatic perfusion from the portal vein and ultimately maximize hepatic function. Surgical techniques aiming at progressive shunt attenuation such as partial suture ligation and ameroid constrictor placement have been shown to perform unpredictably, either resulting in premature vascular occlusion (and development of multiple acquires shunts) or ultimately not achieving complete occlusion (4-6). Cellophane banding has been demonstrated to provide progressive vascular attenuation; however, it remains unclear whether complete attenuation is ultimately achievable with large shunt diameter, and the use of cellophane banding has a reported 27% mortality rate and 55% complication rate (7). During surgery, high complication rates and mortality were recorded in those difficult cases where the identification and isolation of the shunt required an invasive approach. Furthermore, only a minority of these cases could be completely occluded without resulting in life-threatening portal hypertension (7-10).

A less invasive trans-venous coil embolization with or without the deployment of a stent within the caudal vena cava (CVC) at the level of shunt entrance has been advocated (11, 12). However such procedures require experience and knowledge of interventional radiology, endovascular access and the use of relatively expensive equipment such as image-intensified fluoroscopy.

In this case report we describe a novel and relatively simple, safe and inexpensive new approach to attenuate intrahepatic shunts using trans-hepatic ultrasound guided coil deployment.

CASE REPORT

A 16-week-old, intact female Australian Shepherd puppy of 9.8kg bodyweight was presented for assessment to our referral centre for a suspected congenital portosystemic shunt (CPSS). The dog exhibited a variety of neurological signs including hyper-salivation, disorientation, head pressing, lethargy and inappetance, suggestive of hepatic encephalopathy.

On clinical examination the dog was quiet and weak with poor body condition. Routine hematological and biochemical studies were performed. A mild non-regenerative anemia with a packed cell volume (PCV) of 28% (Reference range (RR): 37 to 55 %) and red blood cell count (RBC) of $4.9x10^{12}$/litre (RR: 5.5 to $8.5x10^{12}$/litre). A mild neutrophilia of $16.0x10^{9}$/litre (RR: 3 to $11.7x10^{9}$/litre) and elevated alkaline phosphatase level of 234 iu (RR: 15-150 IU) were also detected. The results of a dynamic bile acid test were 51µmol/l (RR: 0 to 29 µmol/l) and 147µmol/l (1 to 38 µmol/l) pre- and postprandially, respectively. Urinalysis showed haematuria (3+) and proteinuria (1+).

Sonographically, there was a small liver with reduced visibility of the intrahepatic portal veins and a relatively large gall bladder (GB). The portal vein before it entered the porta hepatis was mildly prominent in size and had an abnormally irregular portal flow with increased velocity (up to 50cm/sec). From subcostal and right sided intercostal windows, a relatively short and plump anomalous vessel 0.8cm wide was found involving the right portal vein branch. This vessel looped laterally in the right lateral liver lobe, before connecting the adjacent caudal vena cava (CVC), with a vascular window measuring 1.0 cm. The turbulent flow connection to the CVC was confirmed with color Doppler (Figs. 1A &B).

The kidneys were enlarged (7.5 cm in length on the left and 7.8cm on the right) and cortices were hyperechoic in comparison with the liver. Mineral sediment and small (1-2mm) and calculi were present in the bladder.

Computed tomography (CT)-angiography was performed using a dosage of 2ml/kg of iohexol (Iohexol 300, 300mg/ml, Omnipaque, GE Healthcare, Cork, Ireland) intravenously by manual injection. Pre- and postcontrast transverse images of the abdomen (diaphragm to L4) were acquired helicoidally (MxTwin Dual slice helical CT, Picker, Israel) and reconstructed with a detailed soft tissue algorithm.

On the CT-angiographic images the liver was small and the GB appeared enlarged. The portal vein was marginally prominent at the level of the porta hepatis (similar diameter as the aorta). A fairly short and plump looping intrahepatic portosystemic shunt (Fig. 2) was detected in the right lateral liver lobe, reaching up to 1.0 cm in diameter.

Medical management was instituted, A canine low protein diet was fed with 10ml of lactulose (Lactulose solution BP; Sandoz Pharmaceuticals) and 300mg of ampicillin (Ampifen; Intervet) three times daily.

Due to technical difficulty and higher mortality rate of

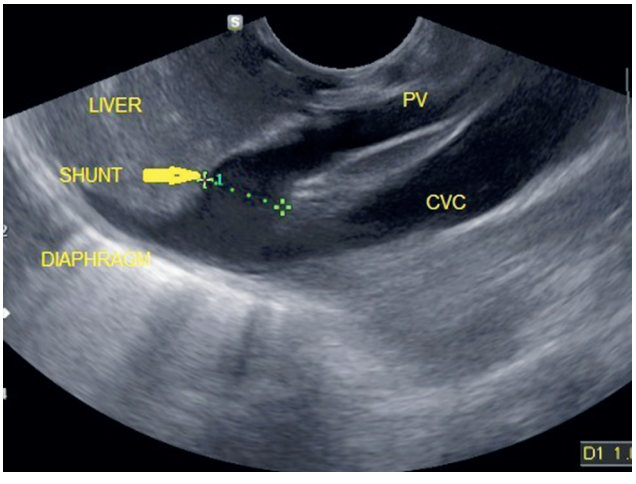

FIG 1A: Longitudinal intraoperative sonographic image of the right liver lobe shows prominent portal vein (PV) communication with the caudal vena cava (CVC) through a shunt (8-10mm in diameter). Note the small liver with poorly developed portal vessels.

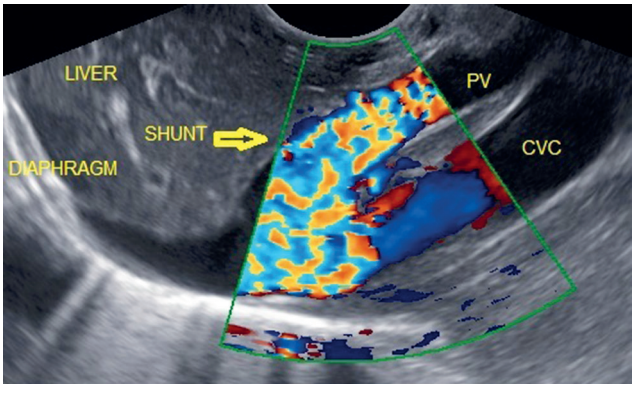

FIG 1B: Longitudinal intraoperative sonographic colour Doppler image of the intrahepatic shunt showing turbulent blood flow as mosaic pattern connecting the PV with the CVC

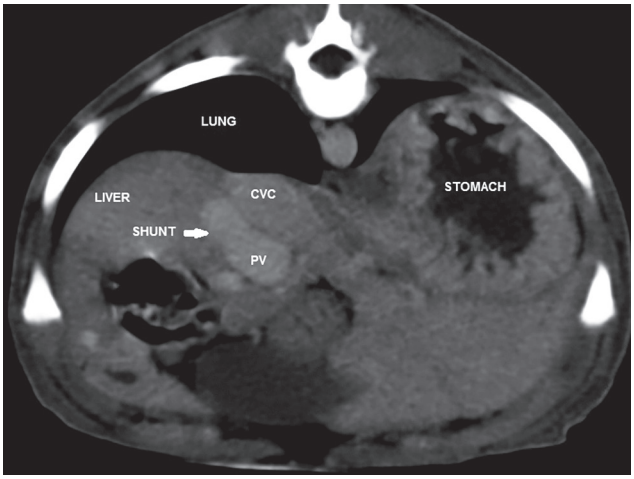

FIG 2: Contrast-enhanced computed tomographic (CT) transverse image of the liver obtained during the portal phase. A fairly short and plump intrahepatic portosystemic shunt is visible (arrow)

surgical ligation of intrahepatic shunt and financial constraints the owner elected to attempt a novel ultrasound guided transhepatic coil embolization of the portosystemic shunt.

The patient was premedicated with 0.2 mg/kg of morphine (Morphine sulphate; Antigen Pharmaceuticals) administered intramuscularly. Intravenous propofol (Rapinovet; Schering Plough Animal Health), at 4 mg/kg, enabled tracheal intubation and general anaesthesia was maintained using isoflurane (Forane; Abbott Laboratories) on 100 percent oxygen administered through a circle breathing system. The abdomen was clipped and prepared aseptically. A laparotomy (from xyphoid to level of 3rd vertebrae) was firstly performed and a 24G over-the-needle catheter was placed into the mesenteric vein for fluoroscopic portography. The mesenteric portographic study confirmed the diagnosis of single right sided intra-hepatic portocaval shunt. Thereafter the liver was scanned (Samsung HM70A) directly using a microconvex (8-12MHz) ultrasound endocavitary transducer covered by a sterile casing. Sagittal and transverse images were obtained using B-mode and color Doppler to evaluate the morphology and blood flow through the shunt which was identified as fusiform-tubular vessel connecting the portal vein and CVC.

A self-manufactured 16G, 15cm long needle was loaded with a stainless steel coil, which was coated by Dacron fibers and was 8-mm in diameter and 5-cm in length (Cook embolization coil, stainless steel). A self-manufactured needle stylet with distance markings was inserted into the needle

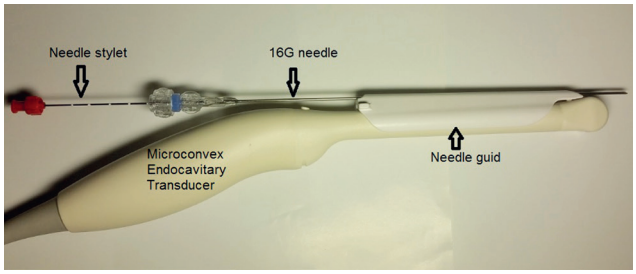

FIG 3: Photograph of the microconvex ultrasound endocavitary transducer with the 16G needle inserted into the needle guide. Note the stylet with the 1cm markings protruding from the needle. The stainless steel coil is loaded within the needle reaching tip of the needle (not seen).

lumen. At that point the loaded needle was inserted into the needle guide which was mounted onto the transducer (Fig 3).

Once the shunt vessel was identified and scanned at a longitudinal orientation the loaded needle was advanced through the liver parenchyma, the wall of the vessel and into the lumen of the shunt. When the needle tip was clearly visible within the lumen the marked stylet was gradually advanced into the needle pushing the coil into the lumen of the shunt. The coil was advanced into the lumen until early intestinal changes of cyanosis, hypermotility and increased mesenteric vascular pulsation were noted. At that stage 3cm of the coil was released according to the markings on the stylet. Sagittal and transverse images of the shunt were obtained using B-mode and color Doppler flow to evaluate the degree of blood flow through the shunt. A void in blood flow was indicative of the presence of thrombus. Approximately 70% occlusion of the shunt lumen diameter was achieved. At that point the needle was retracted while the rest of the coil was being released into the liver parenchyma in order to anchor the coil.

Immediate post procedure ultrasound scan did not show any bleeding or coil displacement (Fig 4A). Using color Doppler (Fig 4B) it was evident that there was approximately 70% attenuation of the shunt vessel and the portal vein flow appeared to have decreased to 20-25cm/sec (normal mean velocity; 15cm/sec).The operative time from applying the transducer directly on the liver to the deployment of the coil was 15 minutes. The abdominal wall was closed in a standard three-layer manner. Anesthetic recovery was slow but uneventful.

The dog began eating small amounts of canine selected protein diet on the first day post operatively. Medication

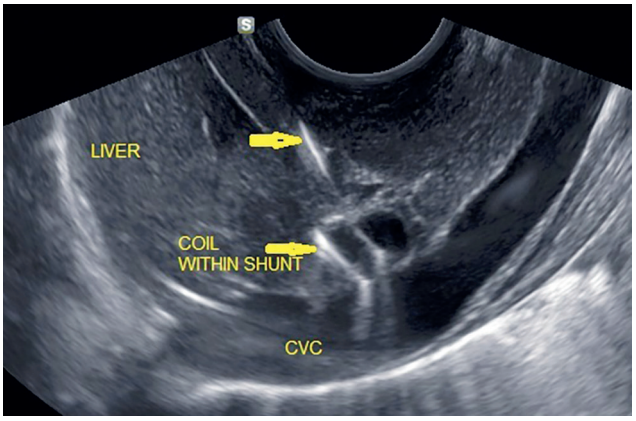

FIG 4A: Sonogram immediately post coil deployment shows the coil at an orthogonal position in relation to the long axis of the vessel obliterating most of the lumen. Note the coil tracking within the liver parenchyma to the margin (arrows).

(Lactulose & ampicillin) was continued as before the procedure. One week post procedure the owner reported that the puppy continued to be bright and had increased stamina and normal demeanor. One month post procedure the dog was found to be clinically normal by the referring veterinarian. Routine blood chemistry and hematology results were reported to be within normal limits. At that time medical management with lactulose ampicillin was stopped.

On follow-up examination two months post procedure the dog looked clinically normal and fasting and post prandial serum bile acid levels returned to normal values5.0 µmol/l and 10µmol/l respectively.

Ultrasound examination of the abdomen at that time was unremarkable. Blood flow through the liver lobes as seen using colour Doppler appeared to have subjectively increased. Flow through the portal vein at the level of porta hepatis was 15-25cm/sec. The tip of the coil was clearly visible as lodged spring like within the liver parenchyma while the coil within the shunt's lumen was covered by a bulky thrombus obliterating the lumen. At this time there was approximately 80% occlusion of the vessel diameter judged by the color Doppler tracing, and a narrowed turbulent flow connecting shunt and CVC was visible intermittently.

The dog was represented eight months post procedure. It weighed 22kg and was bright and healthy. The owner considered the dog to be normal with much improved exercise tolerance. Ultrasonographic examination of the abdomen was normal; the liver was normal in size, shape and portal vasculature. The scan (Fig 5A and 5B) showed echogenic

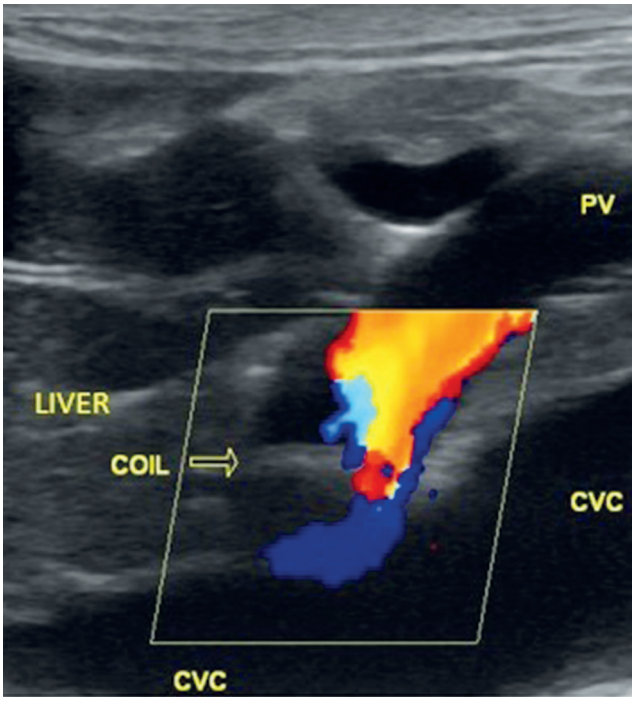

FIG 4B: Colour Doppler sonogram made immediately after coil deployment showing significant attenuation of the shunt with residual flow from the PV through the shunt into the CVC.

coil completely occluding the shunt extending through the liver parenchyma. There was no evidence of abnormal tissue reaction around the coil as it travels through the liver parenchyma. Color Doppler study demonstrated no flow through the shunt and the flow through the CVC was normal and non-turbulent. Flow through the portal vein at the level of porta hepatis was (15-20cm/sec).

DISCUSSION

Intrahepatic PSS are often difficult to identify, isolate and attenuate surgically and have been associated with complication rate as high as 77% (7,8).The use of minimally invasive endovascular attenuation of the intrahepatic PSS by mean of vena cava stent placement and insertion of thrombogenic coils within the shunt has been advocated in recent years. This approach is technically challenging and requires image-intensifying fluoroscopy, knowledge of endovascular procedures and expensive setup costs. A relatively expensive caval stent is used in an attempt to prevent coils from migrating away from the shunt (12) making the cost of the procedure a limiting factor. Additionally, endovascular approach may have multiple complications such as hemorrhage at the vascular

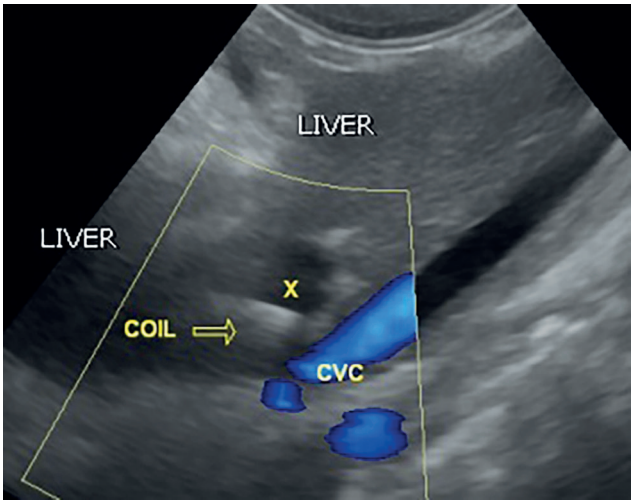

FIG 5A: Longitudinal transthoracic colour Doppler sonographic image of the right liver lobe taken 8 months post coli embolization of the intrahepatic shunt showing laminar normal flow through the CVC and no evidence of flow through the shunt. The coil is obliterating completely the lumen of the shunt.

access-site, stent migration, malpositioning and fracture. The coils may still embolise beyond the stent and into the pulmonary circulation (12). Furthermore the procedure requires the administration of multiple boluses of contrast which can be nephrotoxic at high dosages.

In our novel procedure we perform trans-hepatic, ultrasound guided controlled attenuation of the intra-hepatic shunt. Scanning directly on the liver produces clear images of the parenchyma and vasculature facilitating planning the best access rout to the ostium of the shunt and assessment of the flow through the shunt while it is being attenuated (13). Once the needle tip is within the lumen of the shunt the coil is being pushed gradually into the lumen by marked stylet facilitating a step by step assessment of the attenuated flow through the shunt using real-time color Doppler. The procedure was performed by laparotomy exposing the cranial abdominal viscera facilitating real time assessment of the color and motility of the intestines and mesenteric vasculature. Because we did not have the equipment to measure mesenteric venous pressure we had to rely on signs of intestinal hypermotility and increased mesenteric vascular pulsation to detect early onset of portal hypertension. Once the coil has been released into the liver parenchyma it appeared to be firmly lodged there, thus anchoring the intravascular portion of the coil and did not seem to trigger any reaction in the surrounding tissue. In those cases that require more than

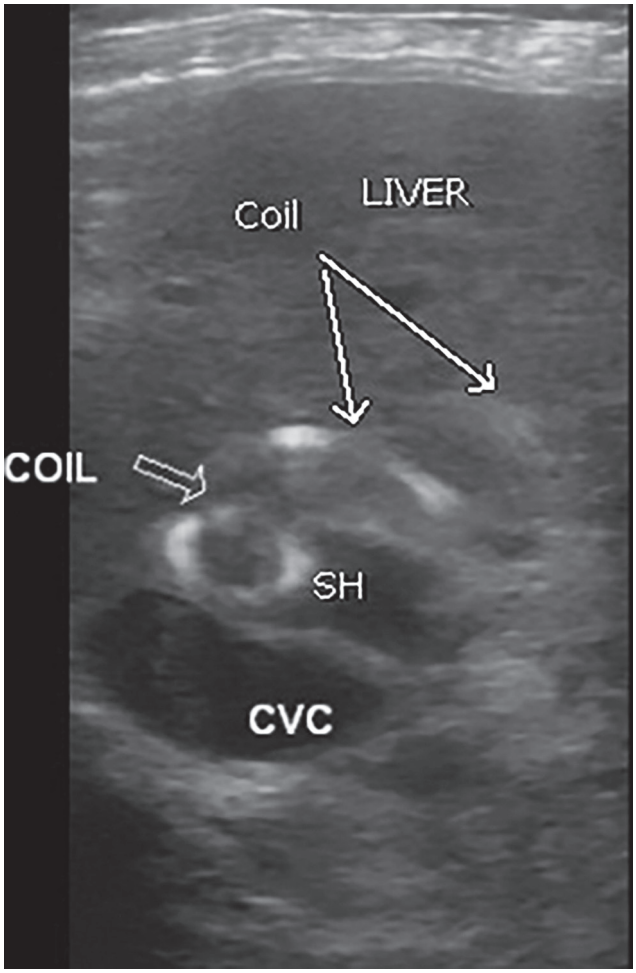

FIG 5B: Oblique transthoracic sonographic image selected to optimize the visibility of the coil taken 8 months post procedure. Note the isoechoic thrombus at the center of the coil completely occluding the shunt. The echogenic coil is also visible within the normalized liver parenchyma.

one coil to form satisfactory shunt attenuation one could deploy additional coils along-side the first coil in the same manner. Alternatively, judging by the degree of attenuation, additional coils of adequate sizes could be released into the shunt up-stream to the anchored first coil.

In our study we noted progressive closure of the shunt between 2 months and 8 months. We postulated that the coil which was lodged within the parenchyma and vessel wall triggered ongoing perivascular inflammation, fibroplasia and subsequent progressive perivascular thickening (14) leading to the eventual luminal closure. As a fail-safe mechanism in the course of the procedure, if significant portal hypertension is triggered it is still possible to retract the coil partially

or completely. We assume that such maneuver will lead to minimal damage to the parenchyma and vessel's wall.

The limitation of this technique is the need for an experienced ultrasonographer and a knowledge in needle guided procedures. Potential complications are similar to those associated with any other percutaneous transhepatic procedure and include transient hemobilia, bleeding (subcapsular hematoma), and infection. Complication more specific for coil embolization of intrahepatic shunt include the need for reembolization and portal hypertension.

In this first attempt we have performed the procedure via laparotomy opening, however it is possible that in some of the future cases where the shunt is clearly visible on the transabdominal ultrasound scan it may be possible to perform the procedure without performing laparotomy or just through a key hole opening.

CONFLICT OF INTEREST

None of the authors of this article has a financial or personal relationship with other people or administrations that could inappropriately influence or bias the content of this paper.

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