Transjugular coil embolization of an intrahepatic portosystemic shunt in a cat

Chick Weisse, VMD; Ken Schwartz, VMD; Rebecca Stronger, DVM; Jeffrey I. Mondschein, MD; Jeffrey A. Solomon, MD, MBA

Intrahepatic portosystemic shunts are rare vascular anomalies connecting the portal venous system to the systemic circulation; such shunts result in debilitating neurologic, biochemical, and hematologic dysfunction.

The prognosis following surgical treatment for cats with intrahepatic portosystemic shunts has not been determined; however, it is unlikely to be more favorable than the prognosis in dogs, for which high perioperative morbidity and mortality rates have been reported.

Interventional radiology techniques involving insertion of guidewires, catheters, and coils under fluoroscopic guidance can be safely used to attenuate anomalous vessels in small animals, potentially avoiding complications associated with invasive surgical techniques.

A 4-month-old male domestic shorthair cat was examined by the referring veterinarian because of a 1-month history of intermittent tremors, ptalism, and lethargy. Episodes varied in intensity from mild to severe, lasted from 3 to 24 hours, and recurred every few days. The owner had noticed these episodes immediately after adopting the cat, and reported that the cat often seemed hyperactive between episodes.

Abnormalities on physical examination included a small, thin stature and an unthrifty coat. Previous medical treatments included routine vaccinations and prophylactic deworming. Biochemical abnormalities at that time included low BUN concentration (13.5 mg/dL; reference range, 16 to 33 mg/dL), low amylase activity (362 U/L; reference range, 500 to 1,400 U/L), and high blood glucose concentration (178 mg/dL; reference range, 85 to 130 mg/dL). Hematologic abnormalities included low RBC count (9.65 × 10⁶/µL; reference range, 4.8 to 9.3 × 10⁶/µL), low mean corpuscular volume (41 fL; reference range, 58 to 79 fL), and low mean corpuscular hemoglobin concentration (14.4 g/dL; reference range, 19 to 28 g/dL). Baseline bile acids concentration was extremely high (142.5 µmol/L; reference range, 0 to 5 µmol/L). Abdominal radiographs were unremarkable, but results of abdominal ultrasonography were suggestive of a portosystemic shunt (PSS). The liver appeared normal ultrasonographically.

The cat was treated with a high-fiber, low-fat diet and lactulose (0.65 g, PO, q 12 h); however, clinical signs persisted. Two months later, therefore, a board-certified surgeon performed exploratory laparotomy in an attempt to ligate the PSS. At surgery, the liver was mottled tan-pink and of normal size and texture. No aberrant vessel was identified; however, jejunal portography revealed a PSS that appeared to be intrahepatic. Shunt attenuation was not attempted. A liver biopsy specimen was obtained, and the cat was castrated.

The cat recovered from anesthesia without complications and was discharged with instructions to feed a diet high in branched-chain amino acids and low in aromatic amino acids and to administer lactulose (1.3 g, PO, q 8 h) and metronidazole (50 mg, PO, q 24 h). Histologic examination of the liver biopsy specimen revealed mild portal venule hypoplasia, mild portal arteriolar hyperplasia, and mild arteriolization of central veins. Subsequent review of the biopsy specimen by another pathologist revealed moderate diffuse hepaticocellular degeneration.

At this time, the cat was referred to the Veterinary Hospital of the University of Pennsylvania. On initial examination, the cat was thin, small, and unthrifty and weighed 2.9 kg (6.4 lb). A grade 2 to 3 of 5 systolic heart murmur was ausculted; the heart rate was 220 beats/min, and the cat occasionally trembled. Laboratory abnormalities included a low Hct (28%; reference range, 31.7 to 48.0%), high aspartate transaminase (57 U/L; reference range, 1.0 to 37 U/L) and alkaline phosphatase (154 U/L; reference range, 22 to 87 U/L) activities, and high baseline bile acids (119.7 µmol/L) and ammonia (185 µg/mL; reference range, 11 to 35 µg/mL) concentrations. A cardiology examination, including echocardiography, revealed systolic anterior motion of the mitral valve with tall papillary muscles. The cardiologist could not rule out early hypertrophic cardiomyopathy and recommended administration of a β-adrenoceptor blocker at a low dosage if tachycardia persisted. On abdominal ultrasonography, the liver appeared to be of normal echogenicity and size with increased hepatic arterial blood flow. A probable intrahepatic PSS originating from the left branch of the portal vein and directly entering the caudal vena cava could be imaged.

The advantages and disadvantages of shunt attenuation via traditional surgical techniques versus interventional radiology techniques were discussed with the owner. The owner elected to have an intervention-
A radiology technique attempted because of the potentially lower morbidity and mortality rates and because such techniques are currently subsidized at our institution, resulting in reduced cost.

General anesthesia was induced with hydromorphone, diazepam, propofol, and glycopyrrolate and maintained with isoflurane in oxygen and etomidate. A balanced electrolyte solution with 2.5% dextrose was administered IV, and skin overlying the right jugular vein was aseptically prepared. A 4-F sheath introducer system was placed percutaneously into the right jugular vein. A 4-F catheter with a gentle bend on the tip was advanced into the caudal vena cava, and a guidewire was advanced through the catheter to the level of the shunt. Once shunt access was obtained, the catheter was advanced over the guidewire until it was located in the portal vein. Carbon dioxide venography was then performed to delineate the vascular anatomy (Fig 1). The catheter was then exchanged for an over-the-wire catheter with an 11-mm occlusion balloon at its tip. The balloon was slowly inflated to temporarily occlude the shunt, and angiography was performed by injecting contrast through the end hole of the balloon catheter to confirm complete temporary shunt occlusion. A water manometer connected to the balloon catheter was used to measure portal vein pressure before and after temporary occlusion. Portal vein pressure was 9 cm H₂O (6.6 mm Hg) before occlusion and 21 cm H₂O (15.4 mm Hg) afterward. With the balloon inflated, venography was performed to assess the intrahepatic portal vasculature; normal caliber and branching of the intrahepatic portal vein was seen (Fig 2).

The hemodynamic and angiographic studies indicated that the shunt could be completely occluded safely in a single procedure. The occlusion balloon catheter was removed over a 0.035-in Newton wire and replaced with a 4-F Berenstein catheter. Under fluoroscopy, the shunt was completely occluded by passing three 5-mm-diameter × 5-cm-long, 0.038-in stainless steel coils through the catheter (Fig 3). Small, short coils were chosen because of the small vessel size and because the neck of the shunt would prevent coil displacement. Venography was performed with the catheter tip in the shunting vessel; contrast could not be refluxed into the shunt, indicating that it was thrombosed.

The cat recovered from anesthesia without complications. The entire procedure, including angiography, pressure measurements, and coil embolization, lasted 2.75 hours. Analgesics were administered prophylactically overnight, but the cat appeared comfortable and was ambulatory, eating, and drinking the next morning.

Two days after the embolization procedure, abdominal ultrasonography was repeated. The region of the PSS was identified, and Doppler-flow ultrasonography confirmed that the vessel was completely occluded (Fig 4). Three days after the procedure, the cat was discharged from the hospital. The owner was instructed to administer lactulose (666 mg, PO, q 12 h), metronidazole (25 mg, PO, q 12 h), and atenolol (6.25 mg, PO, q 24 h) and to have the cat examined by the referring veterinarian in 4 weeks.

Over the next 5 weeks, the owner gradually decreased the dosages of metronidazole (25 mg, PO, q 24 h) and lactulose (333 mg, PO, q 24 h) and switched the cat to a maintenance type diet without any recurrence of neurologic signs. The cat's body weight increased from 2.90 to 3.63 kg (6.38 to 7.99 lb). Follow-up laboratory analyses revealed drastic improvements in baseline bile acids concentration (10.0 µmol/L) and post-prandial bile acids concentration (10.3 µmol/L; reference range, 1.0 to 20 µmol/L). Abdominal ultrasonography revealed a normal-sized liver and portal vein. The systolic heart murmur decreased to grade 1 of 5, and treatment with atenolol was continued.

Ten weeks after the embolization procedure, the owner reported that the cat was still doing well and had dramatically improved. Administration of lactulose and metronidazole had been discontinued 3 weeks earlier without any recurrence of neurologic signs.

Figure 1—Carbon dioxide venogram of a cat with an intrahepatic portosystemic shunt (PSS). A catheter inserted through the right jugular vein can be seen entering the portal vein through the shunt vessel (S). Notice reflux of contrast material into the right and left hepatic veins (HV), as well as filling of the right atrium (RA) and right ventricle (RV) with carbon dioxide. VC = Vena cava.
Portosystemic shunts are abnormal vascular communications between the portal venous system and the systemic circulation. Clinical signs associated with PSSs are the result of insufficient hepatic perfusion and diminished portal blood detoxification and include various degrees of neurologic, biochemical, and hematologic dysfunction. Although medical treatment is often useful for alleviating clinical signs, long-term medical management is rarely successful, and surgical treatment is widely accepted as the definitive treatment for this condition.1

Most congenital PSSs are extrahepatic in origin. Surgical treatment of single extrahepatic PSSs in dogs has been well described, is not technically demanding for experienced surgeons, has a reported success rate of 90 to 95%, and has been associated with low morbidity and mortality rates.1 Cats can have a good prognosis following complete PSS attenuation; however, the prognosis following surgical treatment of single extrahepatic PSSs in cats is generally less favorable than in dogs, especially when only partial attenuation is performed.2,4

Intrahepatic PSSs are less common than extrahepatic PSSs and pose a considerable technical challenge for veterinary surgeons. These anomalous vessels are often surrounded by substantial amounts of hepatic parenchyma, making shunt identification at surgery difficult and increasing the risk of hemorrhage during dissection. In addition, these shunts typically are quite large, increasing the likelihood that multiple procedures will be required for complete shunt attenuation.

Numerous surgical procedures for attenuation of intrahepatic PSSs in dogs have been described and range from careful dissection of the liver around the shunting vessel to complicated procedures involving temporary hepatic inflow occlusion and intravascular repair.2,4,7 However, all of these techniques are technically demanding, even for experienced surgeons, and all are associated with high perioperative morbidity and mortality rates.5-11 Reported mortality rates following surgical treatment of intrahepatic PSSs in dogs, for instance, range from 23 to 66%, with perioperative mortality rates ranging from 13 to 66%.6,7,11-13 The prognosis following surgical treatment of intrahepatic PSSs in cats has not been determined, but it is unlikely to be more favorable than in dogs.

The high morbidity and mortality rates associated with surgical treatment of intrahepatic PSSs has inspired a search for alternative, less-invasive treatment options. In humans, interventional radiology techniques employing catheters, guidewires, stents, and embolization coils have been used for similar procedures with good success and low morbidity rates.
Four recent reports in the veterinary literature have described the successful use of interventional radiology techniques for treatment of intrahepatic PSSs in dogs. Two of these were single case reports that described the successful use of thrombogenic coils for staged embolization of intrahepatic PSSs in dogs; both dogs recovered without any complications. The third discussed successful transvenous coil embolization of a patent ductus venosus in a miniature Dachshund. The fourth described the use of stents and thrombogenic coils for occlusion of intrahepatic PSSs in 10 dogs. To the authors’ knowledge, however, coil embolization of an intrahepatic PSS in a cat has not been reported previously.

The anatomy of the PSS in the cat described in the present report was ideal for coil embolization. The initial portogram confirmed perfusion of the liver via open portal vein branches, suggesting that complete shunt attenuation might be possible. In addition, the portal vein pressure after temporary balloon occlusion of the shunt was within limits considered acceptable for dogs (limits in cats have not been established), and the shunt tapered as it entered the caudal vena cava, minimizing the risk of coil displacement following deployment.

Interventional radiology techniques for attenuation of PSSs are minimally invasive, allow for simultaneous angiography and pressure measurements, and avoid potential complications associated with technically demanding surgical procedures. The authors’ hope is that such techniques will have lower perioperative mortality rates than those associated with traditional surgical techniques. Disadvantages of interventional radiology techniques include the possibility of coil migration into the heart or lungs and the equipment and technical expertise necessary to perform these procedures. Surgical and interventional radiology techniques both carry the risk of acute portal hypertension and the possibility that multiple procedures will be needed before complete shunt attenuation can be attained. Results of the present case support findings in previous reports that suggest that interventional radiology techniques are safe and effective for attenuation of intrahepatic PSSs in small animals.

Figure 3—Ventrodorsal radiographic view of the abdomen of the cat in Figure 1 immediately after placement of 3 stainless steel coils to occlude the PSS.

Figure 4—Color Doppler flow ultrasonogram of the cat in Figure 1 two days after transjugular coil embolization of the intrahepatic PSS. Notice that blood flows through the portal vein (PV), but that the shunt is completely occluded by the coils.
5-mm-diameter × 5-cm-long 0.038-inch stainless steel coils, Cook Inc, Bloomington, Ind.

Atenolol, Geneva Pharmaceuticals Inc, Broomfield, Colo.


References


