Use of endovascular stents in three dogs with Budd-Chiari syndrome

Michael D. Schlicksup, DVM; Chick W. Weisse, VMD, DACVS; Allyn C. Berent, DVM, DACVIM; Jeffrey A. Solomon, MD

Case Description—Three dogs were examined because of Budd-Chiari syndrome (BCS), which is an obstruction of venous blood flow located between the liver and the junction of the caudal vena cava and right atrium. Two dogs had confirmed neoplastic obstructions, and the other dog had a suspected neoplastic obstruction of the hepatic veins and caudal vena cava.

Clinical Findings—All dogs had similar clinical signs of weight gain, lethargy, and ascites that did not respond to medical treatments, and 2 dogs had pitting edema of the hind limbs. Ultrasonography revealed a presumptive venous obstruction, which was confirmed by use of computed tomography.

Treatment and Outcome—Each dog was anesthetized. By use of fluoroscopic guidance, endovascular stents were placed within the left hepatic vein and caudal vena cava in 2 dogs, and a single stent was placed within the left hepatic vein extending into the caudal vena cava of the third dog. After stent placement, venous pressure in the left hepatic vein decreased. Resolution of clinical signs was dramatic in all 3 dogs (survival time ranged from 7 to 20 months), with only mild complications in 1 dog.

Clinical Relevance—Endovascular stents may be an appropriate palliative treatment for dogs with clinical signs attributable to BCS. (J Am Vet Med Assoc 2009;235:544–550.)

A 10-year-old castrated male Boxer (dog 1) was evaluated at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania because of a distended abdomen, lethargy, and inappetence of 3 weeks’ duration. Medical history included a tibial fracture of the right hind limb approximately 3 weeks previously that had been placed in a splint by the referring veterinarian.

Physical examination revealed that dog 1 had a body weight of 48.6 kg (106.9 lb) and was tachycardic (160 beats/min) and tachypneic (60 breaths/min). The dog had profound abdominal distention, a palpable fluid wave, and pitting edema of the hind limbs.

Pertinent clinicopathologic findings included neutrophilia (18.4 × 10³ cells/µL; reference range, 3.1 × 10³ cells/µL to 14.4 × 10³ cells/µL), lymphopenia (0.2 × 10³ cells/µL; reference range, 0.9 × 10³ cells/µL to 5.5 × 10³ cells/µL), and an increase in the serum lactate concentration (3.7 mmol/L; reference range, < 2 mmol/L). Coagulation testing revealed a prolonged prothrombin time (10.5 seconds; reference range, 6.8 to 10.2 seconds) and an increase in the D-dimer concentration (2.08 µg/mL; reference range, < 0.2 µg/mL).

Abdominal ultrasonography revealed a large volume of anechoic fluid with no additional abnormalities. Evaluation of a fluid sample obtained during abdominocentesis revealed a total protein concentration of 4.0 g/dL, specific gravity of 1.026, 450 WBCs/µL, and 9,750 RBCs/µL. Aerobic bacterial culture of the peritoneal fluid yielded negative results.

Thoracic radiography identified widening of the caudal vena cava for both lateral views; however, the cardiac structures appeared normal, and there was no evidence of metastatic disease. Echocardiography revealed an intraluminal mass or thrombus within the caudal vena cava extending from the diaphragm to the level of the junction of the right atrium.

Examination of a helical CT angiogram revealed a contrast-enhancing lobulated soft tissue structure continuous with the pancreas and extending dorsally to the splenic vein on the left side. The thoracic portion of the caudal vena cava was large; during the venous phase, there was an intraluminal filling defect extending cranially from the hilus of the liver to immediately caudal to the right atrium. The hepatic veins were noticeably distended.

A diagnosis of BCS secondary to hepatic vein obstruction by the intraluminal mass in the caudal vena cava that resulted in suspected portal hypertension and massive ascites was made. In addition, the obstruction in the caudal vena cava resulted in edema of the hind limbs secondary to a reduction in venous drainage and an increase in hydrostatic pressure. The owner declined surgical treatment of the dog but consented to palliative treatment with stents to relieve the venous obstruction and improve the dog’s quality of life.

From the Veterinary Hospital, Department of Clinical Studies, School of Veterinary Medicine (Schlicksup, Weisse, Berent), and the Section of Vascular and Interventional Radiology, Department of Radiology, Hospital of the University of Pennsylvania (Solomon), University of Pennsylvania, Philadelphia, PA 19104.

The authors thank Drs. David C. Burgenernd and Wayne Hause for medical assistance with one of the dogs. Address correspondence to Dr. Schlicksup.

ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCS</td>
<td>Budd-Chiari syndrome</td>
</tr>
<tr>
<td>CaVC</td>
<td>Caudal vena cava</td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
</tbody>
</table>

JAVMA, Vol 235, No. 5, September 1, 2009
Dog 1 received hydromorphone (0.1 mg/kg [0.045 mg/lb]) as a premedication; midazolam (0.5 mg/kg [0.23 mg/lb]), lidocaine hydrochloride (2 mg/kg [0.9 mg/lb]), and etomidate (0.63 mg/kg [0.286 mg/lb]) were administered for anesthetic induction. Cisatracurium besylate (0.1 mg/kg) and isoflurane in oxygen were administered for maintenance of anesthesia. Cefoxitin (30 mg/kg [13.6 mg/lb], IV, once; followed by 15 mg/kg [6.8 mg/lb], IV, q 2 h) was administered during the perioperative period. Hair was clipped from the neck and the medial aspect of the right thigh. The dog was placed in dorsal recumbency with the neck and right hind limb extended to provide access to the jugular and femoral veins. Both sites were aseptically prepared. All manipulations of guide wires, catheters, and stents were performed by use of fluoroscopic guidance. An 18-gauge, 1.5-inch over-the-needle IV catheter was introduced percutaneously into the right jugular vein, and a 0.035-inch, angled, hydrophilic guide wire was introduced via the catheter and led through the right atrium into the CaVC. The catheter was replaced by a 12-F vascular introducer sheath and sutured in place with 2-0 nylon suture. A 5-F marker catheter and guide wire combination was advanced through the vascular sheath and into the CaVC distal to the intraluminal mass. A 4-F cobra catheter and guide wire were inserted through the same vascular introducer sheath and advanced into the left hepatic vein distal to the intraluminal mass. The cobra catheter and guide wire combination were manipulated into the left hepatic vein and distal to the intraluminal mass. Selective contrast venography revealed a distended left hepatic vein with multiple acquired venous drainage vessels and a large intravascular filling defect at the level of the diaphragm (Figure 1).

The right femoral vein in the proximal portion of the thigh was isolated via cutdown. An 18-gauge, 1.5-inch IV catheter was placed, and a 0.035-inch, angled, hydrophilic guide wire was advanced into the CaVC immediately caudal to the intraluminal mass. The catheter was replaced with a 9-F vascular introducer sheath that was secured to the skin with 2-0 nylon suture. A 4-F angled catheter was advanced over the guide wire into the CaVC, and the guide wire was then removed. Contrast angiography revealed correct placement of the catheter immediately caudal to the mass. The catheters were manipulated to facilitate measurement of pressures in the CaVC cranial (6.5 mm Hg) and caudal (17 mm Hg) to the mass and within the left hepatic vein (17 mm Hg). Contrast angiography was performed via simultaneous injection through all catheters to determine the precise location and extent of the mass in the CaVC and hepatic vein. The femoral vein catheter was replaced with a 7-F endomyocardial biopsy forceps, which was used to obtain samples of the mass for histologic examination. The biopsy forceps was removed and replaced with a 25-mm gooseneck snare. An exchange-length, 0.035-inch guide wire was advanced through the jugular marker catheter and CaVC and into the gooseneck snare. The snare was closed around the guide wire, and the combination was removed through the femoral sheath, which established through-and-through guide wire access from the jugular vein to the femoral vein.

Diameters of the left hepatic vein and CaVC were extrapolated from angiograms obtained with the marker catheter in place. Stent diameter was approximately 110% to 120% the diameter of the vessel in which it would be placed. Stent length was determined so that each stent would extend at least 1 cm beyond both the cranial and caudal aspects of the mass. A 10-F delivery system containing a 22 × 70-mm vascular stent was advanced over the exchange-length wire and through the femoral vascular sheath until it reached a point approximately 1 cm cranial to the intraluminal mass and immediately caudal to the right atrium. A 7-F delivery system containing a 14 × 60-mm vascular stent was advanced over the hepatic vein guide wire and through the jugular sheath to a point approximately 1 cm caudal to the mass in the left hepatic vein. Positioning of the delivery systems was confirmed via repeat angiography.

Assisted respiration was temporarily ceased during simultaneous deployment of both stents by use of constant fluoroscopic guidance. The delivery systems...
were removed over the guide wires. The CaVC blood pressures after placement of the stents were 8 mm Hg cranial to the mass, 11 mm Hg caudal to the mass, and 12 mm Hg within the left hepatic vein. A 12-mm x 4-cm percutaneous transluminal angioplasty balloon was then advanced within the stent in the CaVC. By use of fluoroscopic monitoring, the balloon was dilated with equal volumes of saline (0.9% NaCl) solution and contrast medium to expand the cranial and caudal aspects of the stent. Repeat angiography revealed a dramatic reduction in distention of the left hepatic vein, lack of filling of acquired venous drainage vessels, and improved flow through and reduction in diameter of the CaVC.

Blood pressures in the CaVC after dilation of the balloon were 9 mm Hg cranial to the mass, 11 mm Hg caudal to the mass, and 11 mm Hg within the left hepatic vein. Selective contrast angiography revealed dilation of the previously obstructed vessels (Figure 1). The femoral vascular sheath was removed, and the femoral vein was ligated. Total duration of anesthesia was 4 hours and 35 minutes, and total duration of surgery was 3 hours.

Dog 1 recovered without complications. There was a noticeable reduction in abdominal distention and complete resolution of edema in the hind limbs 1 day after surgery. On the second day after surgery, paresis of the hind limbs was evident as the dog became more ambulatory. Myelopathy of the T3–L3 region was suspected. Histologic evaluation of the intraluminal biopsy specimen revealed multiple pieces of a thrombus.

Dog 1 was discharged to the owners on day 2 after surgery. At the time of discharge, body weight of the dog was 45.6 kg (100.3 lb), which represented a decrease of 2.95 kg (6.5 lb). The dog was reexamined 12 days after surgery. The owners reported an increase in appetite and activity, and the body weight of the dog was 40 kg (88 lb), which represented a decrease of 8.5 kg (18.7 lb). Dog 1 had no clinical signs for approximately the next 7 months. At that time, the dog was brought to our facility because of acute abdominal distention and edema of the hind limbs. Body weight at that time was 46.9 kg (103.2 lb), which was a decrease of only 1.7 kg (3.7 lb) since the initial examination. The owners elected for euthanasia of the dog.

Necropsy revealed 3.4 L of translucent fluid within the abdomen. A 10 x 6 x 5-cm firm nodular mass was adjacent to the pancreas and extended cranially to the liver; a portion of the mass invaded the portal vein. A second mass (6 x 5 x 4 cm), which appeared identical in appearance to the first mass, was adhered to the ventral surface of the diaphragm adjacent to and invading the stent-containing left hepatic vein and CaVC. A third mass (1.3 cm in diameter) was adjacent to the proximal aspect of the pulmonary artery, and a fourth smaller mass was on the ventral aspect of the trachea at the level of the heart base. Histologic examination of all 4 masses revealed a paraganglioma. Additionally, metastatic paragangliomas were found in multiple liver nodules and a single lung lobe. The metallic stents appeared patent and in place within the CaVC and left hepatic vein. The liver appeared grossly congested and represented 3.2% of the body weight of the dog; the liver had evidence of fibrosis, which possibly caused the recurring ascites.

A 6-year-old castrated male Labrador Retriever (dog 2) was referred to our veterinary teaching hospital for diagnostic testing and treatment because of a suspected mass within the CaVC. The dog had a 2-month history of weight gain and increasing lethargy. Abdominal ultrasonography performed by the referring veterinarian revealed marked peritoneal effusion. Thoracic radiography revealed mild pleural effusion with a suspected distended CaVC in the caudal portion of the thorax. Echocardiography and abdominal ultrasonography revealed a 2.6-cm-diameter soft tissue mass within the intrahepatic CaVC that extended into the left hepatic vein and immediately caudal to the right atrium.

Examination at our veterinary medical teaching hospital revealed that the body weight of the dog was 38.8 kg (85.4 lb). Dog 2 had a distended abdomen with a palpable fluid wave and was tachypneic (70 breaths/min). Pertinent clinicopathologic abnormalities included thrombocytopenia (474 x 10^3 platelets/µL; reference range, 177 x 10^3 to 398 x 10^3 platelets/µL), a low alanine aminotransferase activity (13 U/L; reference range, 16 to 91 U/L), and a high γ-glutamyltransferase activity (75 U/L; reference range, 7 to 24 U/L). Coagulation testing revealed an increase in the D-dimer concentration (0.5 to 1.0 µg/mL). Evaluation of a fluid sample of peritoneal fluid revealed a total protein concentration of 4.1 g/dL, specific gravity of 1.027, 510 WBCs/µL, and < 200,000 RBCs/µL.

A 3-cm-diameter soft tissue mass within or overlying the CaVC in the caudal portion of the thorax was detected during thoracic radiography. Echocardiography revealed a large hyperechoic mass caudal to the atrium that extended caudally to the diaphragm, with no evidence of right-side heart failure. A helical CT with dual-phase angiography revealed a 5-cm-diameter soft tissue mass between the right caudal and accessory lung lobes that extended through the CaVC into the hepatic veins. Cranial to the mass, dimensions of the CaVC were 17 x 15 mm, whereas caudal to the mass, CaVC dimensions were 23 x 17 mm. Portions of the mass were mineralized. The owners agreed to palliative treatment with stents to alleviate clinical signs associated with the neoplastic vascular obstruction.

Anesthesia was induced in dog 2 by IV administration of hydromorphone (0.08 mg/kg [0.036 mg/lb]), midazolam (0.25 mg/kg [0.114 mg/lb]), lidocaine (1.5 mg/kg [0.68 mg/lb]), and propofol (2 mg/kg). Isoflurane in oxygen was administered for maintenance of anesthesia. A surgical procedure similar to that in dog 1 was performed; however, it required 2 additional hours of anesthesia. Pressures before insertion of the stents were measured in the CaVC cranial to the mass (11 mm Hg) and within the left hepatic vein (17 mm Hg). A 16 x 60-mm vascular stent was placed in the left hepatic vein via an introducer in the right jugular vein, and a second 16 x 60-mm vascular stent was placed in the thoracic or cranial abdominal portion of the CaVC via an introducer in the right femoral vein. A brief episode of atrial flutter was recorded during placement of the CaVC stent, which resolved immediately and did not necessitate additional treatment. Selective contrast angiography revealed improved patency of the previously obstructed vessels. After placement of the stents, pressure in the
Dog 2 continued to improve at home. Approximately 1 year later, the dog was examined at another referral institution to determine the cause of lethargy and anorexia. Computed tomography revealed minimal peritoneal effusion and unchanged location and patency of both stents. The appearance of the soft tissue mass was similar to that in the previous CT. Aspirates obtained at the level of the liver revealed an undifferentiated sarcoma. Chemotherapy was instituted, and the dog received 2 doses of doxorubicin (22.6 mg/m²) during the subsequent 2 months. Results of follow-up thoracic radiography were suggestive that the mass may have involved the heart. The chemotherapeutic plan was changed to carboplatin (260 mg/m²). Two months later (approx 17 months after insertion of the vascular stents), abdominal distention returned and the dog was increasingly lethargic. Approximately 200 mL of fluid was removed from the abdomen. However, this did not result in any improvement in the dog’s condition, and the owners elected for euthanasia of the dog. Body weight of dog 2 at the time of euthanasia was 35 kg (77 lb), which was 3.8 kg (8.4 lb) less than at the initial examination. Necropsy was not performed.

A 12-year-old castrated male English Springer Spaniel (dog 3) was evaluated at our veterinary medical teaching hospital to determine the cause of recurrent ascites and a possible mass or thrombus within the CaVC. History included lethargy and weakness of 6 months’ duration. More recently, substantial abdominal distention and loose feces were evident. Initial examination by the referring veterinarian revealed abdominal distention, a palpable fluid wave, and loss of detail on abdominal radiography. Abdominal ultrasonography by the referring veterinarian identified peritoneal effusion and a suspected mass or thrombus within the CaVC at the level of the right adrenal gland. No morphologic or functional cardiac abnormalities were detected during echocardiography performed by the referring veterinarian, and therapeutic abdominocentesis with removal of an unknown amount of fluid was performed.

Physical examination at the time of admittance to our veterinary medical teaching hospital revealed persistent abdominal distention, considerable bilateral muscle atrophy in the shoulder region, edema of the right forelimb and both hind limbs, and abdominal bruising. Body weight was 25.7 kg (56.5 lb). Results of abdominal ultrasonography included a large left adrenal gland (width, 2.7 cm) that extended to the level of the CaVC. The caudal pole of the right adrenal gland appeared normal; however, the cranial pole could not be identified. A suspected mass was detected within the CaVC extending from the level of the left kidney to the diaphragm; the mass was 3.6 cm at its widest point. Thoracic radiography revealed normal cardiac structures with no evidence of metastatic disease. Pertinent clinicopathologic abnormalities included hypocalcemia (9.4 mg/dL; reference range, 9.8 to 11.7 mg/dL), hypoproteinemia (4.6 g/dL; reference range, 5.4 to 7.1 g/dL), and hypoglobulinemia (2.2 g/dL; reference range, 2.4 to 4.0 g/dL). Coagulation testing revealed a prolonged prothrombin time (10.9 seconds) and high D-dimer concentration (0.42 pg/mL). Examination of a fluid sample obtained during abdominocentesis revealed a total protein concentration of 3.5 g/dL, specific gravity of 1.025, 1,660 WBCs/µL, and 190,000 RBCs/µL. The owners declined an ACTH stimulation test.

Helical CT with dual-phase angiography of the abdomen revealed a mass in the left adrenal gland that invaded the CaVC and extended 24 cm toward the right atrium. Numerous heterogeneous enhanced hepatic nodules were also evident.

Preamesthesia medication of dog 3 included IV administration of hydromorphone (0.2 mg/kg [0.09 mg/lb]), midazolam (0.25 mg/kg), and glycopyrrolate (0.01 mg/kg [0.0045 mg/lb]). Anesthesia was induced by IV administration of lidocaine (1.6 mg/kg [0.73 mg/lb]) and propofol (1.6 mg/kg). Anesthesia was maintained by administration of cisatracurium (0.1 mg/kg, IV) and isoflurane in oxygen.

A surgical procedure similar to that described for dogs 1 and 2 was performed. Pressure within the left hepatic vein was 18 mm Hg. During catheter manipulation, dog 3 had a short-term spike in mean blood pressure (from 60 to 150 mm Hg) and heart rate (from 115 to 165 beats/min). The increase in blood pressure resolved once manipulation of the vessel was temporarily discontinued; however, the heart rate remained high (130 to 150 beats/min). A 7 X 80-mm self-expanding, laser-cut, nitinol stent was inserted within the left hepatic vein and CaVC via an introducer in the right jugular vein. Following insertion of the stent, the dog had a second prolonged increase in mean blood pressure. A continuous rate infusion of sodium nitroprusside was initiated; the infusion was continued throughout the remainder of anesthesia. Selective contrast angiography revealed patency of the previously obstructed vessel. Pressure within the left hepatic vein after insertion of the stent was 8 mm Hg. Postoperative thoracic radiography confirmed correct placement of the stent from the left hepatic vein to the thoracic portion of the CaVC. Total duration of anesthesia was 3 hours and 15 minutes, and total duration of surgery was 1 hour and 30 minutes.

Dog 3 recovered without complications and was discharged to the owner 1 day after surgery. Phenoxybenzamine (10 mg, PO, q 12 h) was prescribed. Body weight at the time of discharge was 23.5 kg (51.7 lb), which represented a decrease of 2.2 kg (4.8 lb).

Ten days later, the owner reported that dog 3 had an increase in energy, a decrease in polydypsia, but a persistence of polyuria. Physical examination revealed mild abdominal distention, resolution of the edema in the right forelimb and both hind limbs, and a body weight of 24 kg (52.8 lb), a decrease of 1.7 kg (3.7 lb) from the body weight at the initial examination at our facility. Repeat abdominal ultrasonography revealed mild peritoneal effusion; the stent was in position and patent.

Dog 3 was returned to our facility 1 month later because of mild progression of the abdominal distention and an increase in the polydypsia-polyuria. Abdominal

Unauthenticated | Downloaded 01/10/24 12:24 AM UTC
ultrasonography and echocardiography revealed patency of the stent and no obvious increase in the size of the mass. It was believed that the abdominal ascites had increased since the preceding examination; however, the amount of fluid was still less than that identified at the initial examination. Administration of phenoxybenzamine was continued. A follow-up telephone consult 6 months later revealed that the dog was clinically normal with a weight similar to that following stent placement.

Thirteen months after stent placement, dog 3 was evaluated by personnel in the Emergency Service at our veterinary medical teaching hospital because of ataxia of the hind limbs. The owners reported no changes in the dog’s drinking or urinating patterns since the preceding examination. The dog had continued to receive phenoxybenzamine since the time of the surgical procedure. Physical examination revealed nystagmus (fast phase to the right), ventral strabismus of the right eye, and a large amount of black exudate within both the horizontal and vertical ear canals that completely obscured the tympanic membranes. A diagnosis of peripheral vestibular disease secondary to suspected otitis media-interna was made on the basis of the combination of neurologic and otic clinical signs. Dog 3 was treated empirically. Abdominal radiography revealed normal serosal detail and an unchanged position of the stent. Abdominal ultrasonography revealed that the mass in the left adrenal gland was subjectively larger and continued to invade the vena cava, and the extent of the caval invasion was dramatically decreased. No peritoneal effusion was detected. Echocardiography confirmed that the mass no longer extended to the level of the right atrium (as during the initial examination); instead, it extended only to the level of the diaphragm. Changes in extent of the tumor identified during ultrasonography and echocardiography were presumed to result from a reduction in the associated thrombus around the tumor. Blood flow within the stent could not be confirmed because of artifact caused by the stent and the dog’s respirations. Caval blood flow appeared laminar, and velocity of caval blood flow was 1 m/s (a decrease from 2 m/s at our initial examination). Dog 3 remained without clinical signs at 20 months after stent placement.

Discussion

Budd-Chiari syndrome is an obstruction of venous blood flow located between the liver and the junction of the CaVC and right atrium. The condition was first described by Budd in 1845 and further characterized by Chiari in 1899, and it belongs to a triad of diseases currently known as hepatic venous outflow obstructions. Veno-occlusive disease, characterized as obstruction at the level of the sinusoids and terminal venules, and congestive hepatopathy, defined as venous obstruction at the level of the heart, are the other 2 hepatic venous outflow obstructions that are sometimes mistaken for BCS. The original condition described by Budd and Chiari were primarily obstructions of the hepatic veins; however, most of their patients also had some degree of obstruction of the CaVC. Discrepancies have remained as to whether obstructions solely of the CaVC are BCS-like obstructions or actual BCS. An important distinction is with regard to whether the obstruction is in the hepatic portion of the CaVC (thus not causing obstruction of the hepatic outflow) or more cranial (thus causing obstruction of hepatic outflow). Lesions within the hepatic portion of the CaVC of humans have different causes, clinical signs, and treatments and therefore have been considered a BCS-like disease. Confusion within the human literature has led to more recent classifications and the development of the aforementioned categories for hepatic venous outflow obstruction.

Nonneoplastic causes of BCS or BCS-like syndromes include traumatic kinking of the CaVC within the thorax, numerous vascular abnormalities in a 12-week-old puppy with a stenotic lesion of the CaVC attributable to an unknown cause, and a cat with a web-like lesion in the CaVC that failed to respond to balloon dilatation and subsequently required placement of a stent. In humans in the western hemisphere, BCS commonly develops subsequent to thrombosis and hypercoagulable states. Although none of the 3 dogs described here appeared to develop BCS primarily as a result of thrombosis, all did have high D-dimer concentrations, and there was likely to be thrombosis around the tumors. The extent to which thrombosis contributed to the clinical signs is unclear; however, the presence of local thrombus was supported by examination of the endovascular biopsy specimen obtained from dog 1, which revealed a thrombus or blood clot, but the underlying cause was ultimately determined to be neoplasia. In the veterinary literature, there are more reports of neoplastic-associated BCS or BCS-like disease than nonneoplastic causes. These have included unilateral renal carcinoma, esophageal leiomyoma, thoracic wall chondrosarcoma, and adrenal gland neoplasia. All 3 dogs described here had underlying or suspected neoplastic causes of BCS.

Clinical signs are dependent on the location, completeness, and chronicity of the vascular obstruction. When limited to the hepatic veins, posthepatic sinusoidal venous hypertension can result in hepatomegaly, portal hypertension, abdominal pain, and ascites. Lesions that also obstruct the CaVC can cause dilation of superficial distal (caudal) veins with concurrent edema of the distal portions of the extremities (eg, scrotum, prepuce, and pelvic limbs). Veterinary patients with BCS caused by malignancies have clinical signs (including ascites, hepatomegaly, and abdominal pain) similar to those in humans. Dogs with CaVC obstructions caudal to the hepatic veins have edema of the hind limbs without concurrent ascites. Two of the dogs described here had clinical signs consistent with obstruction of the hepatic veins and CaVC (ie, ascites and edema of the hind limbs). Dis-
transudate. This is a characteristic of hepatic obstructive disease, and a low-cellularity, high-protein abdominal effusion should alert clinicians to the possibility of a postsinusoidal obstruction. The modified transudate is caused by the increased hydrostatic pressure resulting in fluid extravasation into the abdominal cavity and protein leakage through the liver capsule.12

Medical treatment should be directed at the underlying cause, and clinicians should reduce the ascites via the use of diuretics and repeated abdominocentesis, when necessary. Medical treatment can be palliative, but it is generally considered ineffective as a long-term solution. Treatment for neoplastic conditions in humans consists of surgery13, radiation15 or chemotherapy16 (or both) when applicable, and placement of endovascular stents for palliation of clinical signs.17 In 1986, investigators published the first report18 of placement of a metallic stent in the CaVC of 7 dogs with experimentally created stenoses. Four dogs had successful dilation and hemodynamic improvement for the follow-up period of 4 months. In the other 3 dogs, the stent failed to dilate the vena cava, which subsequently resulted in complete occlusion. Placement of endovascular stents to restore venous blood flow associated with malignancies of the CaVC was first reported in humans in 1992.19 To the authors’ knowledge, the 3 dogs described here represent the first report of palliative placement of endovascular stents for malignancy-induced BCS in clinically affected animals.

Palliative placement of stents requires fluoroscopy and a thorough knowledge of vascular anatomy and endovascular instrumentation. Numerous vascular stents are available for use in the central venous system. Endovascular stents were used in dogs 1 and 2 because of their ability to be reconstrained and repositioned (if necessary) before completion of deployment. The foreshortening during deployment for self-expanding, mesh, stainless-steel stents is a recognized drawback, but this was not a problem in either of the 2 dogs in which they were used. In dogs 1 and 2, stents were placed simultaneously in the left hepatic vein and CaVC and allowed to engage at the vascular confluence, thus providing patency of both vessels.20 In dog 3, a self-expanding, laser-cut, nitinol stent was chosen for its ability to be placed precisely with a predetermined length independent of its deployed diameter (ie, no foreshortening).

Size of stent remains an area of controversy in human medicine. In the dogs reported here, size of the normal adjacent CaVC and hepatic veins was extrapolated after adjusting for radiographic magnification of a marker catheter placed within the vessel. Once the vessel diameter was determined, an increase of 10% to 20% was added to the stent size to achieve sufficient dilation and wall apposition, thus preventing stent migration. In humans, vascular stents typically are 100% to 120% of the diameter of the original, healthy vessel.17,20,21

If stent placement does not achieve complete expansion because of compression by a neoplasm or thrombus, balloon dilatation of the stent can be performed. This was used in only 1 dog described here because the stents achieved sufficient expansion and patency in the other 2 dogs. Human patients often receive anticoagulants perioperatively because BCS commonly develops secondary to thrombosis or hypercoagulable states.22 None of the 3 dogs in our report received anticoagulants because thrombosis was not considered a major contributor to their disease conditions at the time of evaluation. However, in light of the final results during reexamination of dog 3, it would appear unlikely that the mass within the CaVC actually regressed in size, and it is more plausible that some degree of the initial vascular obstruction was further complicated by tumor-associated thrombosis that had resolved.

The left hepatic vein was chosen for placement of a stent because it is the largest of the hepatic veins in dogs. The left hepatic vein is also appropriately suited for placement of a stent in the location where it makes a gentle curve as it enters the CaVC; thus, kinking of the stent would be unlikely and stent placement would likely be uncomplicated. No additional hepatic veins received a stent because it has been reported23 that humans with BCS have complete resolution of clinical signs after placement of a stent in 1 hepatic vein when all 3 major hepatic veins are obstructed. Additionally, a single patent hepatic vein was sufficient to prevent the development of ascites in dogs with experimentally induced obstruction.24 Two dogs each received 2 stents (1 stent in the left hepatic vein and 1 stent in the CaVC); however, the third dog only received a stent in the left hepatic vein because of the cardiac arrhythmias and changes in blood pressure during manipulations to structures around the tumor.

Success in patients that receive endovascular stents has been measured by resolution of clinical signs, angiographic resolution of the obstruction, continued patency of the stent or stents, and a reduction in pressure gradients. No ideal reduction in pressure gradient or pressure threshold has been defined; however, a study25 of 23 humans with BCS treated with endovascular stents revealed a mean pressure decrease within the hepatic vein from 25.57 to 9.67 mm Hg. In dog 1, pressure within the left hepatic vein decreased from 17 to 12 mm Hg and then to 11 mm Hg following balloon dilatation. In dogs 2 and 3, a pressure decrease in the hepatic vein from 17 to 12 mm Hg and from 18 to 8 mm Hg, respectively, was recorded. Patency rates measured ultrasonographically in 120 human patients receiving stents in the CaVC because of malignancies were 100% at 2 months, 90% at 6 months, and 80% at 24 months.20 In dogs 1 and 3 reported here, the stents were ultrasonographically patent 5 months and 3 months after surgery, respectively. In dog 2, a CT confirmed patency of the stent 12 months after surgery. Clinical resolution in human patients with BCS is quick and dramatic following stent placement,21,23,25 similar to the situation for all 3 dogs reported here.

Complications associated with placement of endovascular stents are usually minor and transitory in duration, making it a viable treatment option for compromised patients. Complications in humans include fracture, migration, reocclusion, and misplacement of stents.24 Additional complications include non–life-threatening hemorrhage from the vascular access site and procedure-related arrhythmias.25 Two of the dogs reported here had cardiac arrhythmias, only 1 of which required treatment, that were likely attributable to an underly-
ing suspected pheochromocytoma. Stents placed in the jugular vein, cranial vena cava, CaVC, and abdominal aorta of dogs are reportedly incorporated into the tunica intima of the vessel by day 7 after placement, at which time stent migration would be improbable. Prior to that time, stent migration is possible but unlikely if the stent is the appropriate size. 26

Historically, the stent with the shortest length was recommended for use to decrease the possibility of thrombus formation. More recent theories suggest that thrombus formation is less likely caused by metallic load and more likely as a result of turbulence through the stent and defects in the stent during placement (ie, improper size, insufficient expansion, or misshapen stents). 27,28 Concerns remain about stents traversing the ostia of major vessels within the CaVC. In humans, endovascular stents have been placed in the renal and iliac veins without complications. 29,30

Potential benefits of palliative placement of stents, compared with benefits of surgical removal of vascular obstructions, include a theoretic reduction in anesthetic duration, minimal invasiveness, few and predominantly minor complications, and often dramatic improvement in clinical signs. In light of the results reported here, the authors propose that placement of venous stents can be an effective treatment option for management of clinical signs associated with neoplastic BCS in dogs when surgical removal is declined, not indicated, or associated with excessive risk or morbidity.

e. Wallstent endovascular stent, Boston Scientific, Natick, Mass.

References