Evaluation of cellophane banding with and without intraoperative attenuation for treatment of congenital extrahepatic portosystemic shunts in dogs

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Objective—To evaluate the effect of intraoperative attenuation of congenital extrahepatic portosystemic shunts (CEPSSs) during cellophane banding procedures in dogs.

Study Design—Retrospective case series and prospective study.

Animals—18 cases evaluated retrospectively and 14 dogs evaluated prospectively.

Procedures—Gradual occlusion of CEPSSs was performed via cellophane banding. Shunts were occluded to a diameter <3.0 mm during surgery in dogs prospectively enrolled in the partial attenuation group, whereas the shunt was not attenuated during surgery in dogs prospectively enrolled in the no-attenuation group or in dogs that had previously undergone surgery and were retrospectively evaluated. Postprandial serum bile acids (PPBSA) concentrations were measured before surgery and at various time points after surgery.

Results—Mean ± SD PPBSA concentrations were 26.8 ± 24.5 µmol/L at <2.25 months after surgery (n = 16 dogs), 22.1 ± 14.0 µmol/L from 2.25 to 6 months after surgery (12 dogs), and 34.9 ± 32.5 µmol/L at >6 months after surgery (22 dogs). In the prospectively enrolled dogs, mean PPBSA concentrations increased over time in dogs in the partial attenuation group, but not in dogs in the no-attenuation group.

Conclusions and Clinical Relevance—Cellophane banding may be used to occlude larger CEPSSs and may decrease the need for intraoperative monitoring of portal vein blood pressure. The technique may facilitate minimally invasive treatment of CEPSSs in dogs. Intraoperative attenuation of CEPSSs to a diameter <3.0 mm is not necessary and may result in a less favorable outcome. (J Am Vet Med Assoc 2006;228:1355–1360)

Postprandial serum bile acids

Portosystemic shunts are anomalous vessels that join the portal and systemic venous circulations, allowing blood from splanchnic organs to bypass the liver. In small-breed dogs, approximately 80% of shunts are CEPSSs. Fatal liver failure develops by 3 years of age in most dogs that receive only medical treatment, whereas surgical attenuation of shunts is associated with a more favorable long-term prognosis. In 1 study, 100% of patients that underwent complete surgical lig-

Abbreviations

CEPSS Congenital extrahepatic portosystemic shunt
PSS Portosystemic shunt
rNA Dogs retrospectively evaluated that underwent a cellophane banding procedure for CEPSS in which the shunt diameter was not intraoperatively attenuated
pPA Dogs prospectively enrolled that underwent a cellophane banding procedure in which the shunt diameter was partially attenuated
pNA Dogs prospectively enrolled that underwent a cellophane banding procedure in which the shunt diameter was not intraoperatively attenuated
PPBSA Postprandial serum bile acids
occlusion of encircled vessels. In an experimental study in dogs, cellophane bands caused more gradual attenuation of the femoral veins than did amiodar constrictors. In a clinical study, 8 of 8 dogs with CEPSS treated by means of intraoperative cellophane band attenuation to 2.5 mm (vacular diameter) had complete occlusion within 8 weeks, whereas 2 of 3 dogs that underwent cellophane band attenuation to 3.0 mm had delayed closure, results that were determined on the basis of biochemical analyses, Doppler ultrasonography, or both. Investigators in that study concluded that 3.0 mm is the maximum internal diameter at which shunts can be left and still yield complete vascular closure in affected dogs.

The objectives of the present study were to evaluate use of cellophane bands for treatment of CEPSS in dogs and compare outcomes for dogs in which CEPSS was treated with cellophane bands without intraoperative attenuation and those treated with cellophane bands with partial intraoperative attenuation. We hypothesized that there would be no difference in outcome when CEPSS was treated with cellophane bands with and without partial attenuation.

Materials and Methods

Medical records of dogs with a diagnosis of CEPSS and that underwent a cellophane banding procedure at the Colorado State University Veterinary Teaching Hospital were reviewed. From February 2001 to March 2002, dogs that underwent surgical treatment for CEPSS at this institution were treated with cellophane banding without intraoperative shunt attenuation. Those records were reviewed retrospectively, and that group of dogs was designated as RNA. From April 2002 through September 2003, dogs were prospectively and randomly assigned to either the partial attenuation group or the no attenuation group. In the pPA group, the shunt was attenuated to a diameter < 3 mm during surgery.

The diagnosis of CEPSS in all study dogs was made on the basis of findings from the medical history, hematologic and serum biochemical analyses, abdominal ultrasonography, technetium-99 transcolonic nuclear scintigraphic imaging, and portal angiography. Signalment (eg, breed, age at the time of surgery, sex, and body weight) and preoperative hematologic and serum biochemical values were recorded.

After induction of general anesthesia, ventral midline celiotomy and complete abdominal exploration were performed. For dogs in the pPA and pNA groups, the CEPSS was identified and a 22-gauge, over-the-needle catheter was placed in a jejunal vein and connected to a water manometer with the base positioned at the level of the heart. Baseline portal venous pressure was recorded. The CEPSS was isolated by means of blunt tissue dissection and the vascular diameter was measured with sterile calipers. A sheet of sterile cellophane was folded and cut to form a 3-layered band with a width of 4.0 mm. The cellophane band was placed around the catheter and the CEPSS such that the shunt was completely collapsed. The catheter was removed, permitting reexpansion of the CEPSS. After placement of the cellophane band, splanchic organs were observed for 5 minutes. Portal venous pressure and CEPSS diameter at the level of the cellophane band were recorded.

For dogs in the pNA treatment group, the cellophane band was placed in complete contact around the CEPSS and loosely secured with titanium clips without any occlusion. Following placement of the band, splanchic organs were observed for 5 minutes, and portal venous pressure and CEPSS diameter at the level of the cellophane band were recorded.

Dogs with portal vein atresia were excluded from the study. Dogs in the pPA group were excluded if one of the following conditions was noticed at the time of intraoperative partial attenuation: postattenuation portal venous pressure > 17 cm H2O and greater than baseline portal venous pressure plus 10 cm H2O; variation of central venous pressure > 1 cm H2O, and evidence of splanchic cyanosis or congestion.

For dogs in the RNA group, cellophane bands were placed in a similar manner as in dogs in the pNA group; however, portal venous pressure and shunt diameter measurements were not performed. Liver biopsy and routine abdominal closure were performed in all dogs. Ovariohysterectomy, castration, and cystotomy for removal of urinary calculi were performed as indicated in certain dogs.

Follow-up evaluations—Serial postprandial serum bile acids concentrations were obtained. Postprandial bile acids concentrations were categorized temporally from the time of surgery into 3 groups according to a described interval as follows: < 2.25 months, from 2.25 to 6 months, and longer than 6 months.

Serial follow-up examinations or telephone conversations with owners and referring veterinarians were performed, and complications were recorded. Follow-up questions pertained to growth, behavior, neurologic status, vomiting, diarrhea, inappetence, polyuria, polydipsia, dysuria, diet, medications, and recurrence of the clinical signs that had been responsible for the dog’s initial evaluation at the teaching hospital. Dogs were assigned a clinical grade on the basis of long-term follow-up findings and by use of a modified rating scale (Appendix). Long-term follow-up was defined as a follow-up time > 6 months after surgery. The clinical rating scale included a grade of 1.5 for patients that were habitually fed low-protein diets and were never offered a diet formulated for clinically normal dogs after surgery.

Statistical analysis—All analyses were performed with statistical software. Analysis of variance was used to compare the pPA and pNA treatment groups with respect to continuous data. Analysis was used to compare incidence of complications, mortality rate, and rate of postligation neurologic syndrome among dogs in the pPA, pNA, and RNA groups. Analysis of variance for repeated measurements was used to evaluate the effects of treatment (ie, pPA vs pNA), time, and time-treatment interaction on postprandial serum bile acids concentration. Significance was set at a value of P < 0.05.

Data were expressed as mean ± SD.

Results

Thirty-two dogs with CEPSS were treated by means of cellophane banding. Fourteen dogs were male and 18 were female; 1 of the male dogs was cryptorchid. Mean age at the time of surgery was 20.4 ± 24.6 months. Breeds represented included Yorkshire Terrier (n = 9), Miniature Schnauzer (5), Dachshund (3), other purebred dogs (14), and mixed-breed dogs (1). Mean weight was 5.4 ± 6.40 kg (11.9 ± 14.1 lb). Thirty (93.8%) dogs weighed < 10.0 kg (22 lb).

Eighteen dogs were retrospectively evaluated (ie, included in the RNA group), whereas 8 dogs were included in the pPA treatment group and 6 dogs were included in the pNA treatment group. All dogs assigned to the pPA
ventricular contractions were detected preoperatively in a 12-month-old neutered male Standard Poodle (weight, 6.9 kg [14.3 lb]) with complications that resulted in death or euthanasia. A prolonged recovery time from a previous anesthetic episode, and 3 had recurrent urinary tract infections. Sixteen (50.0%) dogs were fed a low-protein diet before surgery and 16 received medications, including antimicrobials, lactulose, or both.

Preoperative hematologic and serum biochemical values were consistent with the diagnosis of PSS. Abdominal ultrasonography was performed at the teaching hospital in 21 dogs. The diagnosis of CEPSS was confirmed ultrasonographically in 14 of the 21 (66.7%) dogs, and cystic calculi were observed in 3 (14.3%) dogs. All cystic calculi were composed of ammonium biurate. Transcolonic nuclear scintigraphy was performed preoperatively in 9 dogs, and the mean shunt fraction was 71.9 ± 10.7% (range, 60.0% to 85.0%).

Twenty-three dogs had portocaval CEPSSs, and 9 had portoazygous CEPSSs. Intraoperative portal venography was performed to identify 1 portocaval and 1 portoazygous CEPSS. Ovariohysterectomy was performed in 7 dogs, castration in 3, and cystotomy in 4. No intraoperative complications were encountered.

Mean duration of postoperative hospitalization was 1.7 ± 0.6 days (range, 1.0 to 3.0 days). Three (9.4%) dogs had complications that resulted in death or euthanasia. A 12-month-old neutered male Standard Poodle (weight, 26.0 kg [57.2 lb]) in the rNA group in which premature ventricular contractions were detected preoperatively died of ventricular fibrillation and cardiac arrest 48 hours after surgery. A preoperative echocardiogram had not revealed cardiomyopathy. A 5.5-month-old sexually intact male Yorkshire Terrier (weight, 1.9 kg [3.3 lb]) in the pNA group was euthanatized because of uncontrollable hypernatremia and seizures 5 hours after surgery; findings consistent with postligation neurologic syndrome. A 36-month-old sexually intact male Yorkshire Terrier (weight, 1.5 kg [3.3 lb]) in the pNA group was euthanatized by the referring veterinarian 3 weeks after surgery because of recurrent seizures thought to be resulting from hepatic encephalopathy. There were no significant differences among the pPA, pNA, and rNA groups with respect to incidence of complications or mortality rate (1/8, 1/6, and 1/18, respectively; P = 0.587) or development of postligation neurologic syndrome (1/8, 0/6, and 0/18, respectively; P = 0.213).

Of the 29 surviving dogs, 1 (in the rNA group) was lost to follow-up after discharge from the hospital, and 5 (1 from the pNA group and 4 from the rNA group) were lost to long-term follow-up. The median long-term follow-up time was 22.6 months (range, 7.2 to 37.0 months).

**Treatment groups**—There were no significant preoperative differences between dogs in the pPA versus pNA groups in age or weight at the time of surgery; neutrophil count; serum concentrations of albumin, BUN, bile acids after withholding food for 12 hours, or postprandial bile acids; or serum activities of alanine aminotransferase and alkaline phosphatase (Table 1). However, significant differences between the groups were detected in preoperative PCV (P = 0.01), serum total protein concentration (P = 0.034), and serum glucose concentration (P = 0.019). No significant differences between dogs in the 2 groups were detected in prebanding CEPSS diameter, prebanding portal venous pressure, portal venous pressure with temporary occlusion, or development of postligation neurologic syndrome. A 36-month-old sexually intact male Yorkshire Terrier (weight, 1.5 kg [3.3 lb]) in the pNA group was euthanatized because of uncontrollable hypernatremia and seizures 5 hours after surgery; findings consistent with postligation neurologic syndrome. A 36-month-old sexually intact male Yorkshire Terrier (weight, 1.5 kg [3.3 lb]) in the pNA group was euthanatized by the referring veterinarian 3 weeks after surgery because of recurrent seizures thought to be resulting from hepatic encephalopathy. There were no significant differences among the pPA, pNA, and rNA groups with respect to incidence of complications or mortality rate (1/8, 1/6, and 1/18, respectively; P = 0.587) or development of postligation neurologic syndrome (1/8, 0/6, and 0/18, respectively; P = 0.213).

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**Table 1**—Mean ± SD values for pre- and intraoperative signalment findings and hematologic and serum biochemical analyses in 14 dogs that were prospectively enrolled in a study to evaluate effects of cellophane banding, with and without intraoperative attenuation of the shunt, as treatment for CEPSS.

<table>
<thead>
<tr>
<th>Variables</th>
<th>pPA</th>
<th>pNA</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mo)</td>
<td>7.6 ± 6.9</td>
<td>34.2 ± 37.5</td>
<td>0.067</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>3.5 ± 3.2</td>
<td>3.7 ± 1.9</td>
<td>0.872</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>38.5 ± 4.4</td>
<td>58.3 ± 6.1</td>
<td>0.010</td>
</tr>
<tr>
<td>Neutrophils (× 10³ cells/µL)</td>
<td>8.8 ± 6.9</td>
<td>10.6 ± 8.4</td>
<td>0.650</td>
</tr>
<tr>
<td>TP (g/dL)</td>
<td>4.5 ± 1.0</td>
<td>5.7 ± 0.9</td>
<td>0.034</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>2.3 ± 0.6</td>
<td>2.9 ± 0.5</td>
<td>0.050</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>437.5 ± 906.5</td>
<td>205.9 ± 327.3</td>
<td>0.508</td>
</tr>
<tr>
<td>ALP (IU/L)</td>
<td>514.1 ± 653.6</td>
<td>130.6 ± 91.1</td>
<td>0.123</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>7.2 ± 3.5</td>
<td>8.4 ± 5.1</td>
<td>0.589</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>105.3 ± 20.9</td>
<td>82.5 ± 11.1</td>
<td>0.019</td>
</tr>
<tr>
<td>PPSBA—presurgical (µmol/L)</td>
<td>118.2 ± 71.2</td>
<td>199.8 ± 139.0</td>
<td>0.192</td>
</tr>
<tr>
<td>CEPSS diameter before banding (mm)</td>
<td>6.8 ± 1.4</td>
<td>5.2 ± 1.4</td>
<td>0.159</td>
</tr>
<tr>
<td>CEPSS diameter after banding (mm)</td>
<td>2.5 ± 0.0</td>
<td>5.2 ± 1.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PVP—baseline (cm H2O)</td>
<td>8.5 ± 2.5</td>
<td>8.6 ± 1.9</td>
<td>0.944</td>
</tr>
<tr>
<td>PVP—temporary complete occlusion (cm H2O)</td>
<td>28.2 ± 13.4</td>
<td>21.6 ± 4.5</td>
<td>0.381</td>
</tr>
<tr>
<td>PVP—after banding (cm H2O)</td>
<td>10.5 ± 2.5</td>
<td>10.4 ± 2.5</td>
<td>0.945</td>
</tr>
<tr>
<td>Duration of surgery (h)</td>
<td>1.7 ± 0.5</td>
<td>2.2 ± 1.5</td>
<td>0.369</td>
</tr>
</tbody>
</table>

ALT = Alanine aminotransferase. ALP = Alkaline phosphatase. PVP = Portal venous pressure. TP = Total protein.

To convert kilograms to pounds, multiply by 2.2.
complete CEPSS occlusion, and postbanding portal venous pressure. Postbanding CEPSS diameter was significantly ($P < 0.001$) different between the pNA and the pPA groups. Mean time for measurement of postprandial serum bile acids was not significantly ($P = 0.542$) different between the groups (pPA group, 15.4 ± 4.6 months; pNA group, 17.3 ± 5.8 months).

Follow-up postprandial serum bile acids concentrations were available for 27 dogs. For the entire study population, mean concentrations were 26.8 ± 24.5 at < 2.25 months (n = 16 dogs), 22.1 ± 14.0 from 2.25 to 6 months (12), and 34.9 ± 32.5 µmol/L at > 6 months (22) after surgery ($P = 0.009$). Clinical grades were 1 (n = 19 dogs) and 1.5 (3); a clinical grade was not available in 3 dogs. In the pPA group, 5 of 6 dogs had postprandial serum bile acids concentrations in reference range by 2.25 months after surgery. In the pNA group, all dogs had high concentrations within 2.25 months after surgery. Mean postprandial bile acids concentrations increased over time in the pPA group, whereas concentrations in the pNA group did not ($P = 0.020$; Figure 1). Clinical scores were not significantly ($P = 0.91$) different between the pPA (1.1 ± 0.2) and pNA (1.1 ± 0.2) groups.

**Discussion**

Cellophane banding elicits gradual occlusion of CEPSSs in dogs. Partial attenuation of a CEPSS to a diameter < 3.0 mm during surgery does not appear to be necessary. In the present study, dogs that were prospectively treated with cellophane banding but no intraoperative partial attenuation had a more favorable outcome over time than dogs treated with cellophane banding and partial intraoperative attenuation of the shunt to a diameter < 3.0 mm. In the short term, postprandial serum bile acids concentrations were lower in the pPA group than in the pNA group. However, bile acids concentrations returned to reference range values and remained in that range in dogs in the pNA group, whereas concentrations increased over time in dogs in the pPA group. The signalment and preoperative clinical signs in dogs of the present study were similar to those that have previously been reported3-5,11,20-24 in affected dogs.

Hunt et al17 reported normal results of liver function tests in 87% of dogs when tests were performed 2 to 6 months after cellophane banding with intraoperative attenuation of the shunt to a diameter < 3.0 mm. Youmans and Hunt18 reported that 100% of dogs had complete CEPSS occlusion within 8 weeks of cellophane banding and attenuation of the shunt to a diameter < 2.5 mm, although shunts with a diameter > 3.0 mm were not occluded by 8 weeks. Findings in the present study were similar, in that 5 of 6 dogs in the pPA group had postprandial bile acids concentrations in reference range within 2.25 months of surgery. It can be concluded, on the basis of these findings, that treatment of CEPSS by use of cellophane banding with intraoperative attenuation to a diameter < 3.0 mm results in complete occlusion of the shunt within 2.25 months of surgery in most dogs. However, in the present study, after 6 months, mean postprandial serum bile acids concentrations continued to decrease in dogs in the pNA group, whereas concentrations increased in dogs in the pPA group.

Partial vascular attenuation at the time of surgery may have increased portal venous pressure enough to induce development of acquired shunts. According to Poiseuille’s law, reduction in diameter of a shunt causes a reduction in flow through the vessel that is proportional to the reduction in radius to the fourth power. We expected that the rate of closure would be similar in the pPA and pNA groups. However, because the postattenuation diameter of the shunts in the pPA group was half the diameter of the shunts in the pNA group after surgery, a similar rate of reduction in diameter over time would have a greater effect on blood flow in the pPA versus pNA group. This is illustrated by the lower value for mean serum bile acids concentration at 2.5 months in the pPA group, compared with the pNA group.

Partial occlusion of CEPSSs may put dogs at higher risk for developing chronic subclinical portal hypertension and acquired shunting. In the pNA group, mean PPSBA concentrations continued to decrease over time and were within reference range 6 months after surgery, a finding that indicates that acquired extrahepatic shunting did not develop in those dogs. On the other hand, between 2.25 and 6 months after surgery, mean bile acids concentrations for dogs in the pPA group increased to values above reference range. The most likely explanation for the finding is development of acquired PSSs in dogs in that group. However, none of the dogs in the pPA group had clinical signs of hepatic encephalopathy after 6 months, possibly indicating that the degree of acquired shunting was minimal.

In the present study, postprandial serum bile acids concentrations were used to diagnose and evaluate the outcome of dogs surgically treated for CEPSS. Serum bile acids concentrations are as sensitive as the ammonia tolerance test and more sensitive than routine...
serum biochemical analyses or sulfobromophthalein retention tests for diagnosis of PSSs. It has been reported that determination of postprandial serum bile acids concentrations has 100% sensitivity at detecting PSS and that 94% of dogs with a PSS have serum concentrations that are more than 10 times the upper reference limit. It has also been reported that persistent biochemical evidence of liver dysfunction is a sensitive predictor of clinical relapse in dogs that undergo partial CEPSS attenuation with silk ligature. Nuclear scintigraphy and portal venography could have been used postoperatively to evaluate the dogs in the present study. Portal venography and angiography are considered the gold-standard techniques for detection of congenital or acquired PSSs. Those studies usually require general anesthesia and surgical intervention to place a catheter in the portal vein or cranial mesenteric artery. Nuclear scintigraphic imaging is less invasive and is reliable for use in follow-up monitoring of postoperative CEPSS occlusion but may not be sensitive enough to detect and quantify small shunts. For these reasons, it was difficult to justify the use of such techniques in clinically normal dogs.

There were no significant intraoperative differences between the prospective groups in mean baseline portal venous pressure, CEPSS diameter, portal venous pressure after temporary complete CEPSS occlusion, or postbanding portal venous pressure. These findings indicate that dogs in the prospective groups had similar degrees of development or compliance of hepatic portal vasculature. Therefore, the difference in postbanding CEPSS diameter is likely responsible for the significant difference in the variation of postprandial serum bile acids concentrations between dogs in the pPA and pNA groups. It would have been ideal to measure portal blood flow instead of portal venous pressure for evaluation of liver circulation, but this is not typically performed in clinical cases.

With the exception of PCV, serum total protein concentration, and serum glucose concentration, there were no significant preoperative differences in laboratory values between dogs in the pPA and pNA groups. Although significantly different, mean values for PCV and glucose concentration were within reference range for both groups. For the pPA group, mean total protein concentration was below reference range values, but not to a degree considered clinically important. It has been reported that in dogs with intrahepatic shunts, low PCV and low serum total protein and albumin concentrations prior to surgery are risk factors for poor short- and long-term outcomes. Findings of low PCV and low serum protein and albumin concentration are not useful as prognostic indicators for survival in dogs with CEPSS.

In conclusion, cellophane banding is a safe, effective technique for management of dogs with CEPSSs and compares favorably with other reported methods. Intraoperative attenuation of CEPSS to a diameter < 3.0 mm is not necessary. The treatment of CEPSS with cellophane banding without intraoperative attenuation may decrease the need for extensive intraoperative monitoring, including portal manometry. This could significantly decrease surgical and anesthetic time and risk, as well as overall expense. It may also facilitate minimally invasive treatment of CEPSS.

Appendix

Clinical grading scale used to assess dogs with CEPSSs.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Absence of clinical signs</td>
</tr>
<tr>
<td>1.5</td>
<td>Absence of clinical signs; owner has not offered diet with standard protein content</td>
</tr>
<tr>
<td>2</td>
<td>Improvement with occasional clinical signs, with or without requirement for a low-protein diet</td>
</tr>
<tr>
<td>3</td>
<td>No improvement or complete relapse</td>
</tr>
</tbody>
</table>

References


