



SURGICAL MANAGEMENT OF A SINGLE, EXTRAHEPATIC PORTOSYSTEMIC SHUNT

CASE REPORT

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REVIEWED BY

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Signalment:

“Harley”

Female Intact Yorkshire Terrier

DOB: 3/8/07

History:

Harley first presented to a local emergency clinic for abnormal neurologic behavior (pacing, dizziness, bumping into walls). Blood testing at that time was consistent with a portosystemic shunt (hypoalbuminemia, hypocholesterolemia, low BUN). She was started on empirical treatment for a suspected portosystemic shunt (lactulose, metronidazole, amoxicillin) which the owner reports was successful in controlling Harley’s neurologic signs. Prior to presentation to VMSG, a bile acids test was also performed (elevated) as well as a technetium scan (positive for shunting -likely single, extra-hepatic vessel).

Clinical Examination:

Harley’s preliminary surgical consult at VMSG was performed 8/27/08. At that time we initiated treatment with potassium bromide (in addition to Harley’s above mentioned medications) in preparation for surgical shunt ligation (ameroid constrictor placement) scheduled for 9/5/07. Unfortunately, when Harley presented for surgery, preanesthetic testing was suggestive of progressive underlying disease (worsening hypoalbuminemia (ALB 1.5, PCV 28%, TS 3.0) and mild ascites. The owner also reported noticing dark stools at home. As Harley’s clinical signs and laboratory findings were consistent with GI blood loss and portal hypertension which precluded anesthesia and surgery at that time, GI protectants (famotidine and carafate) were prescribed and surgery rescheduled for 9/11/07. Preanesthetic evaluation on 9/11/07 found Harley to be a better anesthetic candidate (ALB 1.6, PCV 32%, TS 3.4) and she was hospitalized in preparation for surgery 9/12/08.

Diagnostic Imaging Data:

Abdominal ultrasound (preop): Microhepatica, diffusely thickened bowel walls (2.5 – 4mm), mild ascites.

Diagnosis: Portosystemic shunt (likely single, extrahepatic)

Management/ Treatment:

An exploratory laparotomy was performed 9/12/08 and a single, extrahepatic, portocaval shunt identified. A 5.0 mm ameroid ring constrictor was successfully placed around the shunting vessel and Harley recovered uneventfully from anesthesia. She was monitored in hospital postoperatively with medical therapy including potassium bromide, lactulose, metronidazole, and Clavamox.

Two days following surgery, discomfort and abdominal distension were noted on physical examination. Progressive ascites was seen on abdominal ultrasound and abdominocentesis performed which yielded 260mls of clear, slightly pink fluid. (cytology of the fluid showed non-degenerate neutrophils (10-12/HPF) and no bacteria). Portal hypertension +/- portal vein thrombosis was suspected and Harley was started on heparin and buprenorphine in addition to her current medical therapy. Repeat abdominocentesis performed 48 hours later yielded 60mls of clear ascites fluid and treatment with furosemide and spironolactone was initiated.

Harley then began to show evidence of progressive clinical improvement with static (minimal) abdominal distension and ascites. She was discharged to the owner 8 days following surgery with instructions to return for recheck examination in one week, and recheck CBC/CHEM in 2,4,6, and 12 months. Medications prescribed for use at home included: Clavamox, metronidazole, lactulose, metoclopramide, famotidine, carafate, potassium bromide, furosemide, and spironolactone.

Follow-up:

Since surgery, the owner reports that Harley has been doing very well at home. She has been gradually weaned off of her medications and as of 6/3/08 she is showing no evidence of clinical signs.

Discussion: Congenital, extra hepatic portosystemic shunts (PSS) are usually single vessels that connect the portal venous system to the systemic circulation. Although the majority of these vessels will empty into the abdominal vena cava, they can also empty into the azygos vein. Several small, purebred dog breeds appear to be at greater risk of this condition including Yorkshire Terriers, Havanese, Maltese, Pug, and Miniature Schnauzer.

Abnormalities on physical examination (small stature, poor body condition, unkempt haircoat, copper colored iris), and clinical signs (hepatic encephalopathy, anorexia, vomiting, diarrhea, pica) are proportionate to the quantity of portal blood being diverted away from the liver.

Clinicopathologic changes: Hepatic dysfunction (low albumin, blood glucose, creatinine, cholesterol, BUN, coagulopathy), elevated post-prandial bile acids +/- elevated fasting bile acids, elevated liver enzymes (ALP/ALT), microcytosis and dilute urine +/- ammonium biurate crystalluria.

Hepatic histopathologic findings: Mild to moderate lobular hepatocellular atrophy, inconspicuous portal vein tributaries, arteriolar duplication, lipidosis, and vacuolar change, smooth muscle hypertrophy, increased lymphatics around central veins, and Ito cell hypertrophy. (similar changes noted in dogs with PSS and microvascular dysplasia – a disorder which mimics PSS however a macroscopic vascular anomaly cannot be identified).

Diagnostic Imaging: (Radiographs): Microhepatica (60-100%), bilateral renomegaly, uroliths (may be radiolucent)

Confirmation of a porto-systemic shunt can be achieved via various imaging modalities including: abdominal ultrasound, nuclear scintigraphy, portography, CT/ MRI.

Patients with portosystemic shunting can be managed using either surgical or medical therapy.

- **Surgical management:** Many extrahepatic shunts (48%-68%) cannot be completely occluded during a single surgical procedure without causing life-threatening portal hypertension. Partial/ gradual occlusion of shunt vessel can be achieved via placement of ameroid ring, cellophane band, or silk suture. Although the goal with ameroid ring placement is gradual occlusion of the shunting vessel within 30 days of placement (due to fluid imbibement and subsequent swelling of the casein based ring or inflammation/ fibrosis surrounding ring), this is not always the case. Venous thrombosis formation in the area of the ameroid ring placement may result in more rapid occlusion of the shunting vessel and subsequent portal hypertension. Multiple, acquired shunts may develop post-operatively.
- **Medical management:** Symptomatic treatment can temporarily improve quality of life in patients with portosystemic shunting. Although some dogs can live for years with medical management, some reports show that 1/2 of dogs without definitive treatment of their shunt are euthanized within one year of initiating medical therapy (usually due to uncontrolled hepatic encephalopathy). Medical therapy includes: oral anti-convulsants (potassium bromide or Phenobarbital), lactulose (decrease GI transit time and alters intestinal pH), oral antibiotics (reduce urease producing intestinal bacteria) and a low protein/high carbohydrate diet (reduces the source of ammonia). Surgical patients should also be treated with the above medical therapy measures preoperatively.

Recent studies have shown median long term survival following PSS attenuation to be 72 months in animals over 5 years of age (JAVMA 2008). Post operative complications can occur regardless of the surgical procedure performed. These complications include: seizure activity (including life threatening status epilepticus which can occur up to 3 days post-operatively) and life threatening portal hypertension (clinical signs include abdominal pain, hypotension, ascites, and vomiting)

References:

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