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Advanced Surgical Procedures for Advanced Practitioners Mini Series

Session 3: Remove the Gall Bladder and Close the Shunt

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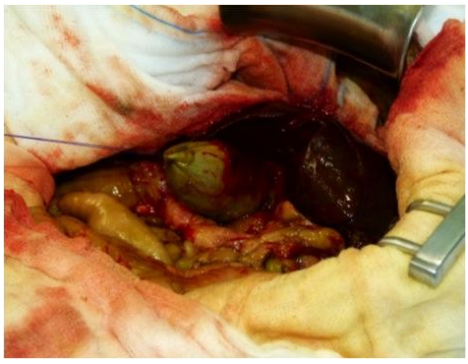

Remove the gall bladder and close the shunt

Anatomy of the Biliary System

The biliary system consists of the gallbladder (GB), cystic duct, common bile duct (CBD), hepatic ducts, interlobular ducts, intralobular ducts, bile ductules, and hepatic canaliculi.

The GB, located and attached to the liver to the right of midline within a fossa between the right medial and quadrate liver lobes, units with the CBD by means of the cystic duct. The cystic duct extends from the neck of the GB to its junction with the first hepatic duct. The communication of the CBD and duodenum is anatomically distinct in the dog and cat. In a medium-sized dog, the CBD is 5-cm long and 2.5 mm in diameter and empties into the duodenum 1.5 cm to 6.0 cm distal to the pylorus at the major duodenal papilla. At the junction with the intestine, the CBD courses intramurally for 2 cm. The CBD of the dog opens near the smaller of two pancreatic ducts (minor pancreatic duct) at the major duodenal papilla; the larger pancreatic duct (accessory pancreatic duct) opens a few centimeters distally. The bile duct of the cat is long and sinuous compared with the dog. In the cat, the CBD fuses in an ampulla with the pancreatic duct just before entering the duodenal papilla 3-cm caudal to the pylorus. In some cats the major pancreatic duct opens separately but immediately adjacent to the CBD. Approximately 2-cm caudal to the major duodenal papilla, the accessory pancreatic duct enters the duodenum (minor duodenal papilla) in 20% of cats. While the pancreas in each species is nearly always drained by two ducts, a great deal of variation exists. Because of the close proximity of the pancreas and CBD, pancreatitis commonly influences bile flow through the major bile duct, causing obstruction to flow and jaundice. In the cat, inflammatory, neoplastic, or obstructive disorders involving the distal CBD can affect both the biliary tree and pancreas. In fact it is possible that microlithiasis or sludged bile transiently obstructing the distal CBD causes intermittent bile duct occlusion and idiopathic pancreatitis in the cat.

Nestled deep within its fossa, an empty GB may not be readily apparent on gross inspection during surgical exploration or laparoscopy. Embryologically derived from the foregut, the structure of the GB resembles the intestines. Its luminal surface is covered with mucosa containing fine microvilli that expand the surface area for resorptive and exchange processes. Similar to the intestine, the GB also has a lamina propria with a resident lymphoplasmacytic population, a muscularis, a layer of connective tissue, and an outermost serosa. Mucus glands within the GB provide mucin that protects the luminal epithelium from the cytolytic effects of bile acids. Mucin production is stimulated by inflammatory cytokines, endotoxins, and prostaglandins and excessive GB mucin production in the dog seemingly contributes to biliary mucocele formation. Having fewer mucus glands in their GB may explain why mucoceles do not develop in the cat.

Arterial perfusion is solely derived from the left branch of the proper hepatic artery (cystic artery). Having only a single source of perfusion makes the GB and CBD uniquely susceptible to ischemic necrosis following blunt abdominal trauma (ie, vascular shearing trauma). Compromised perfusion can lead to biliary tree rupture and bile peritonitis.

The GB functions as a storage reservoir where bile is concentrated, acidified, and modified. However, its function is not essential such that cholecystectomy is usually well tolerated. Bile enters the GB continuously in a low-flow low-pressure system. Pressure within the system is generated by hepatic bile secretion and the tonic contraction of the sphincter of Oddi, the one-way sphincter at the duodenal papilla.

Gallbladder

Gallbladder mucocele

This disorder is characterized by progressive accumulation of tenacious mucin-laden GB bile, which may extend into the cystic, hepatic, and common bile ducts, resulting in variable degrees of EHBO.

Progressive expansion of a biliary mucocele leads to GB ischemic necrosis, which could lead to rupture and bile peritonitis, and sometimes opportunistic infection.

Median age of presentation is 10 years, but can be as young as 3 years. There is no gender predisposition but there is an increased risk in Shetland Sheepdog, as well as Cocker Spaniel and Miniature Schnauzers. It has not yet been convincingly described in cats

Clinical Signs:

Clinical illness averages 5 days, although some dogs have vague episodic signs for months (ie, inappetance, vomiting, vague abdominal pain). In decreasing order and frequency, clinical signs have included: vomiting (87%), abdominal pain (87%), anorexia (78%), jaundice (57%), tachypnea (65%), tachycardia (44%), polyuria and polydipsia (30%), fever (26%), diarrhea (26%), and abdominal distension (13%).

Dogs progressing to GB rupture demonstrate abdominal pain (93%), jaundice (64%), tachycardia and tachypnea (36%), and fever (29%).

Laboratory finding:

Hematology:
Leukocytosis (50%, mature neutrophilia: 40%, monocytosis: 27%)

Biochemistry:
High liver enzymes including ALP (100%), GGT (86%), alanine aminotransferase (ALT) (77%), and aspartate aminotransferase (AST) (60%), and hyperbilirubinemia (63%). Dogs with GB rupture that died generally had higher liver enzyme activity, bilirubin concentrations, and total white blood cell counts than survivors.

Culture:
Aerobic bacteria have been cultured from bile or GB wall, with a number of enteric organisms identified (ie, E. coli, Enterobacter spp, Enterococcus spp, Staphylococcus spp, Micrococcus spp, Streptococcus spp).

Histopathology:
Histologically, cystic mucosal hyperplasia is common in the GB wall. All dogs have had abnormally thick biliary debris: some is viscous and mucin laden, some is more liquid, some is dark green-black, some with a white bile component, some with gritty black material, and some with a firm gelatinous matrix. Cysts in the GB mucosa are common (single layer of epithelial cells surrounding mucus-filled lumen) and mucus-secreting glands are widely distended with mucus resembling GB debris. Transmural ischemic necrosis may develop, leading to necrotizing cholecystitis and GB rupture. The most common site of rupture is the fundus.

Predisposing factors

Factors predisposing to GB mucocele formation include middle to geriatric age, hyperlipidemia/hypercholesterolemia (idiopathic, associated with pancreatitis or nephrotic syndrome or endocrinopathies, including typical and atypical hyperadrenocorticism and hypothyroidism),

Decreased GB motility (geriatric or possibly steroidal influence) leads to luminal bile stasis and enhanced absorption, promoting formation of biliary sludge.

Diagnostic Imaging:

Ultrasound

The US image is diagnostic and is characterized by finding a large GB filled with hyperechoic nongravitationally dependent contents and a hypoechoic ‘‘rim sign’’ (an immobile star-like or kiwi fruit-like appearance). This pattern reflects a dense central biliary conglomerate comprised of thick sludge containing tenacious mucin that is tightly adhered to the GB mucosa.

In the case that free fluid is observed, aspiration should be done as close to the GB as safely possible. Samples collected near the GB are more likely to disclose bacteria or bilirubin crystals. Important for diagnosis of septic or bile peritonitis. Measurement of total bilirubin in collected fluid and comparison to the serum bilirubin concentration helps clarify the likelihood of bile peritonitis (eg, fluid bilirubin **R**10-fold the serum concentration). GB rupture should be suspected if the wall is discontinuous, trilaminated, or the GB is surrounded by hyperechoic pericholecystic fat or a hypoechoic fluid ring; these patterns warrant recommendation for emergency surgery.

US-guided cholecystocentesis should not be performed if a GB mucocele is probable.
Ultrasonography also may detect hepatomegaly and either a heterogenous or hyperechoic hepatic parenchymal pattern. In some dogs, hypoechoic ‘‘nodules’’ correspond to a severe vacuolar hepatopathy. AfterGB removal, sequential hepatic US evaluations are necessary to see if parenchymal lesions resolve. Persistent abnormalities likely reflect underlying medical disorders that need investigation.

Medical treatment

Dogs lacking clinical signs of mucocele leakage or biliary tree obstruction at the time of initial diagnosis may benefit from administration of UDCA, SAMe (S-adenosyl_L-methionine) providing both a choleretic and antioxidant benefit, as well as antibiotic therapy.

Medical management can be considered in early cases especially if they have comorbidities and are poor anesthetic candidates. Providing that sequential biochemical and US evaluations are used to monitor syndrome progression (every 6 weeks).

Surgical treatment

Cholecystectomy is the best course of treatment for GB mucocele. There is a report of rerouting the gall bladder. Most dogs with gall bladder mucocele do not have gallbladders that remain healthy enough to allow for a viable cholecystoduodenostomy, and progressive gall bladder wall necrosis may occur. Cholecystotomy for removal of GB contents without cholecystectomy is not advised either because mucoceles have recurred in several dogs treated with cholecystotomy . Furthermore, microscopic mural necrosis may exist at the time of surgery that is not grossly evident, leading to postoperative GB rupture.

While cholecystectomy is an effective treatment for GB mucocele, perioperative mortality is high for symptomatic dogs with a ruptured GB complicated by sepsis. If bile peritonitis is present, the peritoneal cavity must be extensively cleansed with sterile warm polyionic fluids to remove debris, bacteria, and injurious bile salts. Abdominal drains may be necessary in the case of septic peritonitis. Antibiotics should be administered for 4 to 6 weeks.

For dogs with a firm US diagnosis of GB mucocele, elective cholecystectomy as a prophylactic procedure is recommended. This averts mucocele maturation and subsequent GB ischemic necrosis.

After GB resection, chronic choleretic therapy is recommended. Underlying causes of hyperlipidemia or endocrine disorders should be identified and managed appropriately. Clinicopathologic abnormalities normalize after GB removal in most dogs, except those with associated suppurative cholangiohepatitis or unresolved endocrinopathies.

At surgery, it is important that the CBD is cleansed of obstructing debris and that the distal duct is canulated and flushed to ascertain patency through the duodenal papilla. Opening the bowel to identify the duct stoma has been necessary in some cases.

Because bile stasis predisposes to infection, broad-spectrum antimicrobials are initially recommended (aerobic and anaerobic bacteria), while cultures of bile, GB wall, and liver are pending. Antibiotics should be started before or during the cholecystectomy. Evidence of bacteria in cytologic samples or the presence of suppurative cholecystitis affirm a need for chronic antimicrobial administration.

The resected GB should be submitted for histologic characterization, and a liver biopsy should be collected distant to the site of surgery to survey for concurrent liver disease.

Cholecystectomy

Indications

The most common surgical procedure performed on the gallbladder of dogs and cats is cholecystectomy. Cholecystotomy is normally not indicated as the gall bladder is the cause of the disease and this procedure has a higher rate of complications causing an increase in morbidity and possible mortality, due to dehiscence and bile peritonitis.

The most common indication for cholecystectomy in the dog is biliary mucocele. The primary indication for feline cholecystectomy is necrotizing cholecystitis,. Followed by cholelithiasis.

Technique

The relative difficulty associated with cholecystectomy in dogs and cats pertains, in part, to the extent of adhesion formation between the gallbladder and surrounding tissues (eg, liver, greater omentum, falciform ligament) and the status, including integrity, of the gallbladder Cholecystectomy is usually performed via laparotomy in dogs and cats, although a laparoscopic procedure has been described in dogs.

- Dissect the gallbladder from the hepatic fossa using blunt and sharp dissection. Initially start with sharp dissection of the attachments of the gall bladder to the liver capsule using scissors. Followed by blunt dissection using cotton tips or the canula of a poole suction tip.
- Achieve hemorrhage control of the hepatic fossa via local pressure, electrosurgery, and topical hemostatic agents. Heamorrhage is normally encountered.
- Ensure patency of the bile duct by passing a catheter (eg, 5F red rubber catheter) through the bile duct in a normograde (preferred technique) or retrograde fashion. Retrograde catheter passage requires a duodenotomy to access the duodenal papilla.
- Dissection to the level of the cystic duct usually reveals the cystic artery, which is ligated or occluded collectively with the cystic duct with an appropriately sized vascular clip or suture. The stump is assessed for any leakage or bleeding.
- Confirm hemostasis and perform intraperitoneal lavage before closure.

Extrahepatic portosystemic shunts

Portosystemic shunts (PSS) are vascular anomalies that redirect blood from the portal vein to the systemic circulation, bypassing the hepatic sinusoids and liver parenchyma. Normally, blood draining the stomach, intestine, spleen, and pancreas enters the portal vein and perfuses the liver through the sinusoidal network before entering the hepatic veins, and subsequently the caudal vena cava. Portal blood contains nutrients, trophic hormones (intestinal and pancreatic), bacterial products, and intestinal-derived toxins.1,2,6 The fetal liver has limited function to process these products, and a large shunting vessel (ie, ductus venosus) normally exists to bypass the hepatic circulation as a protective mechanism.

This vessel normally closes shortly after birth, establishing hepatic circulation. If the ductus venosus remains patent, intrahepatic portosystemic shunting persists. Persistence of anomalous connections between the fetal cardinal and vitelline systems results in extrahepatic portosystemic shunting. When blood bypasses the liver, delivery of trophic factors (particularly insulin and glucagon) to the liver is decreased, resulting in poor hepatic development, decreased protein production, reticuloendothelial dysfunction, altered fat and protein metabolism, hepatic atrophy, and eventually hepatic failure. The severity of clinical signs is related to the volume and origin of blood bypassing the liver and may include hepatic encephalopathy (HE), chronic gastrointestinal signs, lower urinary tract signs, coagulopathies, and retarded growth. In animals with portosystemic shunting, concentrations of endogenous and exogenous toxins that are normally metabolized or eliminated by the liver (ammonia, gut-associated encephalopathic toxins, hormone metabolism, benzodiazepine-like substances, aromatic amino acids;
Increase, whereas normal hepatic metabolic function gluconeogenesis, urea cycle, uric acid cycle) decreases.

Anatomy

The portal vein is formed by the convergence of the cranial and caudal mesenteric portal branches within the mesentery. It provides up to 80% of the blood flow (20% through the hepatic artery) and 50% of the oxygen content (50% through the hepatic artery) to the liver.1–7 Additional tributaries from the spleen, stomach, pancreas, and proximal duodenum join the portal vein before its bifurcation. In dogs the portal vein divides into left and right branches and the left branch divides further to supply the central and left lobes. In the cat the portal vein divides directly into left, central, and right branches.The portal vein then branches into smaller venules whereby the blood enters the parenchyma at the portal triads, travels through the hepatic sinusoids, and drains to the central veins, which then confluence to larger hepatic venules and hepatic veins that drain into the caudal vena cava. As it travels through the sinusoids, the portal blood is delivered to the hepatocytes and cleansed by the reticuloendothelial system. If this path is interrupted by an anomalous vessel(s), blood is diverted away from the liver and reaches the systemic circulation before hepatic circulation and cleansing.

PSS can either be congenital or acquired. Congenital PSS most commonly occur as single vessels that provide direct vascular communication between the portal venous supply and the systemic venous circulation (caudal vena cava or azygous vein), bypassing the liver. They commonly occur as a single intra- or extrahepatic communication. Rarely some animals have 2 or more congenital communications.

The types of congenital hepatic vascular malformations found in dogs and cats include single intrahepatic portocaval shunts, single extrahepatic portocaval shunts, portal vein atresia with resultant multiple portal-caval anastomoses, HAVM, and PVH (formally called microvascular dysplasia).
Approximately 25% to 33% of congenital PSS are intrahepatic (IHPSS) in dogs and cats. Single EHPSS are noted in 66% to 75% of cats and dogs with congenital PSS, with the most common location being portocaval.Most IHPSS are found in larger-breed dogs, whereas most EHPSS are seen in smaller breeds. Some EHPSS, such as portoazygous or portophrenic shunts, may be associated with less severe clinical signs, possibly because of intermittent compression by the diaphragm or engorged stomach. Dogs with IHPSS generally have the largest volume of portal blood diverted through their shunt, resulting in more severe clinical signs at an earlier age.

Pathophysiology

Most of the clinical signs associated with PSS result from HE, the pathogenesis of which is complex and largely unknown in our veterinary patients. HE is a neuropsychiatric syndrome involving a gamut of neurologic abnormalities that manifest if more than 70% of hepatic function is lost.The healthy liver serves a filtration function against a multitude of neurotoxic substances that are absorbed across the gastrointestinal barrier. If liver function is altered or shunting occurs, the liver cannot appropriately perform its role in either metabolism or

substance clearance. Toxic substances subsequently accumulate in the systemic circulation and alter multiple aspects of central nervous system (CNS) function. Ammonia may be considered the most important because increased concentrations trigger a sequence of metabolic events that have been implicated in HE in rats, humans, and dogs. Ammonia is the easiest substance measured in veterinary patients and treatments to decrease ammonia concentration reduce the signs of HE. The degree of encephalopathy is not well associated with the blood ammonia levels,suggesting that other suspected neurotoxins are also important in the pathophysiology.

Diagnosis

Congenital EHPSS are seen more commonly in small, toy breed dogs, some of the most common breeds are Yorkshire, schnauzer, maltese. In cats EHPSS is more common than IHPSS but this can also be seen. Intrahepatic shunts are overrepresented in larger breed dogs especially irish woflhound, retriever, Australian cattle dog. Inheritance has been documented in the maltese dog and Yorkshire terrier.

History

Most dogs and cats evaluated for PSS present with signs of chronic or acute illness when they are less than 1 to 2 years, but some animals have presented with more than 10 years.

The history typically suggests the patient has shown failure to thrive since birth, is small in stature (or the runt of the litter), has weight loss (11%) or failure to gain weight, has anesthetic intolerance, is dull or lethargic at times, and displays bizarre behavior (41%–90%; star-gazing, head-pressing, staring into walls or corners, random barking, intermittent blindness,pacing, or aggression).Some animals present with a history of hematuria, stranguria, pollakiuria, or urinary obstruction (20%–53%). Polyuria and polydipsia (PUPD) are common complaints in dogs. Correlation of the onset of signs with meal ingestion has been reported in only 30% to 50% of patients. Gastrointestinal signs of vomiting, pica, anorexia, or diarrhea are also common.

Laboratory analysis

Most common hematological changes include mild to moderate, microcytic, normochromic nonregenerative anemia (60%–72% of dogs, 30% of cats).The cause of the microcytic anemia is not fully known, although studies suggest a defective iron-transport mechanism, decreased serum iron concentrations, decreased total iron-binding capacity, and increased hepatic iron stores in Kupffer cells. Leukocytosis can occur and is suspected to be secondary to stress or inadequate hepatic endotoxin and bacterial clearance.

Serum biochemical abnormalities are extremely common in animals with PSS. In dogs the most common deficiencies include hypoalbuminemia (50%), decreased BUN (70%), hypocholesterolemia, and hypoglycemia, which result from decreased hepatic synthesis. In cats hypoalbuminemia is uncommon, but decreased BUN concentrations are typical.Mild to modest increases (2- to 3-fold) in alkaline phosphatase(ALP) and alanine aminotransferase (ALT) are also reported.

Abnormalities on urinalysis include decreased urine specific gravity (>50% are hyposthenuric or isosthenuric) and ammonium biurate crystalluria.Decreased urine specific gravity most likely results from polydipsia and poor medullary concentration gradient. Hyperammonuria from a deficient hepatic urea cycle, along with inappropriate uric acid metabolism, results in excessive ammonia and urate excretion in the kidneys and ultimately ammonium biurate crystalluria (26%–57% of dogs and 16%–42% of cats)or stone formation.

Liver function testing

Fasting (12 hours) and 2-hour postprandial serum bile acids (SBA) are the most widely used tests for evaluating liver function in animals with PSS. Bile acids, which are synthesized in the liver from cholesterol, conjugated, secreted into the bile canaliculi, and stored in the

gallbladder, are then released into the duodenum after a meal, aiding in fat emulsification, metabolism and lipid absorption. Bile acids are reabsorbed from the ileum, transported into the portal venous system, and extracted by hepatocytes for recirculation.By measuring these levels this entire circuit is evaluated. Although increases in postprandial bile acids have been found to be 100% sensitive for detection of a PSS in dogs and cats in some studies, others have found that paired samples are 100% sensitive, but not individual measurements.

If a false-negative bile acid test is suspected, measurement of plasma ammonia can be performed. Basal ammonia in fasting animals is close to 100% sensitive.

Diagnostic Imaging

Abdominal ultrasonography :

Ultrasound is the most widely used diagnostic tool for PSS in many practices. It is noninvasive, does not require general anesthesia like angiography (although sedation makes finding EHPSS more reliable in many circumstances). Decreased numbers of hepatic and portal veins, a subjectively small liver, and an anomalous vessel are readily seen with ultrasound in PSS. Extrahepatic shunts are typically more difficult to diagnosis due to the small patient and vessel size, and the presence of gas in the bowel and lungs obscuring the image. ultrasonography for detection of shunts (sensitivity 74%–95% and specificity 67%–100%).The results of ultrasound are dependent of the equipment, operator, and experience.

Portovenography:

Portography is less commonly performed in many large facilities than previously because of availability of less invasive imaging modalities (ultrasound, scintigraphy,CT angiography). This procedure requires a laparotomy, portable fluoroscopy (C-arm).Sensitivity of intraoperative portography has been reported to be between 85% and 100% and is dependent on patient positioning.

Treatment

Medical Treatment:

Medical management is recommended before surgical treatment. Medical management controls the clinical signs associated with shunting but does not treat the underlying hepatic perfusion, therefore surgery is recommended in most cases with correctable disease.

Hepatic encephalopathy: aggressive effort should be made to decrease ammonia:

- NPO
- IV fluids to normalize dehydration
- Glucose if needes should be added
- Warme enemas
- Oral or rectal lacutose
- Antibtiotics to decrease urease producing bacteria (metronidazole, ampicillin, neomycin)
- Anticonvulsant treatment if needed
    - Benzodiazepines, diazepam, levetiracetam, midazolam
    - Potassium bromide, phenobarbital

Most of this cases wont tolerate the stress of the anesthesia and surgery, because of this they need to be treated medicall normaly for 2-4 weeks prior to surgery to stabilize them and improve their body conditions, the medical treatment will be composed of lactulose, antibiotics and a liver diet for at least one month prior to surgery.

Prognosis with medical treatment

Prospective studies on the medical management of dogs or cats with portosystemic vascular anomalies have not been reported. In 1 retrospective study of 27 dogs with congenital PSS evaluated after long-term medical management alone, 52% were euthanized with a median survival time (MST) of 9.9 months and 15% were lost to follow-up. One third of the animals survived long term (MST of 56.9 months; range 5 months to >7 years), with many of those still alive at the time of evaluation. Of the 27 dogs, 9 had EHPSS and 17 had IHPSS. Dogs with IHPSS on medical management alone often had persistent neurologic signs with treatment compared with dogs with EHPSS whose clinical signs were either similar or occurred less often once medicated.
29 Of dogs with IHPSS, 65% were euthanized, mostly due to uncontrolled signs of HE. One third of dogs with EHPSS were euthanized because of persistent clinical signs. There were no correlations between levels of bile acid, serum protein, albumin, ALP, ALT, and MCV and survival times.

Surgical Treatment:

A definitive diagnosis of extrahepatic PSS can usually be made during exploratory laparotomy if the veterinarian is familiar with the anatomy of the abdomen.Most extrahepatic portocaval shunts terminate on the caudal venal cava cranial to the renal veins at the level of the epiploic foramen. Occasionally, they may travel along the lesser curvature of the stomach and terminate on the phrenic or left hepatic vein cranial to the liver. Portoazygous shunts usually traverse the diaphragm at the level of the crura or aortic hiatus. Thorough exploration is warranted in all dogs with single congenital PSS because of the possibility, although rare, of a second shunt. In addition, the bladder should be palpated for calculi if preoperative ultrasonography was not performed.

Surgical options

Congenital PSS can be completely or partially ligated with nonabsorbable sutures or gradually attenuated with an ameroid constrictor, cellophane band, or hydraulic occluder. Gradual attenuation is preferred to reduce the risk of postoperative complications. Extrahepatic shunts are occluded as close to their terminus as possible to reduce blood flow from shunt tributaries.

Suture Ligation

Shunt ligation in dogs is often performed with silk because of its superior handling characteristics.
Nonencephalopathic dogs can often tolerate complete shunt ligation; however, up to 80% of animals undergoing acute shunt occlusion require partial attenuation. Degree of attenuation is based on visual inspection for evidence of portal hypertension, such as pallor or cyanosis of the intestines, increased intestinal peristalsis, cyanosis or edema of the pancreas, and increased mesenteric vascular pulsations. The surgeon can
measure portal and central venous pressures. Recommendations for postligation pressures include a maximum portal pressure to 17 to 24 cmH2O, maximal change in portal pressure of 9 to10 cmH2O, and maximal decrease in central venous pressure of 1 cmH2O.

Some investigators recommend second surgery for animals undergoing partial suture ligation of congenital PSS. Partially attenuated shunts can be completely ligated during a subsequent surgery in 75% of animals. Liver function returns to normal in up to 70% of dogs undergoing a single partial ligation, however, indicating that many shunts continue to narrow after the initial attenuation.

Ameroid Constrictor

Ameroid constrictors have an inner ring of casein that is surrounded by a stainlesssteel sheath. Casein is a hygroscopic substance that swells as it slowly absorbs body fluid, reducing the ring's internal diameter by 32%. It also stimulates a fibrous tissue reaction that

results in gradual shunt occlusion over 2 or more weeks. In some animals, thrombus formation could result in more rapid obstruction of partially attenuated shunts. The choice of ameroid constrictor size for PSS occlusion is based on shunt diameter; preferably, the constrictor should have an internal diameter larger than the shunt. Extrahepatic PSS are most commonly attenuated with ring of 5 mm inner diameter. Before placement of an ameroid constrictor, the perivascular fascia is gently dissected away from the shunt. Dissection should be minimized to prevent postoperative movement of the ring and subsequent acute shunt occlusion. The ameroid constrictor is slipped over the flattened vessel, and the slot in the constrictor ring is obstructed with a stainless steel key. Measurement of portal or central venous pressures is unnecessary with ameroid constrictor placement as long as the shunt is not attenuated at the time of surgery.

Cellophane bands

Cellophane bands can be constructed from clear, nonmedical-grade cellophane like that used to wrap flowers and candy baskets. The cellophane is cut into 1 by 10 cmstrips and gas sterilized. During surgery, a strip is folded longitudinally into thirds to make a thick, flexible band. The shunt is dissected with right-angle forceps, whichare then used to gently thread the band around the shunt. The band is held in place by securing the ends together with surgical clips; excess cellophane is removed 1to 2 cm beyond the clips. Like ameroid constrictors, ellophane bands cause fibrous tissue reaction and gradual shunt occlusion. Initially, attenuation of the shunt to less than 3mmin diameter was performed to encourage complete shunt closure. In more recent studies, complete occlusion was demonstrated in dogs and cats that underwent cellophane banding without intraoperative attenuation.

Hydraulic occluders

Hydraulic occluders has been used for gradual attenuation of IHPSS. A hydraulic occluder consists of a silicone and polyester cuff (DOCXS Biomedical Products and Accessories, Ukiah, CA) connected by tubing to a vascular access port The cuff is secured around the shunt with suture and the attached access port is inserted under the skin. After surgery, a small amount of sterile saline is injected through the port every 2 weeks to gradually inflate the cuff. Shunt closure usually occurs in 6 to 8 weeks and is not dependent on fibrous tissue formation.

Complications of attenuation

Acute complications of shunt attenuation include refractory hypoglycemia, prolonged anesthetic recovery, hemorrhage, seizures, intraoperative hemorrhage, and portal hypertension.

Hypoglycemia occurs in 44% of dogs after EHPSS attenuation and is refractory to dextrose administration in 29% of affected dogs.107 Dogs that have refractory hypoglycemia or delayed anesthetic recovery may respond to glucocorticoid administration (eg, dexamethasone, 0.1–0.2 mg/kg intravenously).

Clinical signs of portal hypertension include pain, abdominal distension from ileus or ascites, decreased central venous pressure, prolonged capillary refill time, pale mucous membranes, poor peripheral pulses, and gastrointestinal hemorrhage. Acute portal hypertension is most commonly seen in animals undergoing suture ligation.

Postoperative seizures develop in 3% to 7% of dogs and 8% to 22% of cats after shunt attenuation. Seizures may occur up to 80 hours after surgery and are not associated with hypoglycemia, hyperammonemia, or attenuation technique. Initial treatment includes intravenous boluses of midazolam or propofol. If seizures persist or reoccur, the animal is anesthetized with an intravenous bolus of propofol (5–8 mg/kg) and maintained under anesthesia on a propofol continuous-rate infusion (CRI; 0.1–0.2 mg/kg/minute). There is a suggestion that propofol may not eliminate cerebral seizure activity when monitored on EEG, and other injectable anticonvulsants should be considered, although in the authors' experience propofol has been effective in seizure management of PSS patients. Mannitol is

administered intravenously every 6 hours to reduce intracranial swelling. Electrolyte and glucose abnormalities are corrected, and supportive care is provided.

The most common chronic complication of PSS attenuation is persistence or recurrence of clinical signs. Differentials include continued flow through the original shunt, the presence of a second shunt, development of multiple acquired shunts, or the presence of congenital PVH. In animals with cellophane bands or ameroid constrictors, high-dose steroids can interfere with fibrous tissue formation and shunt closure.

Animals with clinical signs or biochemical changes that indicate liver dysfunction should be evaluated with ultrasonography, scintigraphy, portography, computed tomography, or magnetic resonance imaging for evidence of shunting. Surgical interventionis recommended for patients with a second shunt or clinical signs related to persistent flow through the original shunt. If shunting is not detected, the most common cause is congenital PVH (MVD-PVH), which can be confirmed by histologic
evaluation of liver biopsy samples.

Prognosis for Congenital Portosystemic Shunts Treated Surgically

In dogs with congenital EHPSS, mortality rates were 2% to 32% after ligation, 7% after ameroid constrictor placement, and 6% to 9% after cellophane banding.

The most common cause of death after PSS attenuation is severe persistent neurologic signs. Other causes include intraoperative hemorrhage, postoperative coagulopathy, portal hypertension,and hemorrhagic gastroenteritis.

In surviving dogs available for follow-up, good to excellent outcomes were noted in 84% to 94% of animals undergoing ligation, cellophane banding, or ameroid constrictor placement for EHPSS.Most dogs continued to have mildly increased bile acids.

In dogs undergoing ameroid constrictor placement, preoperative hypoalbuminemia was associated with persistent postoperative shunting.Preoperative hypoalbuminemia or leukocytosis, occurrence of seizures after surgery, and persistent shunting at 6 to10 weeks after surgery were predictive of poor long-term outcome.

In cats, perioperative mortality rates are 0% to 4% after ligation or ameroid constrictor placement, and 0% to 23% after cellophane banding.Up to 75% of cats have postoperative complications. The most common complication is neurologic dysfunction, including generalized seizures in 8% to 22% and central blindness in up to 44%. Blindness usually resolves within 2 months after surgery.1Of surviving cats available for follow-up, good or excellent long-term outcome was reported in 66% to 75% undergoing ligation, 33% to 75% undergoing ameroid constrictor placement, and 80% undergoing cellophane banding.
Excellent outcome has been reported in 25% of cats with persistent shunting, and continued or recurrent neurologic abnormalities have been reported in 57% of cats with normal scintigraphy or hepatic function tests.

Postoperative care

After surgery, patients are maintained on intravenous fluids until they are eating and drinking. Dextrose is added to the fluids if the blood glucose level is less than 80 mg/dL. Patients are monitored for hypoglycemia, hypothermia, delayed anesthetic recovery, hemorrhage, seizures, and signs of portal hypertension.

Animals usually require opioid analgesics such as buprenorphine for 1 to 3 days. Sedationwith a low dose (0.01–0.02 mg/kg intravenously) of acepromazine may be necessaryif dogs are vocalizing or abdominal pressing, because these activities will increase portal pressure. Acepromazine does not precipitate seizures in dogs with shunts;however, it should not be used in hypotensive animals.

A protein-restricted diet and lactulose are continued after surgery until liver function improves. Bile acids and a biochemical panel are evaluated 2 to 3 months after the surgery. If liver function is normal, medical management is discontinued. Clinical response to diet change can be evaluated in animals with mildly elevated bile acids and normal albumin. If bile acids are moderately increased, medical management is continued and animals are rechecked 5 to 6 months after surgery. If this is all well tolerated, blood count and serum biochemical parameters improve (albumin, BUN, cholesterol, MCV, and glucose), and no return of clinical signs occurs, then the outcome is considered good, regardless of the results of bile acids tests. Many articles on congenital PSS fixation have shown that return of bile acids to normal is not necessarily correlated with long-term outcome.

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