

Clinical presentation, treatment, and outcomes for 15 dogs with two or three congenital extrahepatic portosystemic shunts

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Abstract

Objective: To assess clinical presentation, treatment, and outcomes of dogs with two or three congenital extrahepatic portosystemic shunts (M-EHPSS).

Study design: Multi-institutional retrospective study.

Sample population: A total of 15 dogs with M-EHPSS.

Methods: Medical records were reviewed, and data was collected on dogs with M-EHPSS diagnosed via computed tomography (CT) between 2008 and 2024. Signalment, clinical signs, bloodwork values, postoperative complications, long-term clinical outcome, survival, and owner reported quality of life were recorded.

Results: Approximately half of the dogs (7/15; 47%) were Miniature Schnauzers. Clinical signs and hematologic indicators at presentation were comparable to EHPSS. The mean dog age at diagnosis via CT imaging was 1451 days, and all dogs underwent surgical attenuation (SA) except for one that was ultimately lost to follow up examination. A total of 12 dogs had two shunts, and three dogs had three shunts. Seven dogs did not undergo attenuation of one shunt; these other shunts were either not initially suspected (5/7), the

attenuation device was misplaced (1/7) or surgeon preference to not place attenuation device (1/7). Left gastro-phrenic EHPSS were the only EHPSS not suspected initially. At the time of last follow-up (median: 1253 days), 10/15 dogs were alive. Clinical outcomes were good or excellent in most dogs (12/14; 86%).

Conclusion: These findings suggest that M-EHPSS dogs have a similar prognosis to dogs with standard EHPSS dogs when all EHPSS are identified and treated with appropriate attenuation placement. A potential breed predisposition towards Miniature Schnauzers was identified.

Clinical significance: This information provides guidance for treatment and prognosis of dogs with M-EHPSS.

1 | INTRODUCTION

Congenital extrahepatic portosystemic shunts (EHPSSs) are abnormal blood vessels establishing communication between the portal and systemic circulations.¹ EHPSSs result both in reduced blood detoxification and hypoperfusion (and subsequent synthetic failure) of the liver as the portal vein is responsible for 70%–80% of hepatic circulation.^{2–4} While most of these dogs have a single shunt, some have more than one congenital EHPSS (M-EHPSS) identified during diagnosis or surgery.^{5,6}

The treatment of choice for dogs with single EHPSS is surgical attenuation (SA) via open or minimally invasive surgery⁴ or by intravascular attenuation. Compared to medical management alone, attenuation of EHPSSs is associated with an increased likelihood of resolution of clinical signs and increased median survival time,⁷ as well as improvements in serum bile acid concentrations and increased liver volume.^{8–10} However, with SA, there are also certain conditions such as primary hypoplasia of the portal vein that can lead to portal hypertension resulting in the development of multiple acquired portosystemic shunts^{3,11} (MAPSS). M-EHPSS presents as a small number of shunting vessels (usually 2 or 3), which connect portal to venous blood supply in one of a modest number of characteristic configurations. By contrast, MAPSS presents as numerous and chaotic shunting vessels originating from portal circulation in inconsistent and unpredictable patterns. MAPSS are therefore not typically amenable to routine surgical intervention and therefore need to be distinguished from M-EHPSS in which SA might be indicated. There is currently limited evidence-based guidance for the management of M-EHPSS in dogs.

The purpose of this multi-institutional retrospective study on M-EHPSS was to report the clinical presentation, treatments, and outcomes of dogs with two or three congenital EHPSS. The authors hypothesized that dogs

with M-EHPSS would respond favorably to SA in terms of the resolution of clinical signs, and that a majority of patients would report good or excellent clinical outcomes.

2 | MATERIALS AND METHODS

Data were collected from the medical records of the institutions participating in a separate EHPSS study between 2008 and 2022.¹⁰ Additional dogs identified through 2024 were also included. Dogs diagnosed with 2–3 EHPSS via a computed tomography (CT)-angiography scan were included. Dogs were excluded if evidence of multiple acquired shunts (MAPSS) was found on initial diagnostic imaging.

Data collected from the medical records included signalment, clinical signs associated with M-EHPSS (categorized as neurologic, gastrointestinal, or urinary), and laboratory data collected prior to surgery. These data were also collected at the most recent available follow-up examination. White blood cell (WBC) count, mean corpuscular volume (MCV), albumin (ALB), blood urea nitrogen (BUN), hematocrit (HCT) cholesterol (Chol), and bile acid levels (pre- and post-prandial) were also recorded. Laboratory reference ranges were used in evaluating these parameters.¹²

Diagnostic reports generated at the time of diagnosis were examined, and diagnosis of multiple shunts for each dog was confirmed by CW via review of the CTs. Diagnostic imaging was used to determine the number of EHPSSs present, to categorize and classify each shunt according to the portal and systemic vessels involved, and to report the subjective portal perfusion score.⁵ Portal perfusion scores were evaluated on a scale from 1 to 5,⁵ where a score of 5 corresponds to excellent/normal portal perfusion and a score of 1 corresponds to poor to absent portal perfusion.

Information regarding preoperative medical management was collected. Treatments included the use of

synthetic disaccharides, anticonvulsants, antimicrobials, proton-pump inhibitors, antioxidant hepatoprotectants and protein-restricted diets. The time between the start of medical management and the time of surgical treatment was recorded.

The number and type of shunts attenuated, and the type of attenuation (ameroid constrictor, thin film banding, or suture) utilized was recorded. All intraoperative complications and any concurrent procedures were recorded.

Short-term (within 30 days of SA) and long-term (greater than 30 days after SA) complications were reported, and follow-up examinations were conducted when possible. Complications were considered minor if conservative or medical management alone facilitated resolution, whereas major complications necessitated an additional surgical procedure. For deceased dogs, survival time following treatment was recorded as well as the cause of death when identified. Resolution of preoperative signs associated with M-EHPSS, the development of new clinical signs that developed after SA or medical management of M-EHPSS, and ongoing medical management strategies were investigated during follow-up examination or phone contact. A subjective clinical outcome score was determined based on the criteria described by Mehl et al.¹³

3 | STATISTICAL ANALYSIS

Descriptive statistics were used to analyze the collected data. To evaluate potential correlations between variables, a Chi square test was used to determine the *p*-value. The difference between initial and final bloodwork values is expressed in terms of the percent change from the initial value, and the mean value change was evaluated as the mean of these changes. The last available bloodwork was used for the purposes of comparison. For consistency with the existing literature about retrospective clinical outcomes in dogs with EHPSS,^{13–16} “Excellent” outcomes referred to dogs with resolution of preoperative clinical signs in the absence of medical management, and improvement in bloodwork indicators. “Good” outcomes referred to dogs with absent clinical signs, but medical management continued and/or persistent bloodwork abnormalities, and “poor” outcomes referred to dogs with persistent clinical signs with medical management, or life-threatening complications following surgery.

4 | RESULTS

A total of 15 cases satisfying the inclusion criteria were considered in this study. A total of 13 of these were

previously included in the study of 1082 dogs.⁵ The medical records were obtained from seven participating institutions where these dogs were treated: University of Florida in the US (4 dogs), the Schwartzman Animal Medical Center in the US (3 dogs), the Queen Mother Hospital for Animals in the UK (3 dogs), Nihon University in Japan (2 dogs), Texas A&M University in the US (1 dog), Massachusetts Veterinary Referral Hospital in the US (1 dog), and University of California-Davis, Veterinary Medical Teaching Hospital in the US (1 dog).

At the time of initial clinical evaluation, the mean age of the dogs was 1451 days (median 1019 days, range: 221–4602 days). A total of 10 dogs were female (9 spayed and 1 intact), and five dogs were male (4 castrated and 1 intact). Nearly half of the dogs included in the study were Miniature Schnauzers (7/15; 47%). The remaining dog breeds included mixed breed (*n* = 2) and one each of Bichon Frisé, Yorkshire Terrier, Jack Russell Terrier, Beagle, Poodle, and Toy Poodle. The mean weight at time of presentation was 6.7 kg (median 6.4 kg, range: 2.6–18.8 kg). The mean body condition score of the dogs was 5 out of 9 (median 4.75 out of 9, range: 4–7). Clinical signs were reported at initial examination in 14/15 dogs; these included neurologic signs (14/15), gastrointestinal signs (7/15) and urinary tract signs (5/15).

Initial bloodwork was available for analysis in 14 of 15 dogs (Table 1). All 15 dogs underwent CT imaging; 12 dogs had two shunts, and three dogs had three shunts ultimately identified. The most common shunt configuration was left gastro-phrenic and left gastro-azygos (LGP LGA) which was identified in 10/15 (67%) dogs; 4/7 Miniature Schnauzers had LGP LGA shunts. No relationship between the breed of the dog and the shunt configuration was identified based on the available data (*p* = .46). Other EHPSS configurations were only found in a single dog each (Table 2). The mean subjective portal perfusion score of the dogs in this study was 4.3 (range: 2–5) based on the criteria described by Weisse et al.⁵ In 5/14 dogs, additional shunts were not discovered until a review of CT images was performed by CW and thus not treated (Table 3).

Treatment for the M-EHPSS was performed in 14/15 dogs; 1/15 dogs had no further medical records available after the initial diagnosis and imaging. A total of 12 (12/14; 86%) dogs received preoperative medical management. Of those, 11 received a synthetic disaccharide (lactulose) and protein-restricted diets, eight received an antimicrobial, and two received anticonvulsant therapy (levetiracetam). The most common antimicrobial treatment was metronidazole (7/8) with the remaining dog being prescribed amoxicillin and clavulanic acid. The protein restricted diets included Hill's l/d, Purina HA, Royal Canin Hepatic, and the Just Food for Dogs renal diet. Four dogs received antioxidant hepatoprotectants,

TABLE 1 Change in key biochemical and hematologic parameters from preoperative bloodwork to most recent available follow-up bloodwork for all patients.

Parameter	Mean; median (range) initial	Mean; median (range) follow-up	% Change
HCT (%)	38.4; 42.6 (0.5–56.0)	51.3; 51.0 (45.7–62.7)	+33.7
MCV (fL)	63.3; 63.6 (54.5–70.0)	67.5; 68.0 (64.0–71.0)	+6.7
WBC (K/ μ L)	11.8; 12.1 (7.5–17.7)	9.2; 67.5 (6.1–12.4)	–22.5
Albumin (g/dL)	2.9; 2.9 (2.4–3.4)	3.1; 3.0 (2.4–4.0)	+9.8
BUN (mg/dL)	8.3; 7.0 (2.0–23.0)	8.3; 10.0 (3.0–13.0)	+0.1
Pre-SBA (μ mol/L)	93.7; 86.9 (9.5–226.8)	74.7; 18.3 (1.0–312.9)	–20.3
Post-SBA (μ mol/L)	193.9; 140.5 (36.8–452.6)	82.7; 42.4 (12.4–265.8)	–57.4

Abbreviations: BUN, blood urea nitrogen; HCT, hematocrit; MCV, mean corpuscular volume; Post-SBA, postprandial serum bile acids; pre-SBA, preprandial serum bile acids; WBC, white blood cells.

TABLE 2 Summary of dog data, shunt configurations, and attenuations for study dogs.

Breed	Age at diagnosis (days)	Number of shunts	Shunt configuration*	Shunts attenuated	Notes
Yorkshire Terrier	616	2	LGP LGA	2	
Miniature Schnauzer	1319	2	LGP LGA	2	
Miniature Schnauzer	2031	3	LGP LGA LCLI	2*	Third shunt identified retrospectively, revision for LCLI shunt
Miniature Schnauzer	395	2	LGP LGA	0	Lost to follow-up
Miniature Schnauzer	949	2	LGP LGA	2	
Miniature Schnauzer	253	2	LGP RGP	2*	*Second shunt ligated in surgical revision
Miniature Schnauzer	627	2	LGP LGA	1	Second shunt identified retrospectively
Miniature Schnauzer	4602	2	LGP LGPhA	2	
Beagle	221	2	LGP LGA	1	Second shunt identified retrospectively
Pit Bull Cross	1467	3	LGP LGA LGPhA	2	Third shunt identified retrospectively
Jack Russell Terrier	1089	2	LGP LGA	1	Second shunt identified retrospectively
Terrier Cross	823	3	LGP, spleno-gonadal, spleno-iliac	3	
Bichon Frise	1626	2	LGP LGA	1	
Toy Poodle	1636	2	LGP LGA	2	
Poodle	4133	2	LGP LGA	2	

Abbreviations: LCLI, left colic left iliac; LGA, left gastric azygous; LGP, left gastrophrenic; LGPhA, left gastric phenecoabdominal; RGP, right gastrophrenic.

*Achieved the “shunts attenuated” number via revision.

and two dogs received proton pump inhibitors. The median duration of preoperative medical management was 26 days (range: 9–75 days).

Exploratory laparotomy was performed in 14/14 dogs for which medical records were available. For all five

dogs in which one EPHSS was not diagnosed via CT except during retrospective analysis, the untreated EHPSS was a left gastro-phrenic (LGP) EHPSS. Shunt attenuation was performed with ameroid constrictors (5 dogs), polypropylene suture ligation (4 dogs), thin film

TABLE 3 Description of cases for which all present shunts were not initially identified during initial SA.

EHPSS identified on initial CT	EHPSS initially attenuated	EHPSS identified on repeat CT	EHPSS attenuated on revision surgery	EHPSS identified retrospectively
LGA	LGA	-	-	LGP LGA
LGA + LGPhA	LGA + LGPhA	(a)LGA (a)LGPhA	-	LGP LGA LGPhA
LGA	LGA	(a)LGA	-	LGP LGA
LGA	LGA	-	-	LGP LGA
LGA LCLI	LGA LCLI	(a)LGA LCLI	LCLI	LGP LGA LCLI

Abbreviations: CT, computed tomography; EHPSS, extrahepatic portosystemic shunts; LCLI, left colic left iliac; LGA, left gastric azygous; LGP, left gastrophrenic; LGPhA, left gastric phenecoabdominal; SA, surgical attenuation.

Shunt description with the lowercase "a" i.e., (a)LGP refers to attenuated shunting vessels identified on postoperative CT.

banding (4 dogs), and complete silk suture ligation (1 dog). All dogs underwent the same type of attenuation (ameroid constrictor, thin film banding, suture, etc.) for all attenuated shunts. All 14 dogs had liver biopsies performed at the time of EHPSS attenuation. Four of 14 dogs had concurrent cystotomies and 2/14 had concurrent ovariohysterectomies. One (1/14) dog had a concurrent splenectomy because of splenic laceration during SA.

Major intraoperative complications were observed in two dogs. One dog experienced splenic laceration and underwent a concurrent splenectomy, and another dog experienced an intestinal perforation during the approach to the celiotomy due to significant adhesions to the abdominal wall and underwent concurrent intervention (Table 4).

One (1/14; 7%) dog, which underwent complete attenuation of an LGA shunt via suture and partial attenuation of an LGP shunt via suture, developed post-attenuation neurologic syndrome¹ (PANS) and died approximately 31 h postoperatively. No other major complications occurred within 30 days of surgical intervention. Three dogs experienced minor short-term complications. One experienced hypothermia, hypotension, hypoproteinemia, and anemia due to the previously mentioned splenic laceration in surgery. One dog that underwent a concurrent cystotomy experienced regurgitation and intermittent hematuria that were treated with metoclopramide and erythromycin that resolved within 4 days of the dog's surgery. One additional dog experienced intermittent vomiting postoperatively, for which no treatment or date of resolution was recorded.

Histopathology reports were available for 12/14. Each available report noted findings consistent with the presence of EHPSS. Common findings were arteriolar duplication/hyperplasia (11/12), lipogranulomas (8/12), and lobular atrophy (7/12). No additional hepatic pathology was noted in any of these dogs. Portal venules were noted at the portal triad for all dogs, but in 5/12 dogs either

hypoplasia (3/5) or a decreased number (2/5) of the portal venules within the portal triads were noted.

Repeat diagnostic imaging was performed in seven of the 13 living dogs. The reasons for repeat imaging were not reported in all dogs; however, of the seven dogs that underwent follow-up imaging, 3/7 had clinical signs, and 1/7 had persistently increased bile acids. Imaging included abdominal ultrasound (1 dog) and CT (6 dogs). Evidence of residual shunting was noted in 5/7 (71%) of the dogs that underwent follow-up imaging. Four of these dogs had residual flow through an attenuated EHPSS and one was noted to have developed MAPSS via CT imaging. Dogs with confirmed portal venous shunting after previous surgery had undergone thin film banding (2/4), ameroid constrictor attenuation (1/4), and suture attenuation with 3-0 silk (1/4). The dog undergoing silk attenuation demonstrated apparent failure of the initial ligature on follow-up two years after initial ligation. The dog that was found to have developed MAPSS had undergone ameroid constrictor shunt attenuation. The remaining two dogs (2/7; 29%) had one EHPSS undetected at the initial surgery. There was no correlation between cases for which an EHPSS was initially not detected and dogs for which evidence of residual shunting was identified on follow-up examination. Two dogs with evidence of residual EHPSS blood flow had no SA of the persistent shunts.

For five of the 14 SA dogs (35.7%), one EHPSS was not identified (nor treated) until retrospective analysis by CW. For three of these dogs, a single LGA EHPSS was identified and treated, for one of these a LGA EHPSS and a left gastro-phrenicoabdominal (LGPhA) EHPSS were identified and treated, and for one dog an LGA and left colic left iliac (LCLI) EHPSS were identified and treated. In all five of these cases, an LGP EHPSS was missed at the time of initial workup. Only two of these dogs (the dogs with the LGP-LGA, LGPhA and LGP LGA LCLI EHPSS) had follow-up CT imaging performed. Both of those dogs exhibited evidence of residual shunting on follow-up CT. However, none of the five dogs underwent

TABLE 4 Summary of concurrent procedures, surgical complications, short-and long-term complications, and outcome scores for dogs considered in the study.

Breed	Number of shunts	Concurrent procedures	Intraoperative complications	Long-term complications	Short-term complications	Outcome (score)
Yorkshire Terrier	2	Cystotomy	N/A	Revision SA, MAPSS	Regurgitation, intermittent hematuria	Poor
Miniature Schnauzer	2	N/A	N/A		N/A	Good
Beagle	2	Ovariohysterectomy	N/A		N/A	Good
Miniature Schnauzer	3	N/A	N/A	Revision to attenuate LCLI shunt	N/A	Good
Miniature Schnauzer	2	N/A	N/A	N/A	N/A	N/A
Miniature Schnauzer	2	N/A	N/A		N/A	Good
Pit bull cross	3	Enterotomy	Small intestine perforation		Intermittent vomiting (suspect bilious)	Good
Miniature Schnauzer	2	Cystotomy, ovariohysterectomy	N/A	Revision to attenuate LGA shunting	N/A	Good
Miniature Schnauzer	2	Splenectomy	Splenic laceration		Hypothermia, hypotension, hypoproteinemia, anemia	Excl
Miniature Schnauzer	2	Cystotomy	N/A		N/A	Excl
Jack Russell Terrier	2	N/A	N/A		N/A	Excl
Terrier Cross	2	Cystotomy	N/A		N/A	Good
Bichon Frise	2		N/A		N/A	Excl
Toy Poodle	2	N/A	N/A		N/A	Good
Poodle	2	N/A	N/A		Cardiopulmonary arrest	Poor

Abbreviations: Excl, excellent; LCLI, left colic left iliac; LGA, left gastric azygous; MAPSS, multiple acquired portosystemic shunts; N/A, not available; SA, surgical attenuation.

revision surgery to address these missed shunts. The dog with LGP LGA LCLI EHPSSs did undergo revision surgery, but this revision did not address the dog's LGP EHPSS.

Revision surgery was performed in three dogs, and these were considered major long-term complications (3/14; 21%). One dog underwent revision surgery due to the MAPSS, but during the exploratory laparotomy, significant portal hypertension was confirmed and no corrective action was taken. This dog (with MAPSS) had a subjective portal perfusion score of 5 at the time of the initial CT scan. One dog had SA performed for a single EHPSS even though two shunts had been identified on

preoperative CT. Revision surgery was performed during which an ameroid constrictor was placed on another shunting blood vessel. The third dog, which had a third shunt missed on initial workup as discussed above, underwent CT imaging due to persistently elevated bile acids. The repeated CT scan found persistent LCLI shunting despite initial attenuation of two EHPSS with thin film banding. A thin film banding attenuation was then performed on the additional EHPSS.

Long-term follow up information was available for all (13/14 [93]) surviving dogs that underwent M-EHPSS treatment. The median time to follow-up evaluation was 1253 days (mean 1710 days, range: 40–4097 days).

Paired initial and follow-up bloodwork results were available for 8/13 dogs (Table 1). Two dogs were found to have significantly elevated pre- and post-prandial serum bile acids both before and after surgery, one of which was retrospectively identified as a dog with a second, untreated shunt. These two patients each had postprandial bile acid concentrations greater than 180 $\mu\text{mol/L}$ and post-prandial bile acid concentrations greater than 170 $\mu\text{mol/L}$ at last follow-up. The median change in pre- and post-prandial bile acids was -53.5% and -56.0% , respectively for cases with EHPSS previously not identified, and -67.5% and -75.7% , respectively in dogs without unidentified shunts.

Two (2/13; 15%) dogs were noted to have clinical signs that may have been associated with EHPSS at the time of last follow-up examination. One dog, which had all shunts attenuated, but which developed MAPSS postoperatively and was not amenable to revision SA, was reported to have exhibited vomiting, intermittent suspected melena, and seizures and was placed on a postoperative medical management protocol that failed to

eliminate the clinical signs. A second dog's owner indicated potential mild polyuria/polydipsia in the long-term follow up questionnaire but stated that the dog was not on any medical management protocol and did not experience any other clinical signs. This dog was one of the four dogs included in this study whose second EHPSS was identified retrospectively and never attenuated.

Preoperative signs resolved in 10/13 dogs, and one dog was subclinical before attenuation. At the time of most recent follow-up, 9/13 dogs were on medical management protocols. These included protein-restricted diets (8/13), anticonvulsants (2/13), antimicrobials (2/13) and synthetic disaccharides (2/13). Clinical outcome was excellent in 3/14 dogs, good in 9/14 dogs, and poor in 2/14 dogs. All five dogs for which a second EHPSS was only found retrospectively (and therefore underwent only selective attenuation of their M-EHPSS) reported good or excellent qualitative clinical outcomes. Figure 1 summarizes the overall patient population for this study from initial diagnosis to final clinical outcome.

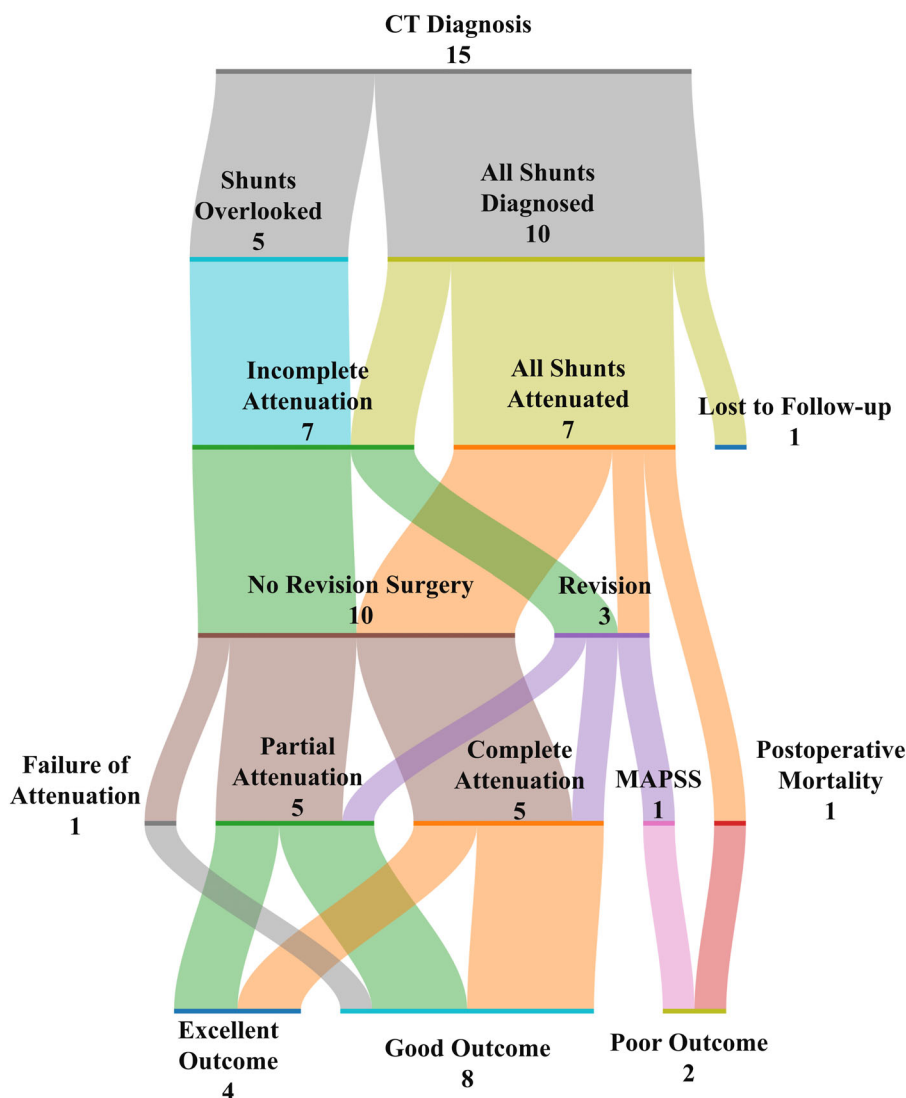


FIGURE 1 Summary of patient treatment and outcomes.

Three dogs (3/13; 23%) were known to have subsequently died; two of these dogs were euthanized, and neither dog's death was suspected to be related to the EHPSSs. The circumstances of the third dog's death are unknown, but it occurred approximately nine years after M-EHPSS treatment.

5 | DISCUSSION

This study specifically evaluated dogs with two or three congenital EHPSS. M-EHPSS patients were not found to have worse expected prognosis than EHPSS patients. The LGP-LGA shunt configuration was most common, and Miniature Schnauzers represented nearly half of M-EHPSS patients, but no association was found between the two.

Patients were identified via diagnostic imaging by the authors, and only one of the six observed configurations was found in more than one dog. Of the 1082 EHPSS dogs considered in the original study,⁵ 12/1082 (1.1%) had two EHPSS identified and 1/1082 (0.1%) had three EHPSS identified. This infrequency may explain the lack of available literature regarding M-EHPSS which consists of a small number of case reports.^{6,17} The present study aimed to analyze the available data for M-EHPSS dogs to examine expected clinical signalment, surgical outcomes, and expected prognosis, as well as provide insights to direct future research.

Dogs in this study were diagnosed later in life and demonstrated relatively moderate clinical signs compared to other dogs with EHPSS in the literature.^{5,6} Additionally, the mean subjective portal perfusion score (PPS) for the present cohort of dogs (mean 4.25) was higher than the study from which many were identified (mean 3.34)⁵; that study demonstrated dogs with better subjective PPS were generally diagnosed at a later age. Female dogs were more commonly affected than male dogs, as has been reported to be the case for EHPSS.^{5,18} Despite the dogs' older mean age at the time of diagnosis, the present literature still indicates that surgical intervention is preferred over MM alone.¹⁹

Miniature Schnauzers have been identified as predisposed to EHPSS in prior literature.^{7,18,20} The incidence of Miniature Schnauzers in the present cohort was significantly greater (46.7%) than in previous studies of EHPSS with larger sample sizes^{5,18} (10.3%). No other breed exhibited similar trends in the present study. While the present study only included a small number of dogs, the incidence of Miniature Schnauzers in the present dataset was noteworthy. Considering only the dogs extracted from the analysis of 1082 dogs,⁵ approximately 6% of Miniature Schnauzer EHPSS dogs had M-EHPSS.

The authors recommend that clinicians with Miniature Schnauzer EHPSS dogs pay specific attention to the possibility of M-EHPSS, especially for dogs with persistently elevated bile acids after SA.

The clinical signs and serum biochemical indicators of the dogs in this study were in line with previous studies of EHPSS dogs.^{13,20–22} The preoperative data available for the present M-EHPSS dogs did not reflect more severe or more numerous clinical signs or diagnostic indicators than would be expected from single EHPSS dogs.

This study considered M-EHPSS dogs together regardless of individual shunt configuration. Of the 15 cases analyzed here, 10 had left gastric-phrenic and left gastric-azygos shunts (LGP-LGA). For each other EHPSS configuration present, only one example was identified in the dataset. It has been noted in the prior literature that the two most common EHPSS configurations in dogs with a single EHPSS were LGP and LGA.⁵

Of the dogs considered in the study, 5/14 (35.7%) had EHPSS that were not identified during their initial course of diagnosis and treatment and therefore did not undergo surgical attenuation. Of the dogs whose EHPSS were initially missed, all five had an LGA shunt attenuated and an LGP shunt that went undiagnosed/untreated. Of all the dogs that presented with LGP and LGA shunts, 45.5% (5/11) had their LGP shunt missed on initial workup. It was observed in the present cohort of dogs that the dogs with undetected EHPSS showed less improvement in bile acid results than those without. Undetected/untreated shunts may be a contributing factor to observations of persistently elevated bile acids in EHPSS dogs after SA in the literature.²³ Dogs whose EHPSS were only identified retrospectively, and thus still had untreated EHPSS when follow-up bloodwork was taken, tended to exhibit less improvement in bloodwork values as compared to dogs whose EHPSS were all attenuated at follow-up. Clinical records do not provide insight into the reasons LGP shunts in particular were overlooked. Possible, though speculative, explanations include the possibility that LGP shunts are generally located cranial to other common shunting sites, and that practitioners exhibit a premature closure bias which leads them not to seek out more shunting vessels once one has been identified. Further retrospective studies would be needed to examine this phenomenon specifically and develop better-supported hypotheses to explain it.

Two dogs underwent SA for one EHPSS even though two EHPSS had been identified on CT at the time of treatment. For one of these cases, SA via an ameroid constrictor was performed for the dog's LGA shunt, and a LGP shunt was left intact. The reason for this was not clear from the dog's medical records. It is possible that attenuation of a single EHPSS was chosen by the

surgeon, or perhaps attenuation of both shunts at the level of the left gastric vein was initially attempted. Proper placement of an attenuation device is also a consideration noted in the literature for single EHPSS dogs.²⁴ Because of the possibility of persistent shunting due to collateral blood supply, best practices for EHPSS treatment are to attenuate shunts immediately adjacent of their entrance into systemic circulation when possible.^{1,23,25} Medical records for these patients do not specify if additional confounding factors, such as a shunt placement that would require a more invasive approach to SA, were present for these patients.

Silk suture has been considered a viable option for EHPSS ligation²⁶ due to its ease of handling and knot security. However, the present study identified one dog in which silk ligation was used, and in which follow-up CT two years after SA found failure of ligation of the EHPSS despite complete initial ligation. Successful ligation of both shunts was confirmed in this patient via the measurement of pre- and post-ligation portal pressures, suggesting a failure of the ligation rather than incorrect placement of the ligature. While silk suture is classified as non-absorbable it can be degraded via proteolysis by matrix metalloproteinases,^{26,27} which are upregulated during inflammatory states. EHPSS dogs are known to be prone to exhibit elevated markers of inflammation, for which multiple explanations have been examined in the literature.^{1,28} Biodegradation of silk suture is reported to take 2 years,²⁷ and the failure of ligation in this case was known to have occurred within two years of initial SA. Because failure was only observed in one instance and the mechanism was unknown, follow-up studies would be required to evaluate this possibility. Pending further study, ameroid constrictor, thin film banding, or “more” permanent suture material attenuation which have been used widely and have been found to be equally efficacious^{29,30} might be considered over silk.

The present study found that 21% (3/14) of dogs had an excellent clinical outcome and 64% (9/14) were found to have a good outcome; however, many remained on some type of medical management. Due to the retrospective nature of the study, the necessity of postoperative medical management could not be determined in all dogs. Therefore, it is likely that owner preference led to some dog outcomes that might have been classified as excellent to be classified as good. To account for this possibility, and following the convention outlined in previous retrospective studies,^{14,16} good and excellent outcomes were grouped when comparing the present data to prior literature. The present study found that 86% (12/14) of dogs had good or excellent outcomes, and 14% (2/14) of dogs had poor outcomes. Ameroid constrictor

attenuation has been found to yield a good or excellent clinical outcome in 84% of dogs¹⁴ and thin film banding has been reported to have 82% (34/42) good or excellent outcomes.¹⁴ Complete suture ligation has been reported to produce good or excellent outcomes achieved in 100% (20/20) of dogs, and partial suture ligation has been reported to produce good or excellent outcomes in 90% (9/10) of dogs.¹⁶ The majority of dogs undergoing surgery in this study underwent either ameroid or thin film band attenuation (9/14) as opposed to polypropylene or silk suture ligation (5/14); the reported surgical outcomes for M-EHPSS were found to be in line with the existing literature for single EHPSS treated using the same techniques. This finding is particularly noteworthy because five of the dogs in this study did not actually undergo complete attenuation of all shunts due to missed LGP shunts at the time of diagnosis. Prior literature reports an association between shunting vessels with phrenic involvement (such as LGP) and reduced severity of EHPSS clinical signs.³¹ The mechanism underlying this association has been suggested to be periodic compression of the shunting vessel by the diaphragm.¹ The present findings may support this prior finding, though patients with unattenuated shunts exhibited less improvement in biochemical indicators than those that underwent complete attenuation. These dogs did not exhibit worse subjective clinical outcomes, and stronger conclusions could be drawn if cases in which medical management or selective attenuation (leaving some shunts open) was pursued deliberately were identified for comparison.

In conclusion, the present study examined the medical records of M-EHPSS patients to identify notable trends in demographics and compare clinical outcomes to EHPSS patients. A potential predisposition of Miniature Schnauzers to M-EHPSS was identified. It also found that the LGP-LGA shunt configuration was the most common M-EHPSS configuration and LGP shunts were the most likely to be undetected on initial workup. Several dogs included in the present study were not treated for all their EHPSS because at least one EHPSS was not identified during the course of the dog's treatment. These findings suggest practitioners diagnosing EHPSS dogs be mindful of the possibility of M-EHPSS, especially in Miniature Schnauzer dogs. Based on the dogs examined in the present study, there was no evidence to suggest that the presence of M-EHPSS is a negative prognostic indicator as compared to singular EHPSS. Similarly, there does not appear to be a correlation between the number of EHPSS present and the severity of clinical signs and biochemical parameters at presentation. Surgical outcomes were found to be similar with singular EHPSS cases, even

in cases in which an LGP shunt was not attenuated. The present study supports the use of EHPSS best practices for the treatment of M-EHPSS dogs in general. However, these findings also support the use of clinical judgment in cases where attenuation of LGP shunts would require surgical revision. Further research should be undertaken to clarify the circumstances in which good clinical outcomes are likely even without attenuation of all shunts.

AUTHOR CONTRIBUTIONS

Vogel J, DVM: Study conception and design, collection of initial and follow-up data on subjects, responsible for majority of data analysis, writing, and figure image preparation of submitted manuscript. Weisse C, VMD, DACVS: Study conception and design, contributed cases (3), review of computed tomography images for all subjects, collection of initial and follow-up data, review of data analysis, manuscript writing, figure image preparation, and final approval of submitted manuscript. Culp WTN, VMD, DACVS: Contributed case (1), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. Vilaplana Grosso FR, DVM, DECVDI, DACVR: Contributed cases (4), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. Asano K, DVM, PhD, DJCVS: Contributed cases (2), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. Lipscomb VJ, MA, VetMB, DECVS: Contributed cases (3), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. Amato NS, DVM, DACVS (Small Animal): Contributed case (1), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. Davidson JR, DVM, MS, DACVS, DACVSMR: Contributed case (1), collection of initial and follow-up data, manuscript revision, and final approval of submitted manuscript. All authors are accountable and in agreement regarding the accuracy and integrity of this work.

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CONFLICT OF INTEREST STATEMENT

The authors declare no financial or other conflict of interest with this study related to a company or product used in the report.

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