Retrospective Review of 27 Cases of Congenital Portosystemic Shunt in Dogs from 2015 to 2023

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Abstract:
The study aims to conduct a retrospective review of 27 cases of a congenital portosystemic shunt in dogs from 2015 to 2023, providing insights into classification, clinical manifestations, diagnostics, anaesthesia, surgery, and perioperative care. Initiation of medical therapy at least 14 days before surgery aims to alleviate clinical signs and mitigate anaesthesia and surgery risks, emphasising a low-protein diet and lactulose to trap ammonium ions in the intestinal lumen and was strictly followed in all patients. Changes in drug uptake, metabolism, and excretion should be considered when choosing an anaesthesia protocol. All dogs, except one with absent portal vein, underwent cellophane placement, facilitating gradual shunt closure. All dogs recovered uneventfully from surgery without evidence of portal hypertension and showed clinical improvement after that.

Keywords: Portosystemic shunts, dogs, cellophane attenuation, perioperative preparation

1. Introduction

A portosystemic shunt (PSS) in dogs is an abnormal connection between the portal vascular system and the systemic circulation (Buttler et al., 1990; Watson and Hertage, 1998; Mullins, 2019a). This condition diverts blood from the abdominal organs, bypassing the liver, which filters and detoxifies the blood before it returns to the heart (Sleight and Thomford, 1970; Tieman Mankin, 2015; Bertolini, 2019).
PSS are classified into acquired and congenital. Congenital PSS (CPSS) result from abnormalities in the development of the portal venous system or foetal vessels and are divided into two categories: extrahepatic and intrahepatic (Bertolini, 2019; Mullins, 2019a). CPSS predominantly enter the prehepatic caudal vena cava (Mathews and Gofton, 1988) at the epiploic foramen, termed portocaval shunts (Mullins, 2019b). Portophrenic shunts entering the left hepatic vein or caudal vena cava at the diaphragm level exhibit milder symptoms than other shunts (Berent and Tobias, 2018). Portoazygos shunts, typically involving the left gastric vein, occasionally the right gastric vein, transverse the diaphragm through the oesophageal hiatus and enter the azygos vein in the thorax (Harari et al., 1990). Acquired PSS are commonly caused by portal hypertension resulting from liver cirrhosis or liver failure (Boothe et al., 1996; Tieman Mankin, 2015; Berent and Tobias, 2018).

Breeds at an increased risk of extrahepatic CPSS are Cairn and Yorkshire Terriers, Maltese, Tibetan spaniels, miniature Schnauzers, Shih Tzu, Poodles (Tobias and Rohrbach; 2003), Pugs and Dachshunds (Wolschrijn et al., 2000; Tobias, 2003).
The clinical manifestations of CPSS lack specificity and may fluctuate over time, impacting the central nervous system, gastrointestinal system, and urinary tract (Konstantinidis et al., 2023). General signs in dogs include poor weight gain and stunted growth (Tobias, 2012). Hepatic encephalopathy and seizures may also be seen in PSS with the signs of hepatic encephalopathy, which are ataxia, depression, changes in behaviour, circling, disorientation, head pressing, blindness, seizures, and coma. In about 30% of dogs, vomiting, diarrhoea, anorexia, and salivation occur (Howe and Boothe, 2002; Konstantinidis et al., 2023). Clinical signs of the urinary system are associated with ammonium biurat crystals and urate calculi and include dysuria, hematuria, pollakiuria, stranguria and decreased urine specific gravity (Howe and Boothe, 2002; Broome et al., 2004; Tobias, 2012; Caporali et al., 2015). In dogs, PSS can decrease the levels of various blood parameters such as total protein (TP), albumin, blood urea nitrogen (BUN), total cholesterol, and glucose. Elevated ammonia levels and serum bile acid are notable and serve as reliable indicators for diagnosing PSS in dogs (Ruland et al., 2010). Leucocytosis may be evident (Winkler et al., 2003), and mild to moderate microcytic normochromic nonregenerative anaemia is often observed alongside (Tobias, 2012; Tieman Mankin, 2015).

Surgical intervention is widely acknowledged as the preferred treatment for PSS, given that medical treatment alone cannot address the underlying vascular anomaly. The primary advantage of medical therapy lies in its ability to minimise ammonia absorption from the gastrointestinal tract, aiming to prevent hepatic encephalopathy and seizures. The increased ammonia concentration in plasma could also be attributed to bacterial degradation of amino acids in the gastrointestinal tract (Self, 2016). Before undergoing anaesthesia, dogs with PSS should undergo medical stabilisation (Dugdale et al., 2020).
Dogs with PSS are often young, and to avoid the risk of hypoglycemia, food should only be withheld four to six hours prior to anaesthesia (Self, 2016). Dogs with PSS require minimal premedication with opioids, induction with propofol or alfaxalone, and anaesthesia should be maintained with isoflurane or sevoflurane (Self, 2016; Dugdale et al., 2020). Low doses of alpha-2-adrenoceptor agonists can also be used for premedication, and opioids should be continued during surgery (Self, 2016). Nonsteroidal anti-inflammatory drugs should be avoided due to coagulopathies and hypoproteinaemia, and caution is advised also with paracetamol application because of hepatic insufficiency (Dugdale et al., 2020). Notably, due to their small body size and reduced metabolic heat production by the small liver, dogs with PSS are particularly prone to hypothermia (Self, 2016). Intravenous administration of Hartmann's solution is necessary during both the surgical procedure and the recovery period. Continuous monitoring of blood glucose levels and body temperature

is imperative during recovery, with particular attention to observing seizures. Seizures can be effectively managed by addressing metabolic disturbances such as hypoglycemia and elevated ammonia levels. Additionally, pharmaceutical options, including levetiracetam, phenobarbitone, propofol, or low doses of dexmedetomidine or medetomidine, are viable choices for seizure control (Dugdale et al., 2020). Post-surgery, ensuring adequate analgesia is essential, and opioids represent a recommended approach for managing pain (Self, 2016).

2. Materials and methods

Patient data regarding CPSS were collected from medical records at the Veterinary Faculty in Ljubljana, Slovenia, covering the period from 2015 to 2023. Out of the 27 dogs included in the study, detailed medical history and laboratory results were available for 23 dogs, while CT scans were available for 25. However, due to the retrospective nature of the study, complete data retrieval was not possible for all 27 dogs (Erjavec et al.,2024). Additionally, over one-third of the dogs were referred from other clinics and were under the care of their primary veterinarian after surgery; thus, complete data for these dogs were also not available. Since this study involved the review of existing medical records and did not involve any new interventions or procedures on animals, formal ethical approval was not required.

All dogs underwent surgical treatment, and among the 27 surgically treated patients, two (a Golden Retriever and a mixed breed dog) were found to have an intrahepatic PSS. The diagnosis was established based on clinical signs, laboratory findings (bile acids, blood ammonia, complete blood count (CBC) with differential, urea, creatinine, albumins, TP, potassium, sodium and chloride, glucose, ALT (alanine aminotransferase), urinalysis and confirmed with computed tomography (CT).

At least 14-day preparation regimen was implemented with the following oral therapies: lactulose (Portalak 667 mg/ml, Belupo, Koprivnica, Croatia) at a dose of 0.5 ml/kg, metronidazole 10 mg/kg/12h (Metrobactin, Dechra LelyPharma BV, Lelystad, Nederland), gastroprotective drugs - esomeprazole (Nexium, Grünenthal GmbH, Aachen, Germany), hepatoprotective (Epato 1500 plus, DRN s.r.l., Italy, α IT000187AL, marketing@drnsrl.it) and ursodeoxycholic acid (Ursofalk 250 mg, Dr. Falk Pharma GmbH, Freiburg, Germany).

Additionally, a low protein diet (Hill's k/d) was administered, and on the day before surgery subcutaneous vitamin K (Konakion MM, Phytomenadione 10 mg, Cheplapharm, Arzneimittel GmbH, Ziegelhof, Greifswald, Germany) at a dose of 5.0 mg/kg was given first day and continued 2 days after surgery at a dose of 2.5 mg/kg. The dogs that already had epileptic seizures received antiepileptic drugs phenobarbitone 1–4 mg/kg/12h (Epiphen 30mg, Vetoquinol, Buckingham, UK) and/or levetiracetam 20 mg/kg/8h (Keppra 100 mg/ml, UCB Pharma SA, Brussels, Belgium). Hospitalisation began a day before the scheduled surgery to facilitate preparation and blood examination (CBC with differential, urea, creatinine, albumins, TP, potassium, sodium and chloride, glucose, ALT, PT (prothrombin time), APTT (activated partial thromboplastin time). On the morning of the surgery, the dogs received their complete prescribed therapy, including lactulose, metronidazole, vitamin K, intravenously pantoprazole (Nolpaza, Krka, Novo mesto, Slovenia) and dogs with seizures antiepileptic drugs. All medicine except vitamin K was continued at least 2 weeks after surgery, i.e., to the first check-up, and then gradually discontinued according to the clinical signs and laboratory results.

Induction of anaesthesia was performed with analgetic fentanyl (Fentanil Torrex 50 µg/ml, Chiesi Pharmaceuticals GmbH, Wien, Austria), 2 µg/kg or remifentanyl (Remifentanil-hameln 1 mg, Siegfried Hameln GmbH, Hameln, Germany) 2 µg/kg and propofol (Propoven 10 mg/ml, Fresenius Kabi Austria GmbH, Graz, Austria) slowly to effect, 2–3mg/kg intravenously. Anaesthesia was maintained with isoflurane in oxygen, and constant rate infusion (CRI) of fentanyl (2 µg/kg/h) or remifentanil (5 µg/kg/h) was administered during surgery and continued as CRI two to three days after surgery. During anaesthesia lactated ringer solution (Hartmannova raztopina Braun, B. Braun Melsungen AG, Melsungen, Germany) 5 ml/kg/h was given, blood glucose levels were monitored at 30-minute intervals and monitoring of arterial blood pressure was assessed using a

Doppler flow detector (Model 811-BL, Parks Medical Electronic), end-tidal CO_2 concentration, oxygen saturation measured with a lingual SpO_2 probe, body temperature with an oesophageal thermometer, and electrocardiogram (ECG) was continuous.
In all animals with PSS, a ventral medial celiotomy was performed, PSS was identified, and a cellophane band was applied in all dogs except for one dog where the portal vein was absent. Before use, 1.2 cm wide strips of cellophane were prepared and sterilised. After taking them from the sterilisation pouch, cellophane strips were immersed in saline, and then one strip was folded longitudinally into 3 layers. Initially, the tissue around the shunt was carefully dissected with right-angled dissecting forceps, and subsequently, the cellophane strip was carefully passed around the shunt. Three to four titanium clips were used in alternating directions to fixate both ends of the cellophane, and the remaining parts of the cellophane were cut with scissors. Shunts were partially or not at all attenuated at the end of surgery. The abdomen was closed routinely in all dogs. Dogs were typically hospitalised for up to 3 days following surgery after which they underwent check-ups at 7-14 days post-surgery, followed by monthly appointments for 3-4 months thereafter. Further follow-up appointments were scheduled as needed based on the presence of clinical signs.

3. Results

The inclusion criteria for selecting cases encompassed a thorough review of medical records pertaining to 27 dogs, operated by the same surgeon (VE), comprising 18 females and 9 males, representing various breeds such as mixed breed (6), Yorkshire Terrier (5 dogs), Pug (2), Maltese (2), Shih Tzu (2), Jack Russell Terrier (2), Miniature Schnauzer (2), and one each of Bearded Collie, Chihuahua, Cavalier King Charles Spaniel, West Highland White Terrier, Golden Retriever, and Pomeranian. The age range of the dogs on the day of the operation was 4 to 100 months, with twelve out of 27 dogs being younger than 12 months. Dogs were presented with various clinical signs, 19/23 dogs were apathic, 14/23 dogs were vomiting, neurological signs such as seizures, disorientation, ataxia, restlessness, blindness, and head pressing were found in 13/23 dogs, 7/23 had polydipsia/polyuria, 6/24 had diarrhoea, and 2/23 were salivating.

On the day of surgery or a few days before blood analysis was performed, serum bile acids were elevated in all dogs (22/22), ALT was elevated in 13/25 dogs, urea was low in 17/25 dogs, PT was prolonged in 8/24, and APTT was prolonged in 15/24 dogs. Nephroliths or uroliths were found in 13/26 dogs. Ammonia was elevated in 8/8 dogs. Leucocytosis was found in 16/24 dogs. Eleven dogs from 25 (11/25) were hypoproteinaemic and hypoalbuminemic, 6/25 were anaemic with low red blood cells and low haematocrit. Before the operation, two dogs needed a transfusion. During anaesthesia, 9 of 25 dogs needed a bolus of 20% glucose (Glukoza Braun 200 mg/ml, B. Braun Melsungen AG, Melsungen, Germany) 1-2 ml/kg intravenously. Twenty out of 25 dogs required Voluvene for maintenance of blood pressure or due to hypoproteinemia (Voluven 60 mg/ml, Fresenius Kabi Deutschland GmbH, Bad Homburg, Germany) administered either through CRI at 1–2 ml/kg/h or as a bolus (1–2 ml/kg) in case of observed hypotension. This paper does not present results from potassium, sodium, chloride, venous blood gas analysis, and urinalysis.
Based on CT angiography images of 22 dogs with extrahepatic shunts, the distribution included right gastrocaval shunts in 7 dogs (32%), each of spleno- and portocaval shunts in 3 dogs (14%), splenoazygos and left gastroazygos shunts in 2 dogs each (10%), and singular occurrences of gastrophrenic, left gastric, left gastrocaval, gastroduodeno caval, and right gastrocaval with a caudal loop shunt in one dog.

On the day after surgery, dogs required intensive care, and vital signs, including body temperature, pulse rate, respiration, urine production, capillary refill time, mucous membrane colour, arterial blood pressure, and blood glucose, were recorded hourly and monitored for neurological signs. Monitoring urine production and/or blood pressure was also undertaken to adjust postoperative fluid therapy. Fluid therapy using lactated Ringer's solution was given at 3 – 5 ml/kg/h. The dogs received intensive medical attention

until they regained the ability to move and independently consume a regular diet. Analgesia was maintained as described before, with the addition of methadone if necessary. Throughout the recovery phase, no indicators of portal hypertension, such as hypovolemic shock, progressive hypothermia, and severe abdominal pain (Tobias, 2012), were observed. We do not have uniform results after surgery because the dogs underwent check-ups at different times and with various veterinarians who did not monitor the same blood parameters. However, we found that bile acids were elevated in 12/16 dogs, ALT was elevated in 6/20 dogs, urea was low in 2/13 dogs, TP was low in 2/18 dogs, and albumins were low in 3/21 dogs (Erjavec et al., 2024).

4. Discussion

The investigation of PSS in dogs provides valuable insights into the pathophysiology, clinical presentation, diagnosis and outcomes following surgical intervention. The clinical signs that raised suspicion of portosystemic shunt (PSS) in dogs in our study were primarily neurological and gastrointestinal, with a notable prevalence of apathy among the affected dogs. Notably, salivation, a more common clinical sign in cats than in dogs (Howe and Boothe, 2002), was also reflected in our study. We primarily confirmed the suspicion with results indicating elevated bile acids, ammonia levels and urinalysis, while the diagnosis was subsequently confirmed through CT imaging. Preoperative management is crucial in minimising ammonia absorption from the gastrointestinal tract, preventing hepatic encephalopathy and seizures in dogs, and enhancing postoperative recovery. When ammonia was measured, it was increased in all dogs. Although ammonia is a valuable parameter, we did not measure it in all dogs due to the complex process of sample preparation and transport to an external laboratory.

While coagulation times are frequently prolonged in affected dogs, they generally do not have significant clinical implications and spontaneous bleeding is uncommon (Kummeling et al., 2006; Kelley et al., 2013) which was consistent with our study where coagulation times were prolonged, but no issues with bleeding during surgery occurred, which we attribute to the administration of vitamin K. Following PSS occlusion, gastrointestinal ulceration and gastritis may persist years after surgery in majority of dogs, prompting consideration on proton pump inhibitors, especially for those receiving NSAIDs post PSS surgery (Weisse et al., 2014; Dugdale et al., 2020).

In cases of hypoalbuminemia, caution should be exercised regarding the infusion of large volumes of crystalloids. Instead, synthetic colloids or plasma infusions may be considered to maintain osmotic pressure (Self, 2016), which was carefully considered in our dogs, where the majority of dogs additionally received Voluven. The slow elimination of certain drugs may contribute to the prolonged recovery phase, potentially leading to hypothermia and hypoglycemia (Self, 2016; Dugdale et al., 2020), so maintaining body temperature is crucial, and warming measures were implemented from the premedication phase through full recovery.

Cellophane banding emerges as a straightforward surgical procedure for the progressive attenuation of PSS in dogs (Youmans and Hunt, 1998). This method involves applying a cellophane band around the shunt to reduce blood flow. While the benefits of surgically attenuating shunts are widely acknowledged, rapid closure or ligation of the shunt may not yield optimal results for many dogs, as it can lead to the development of portal hypertension and cardiovascular compromise. Because the occlusion of the PSS with cellophane progresses slowly through inflammatory reaction, the shunt closes gradually, while the portal vein can uptake larger volumes of blood (Holt, 1994; Youmans and Hunt, 1998; Frankel et al., 2006). Since this process occurs concurrently, no portal hypertension occurred. Using inexpensive and readily available cellophane allows progressive attenuation in the weeks following cellophane placement. Slow attenuation of CPS may also allow more time for the cardiovascular and central nervous systems to adapt to changing hepatic metabolism (Youmans and Hunt, 1998).

The incorporation of preoperative imaging can reduce both surgical duration and the extent of dissection required for evaluating the shunt (Or et al., 2016). Attenuation location for different shunt types vary; portocaval shunts are attenuated at the level of epiploic

foramen, portophrenic at the abdominal surface of the diaphragm and newer studies suggest attenuation of portoazygos shunts within the thorax (Mullins, 2019b), while older literature suggested attenuation at the diaphragm level (Or et al., 2016), where azygos shunts were also attenuated in our study. The following considerations guided us during the surgeries: A healthy canine circulatory system should exhibit no large vessels entering the caudal vena cava between the phrenicoabdominal vein and the porta hepatis (Berent and Tobias, 2018). Moreover, it is common to observe turbulent blood flow in the caudal vena cava where the shunting vessel enters (Mullins, 2019a).

After the surgery, laboratory parameters such as ALT, urea, TP, albumines, and bile acids improved in most dogs, alongside clinical signs. Subsequently, medical therapy was discontinued. Based on clinical signs, lactulose treatment should be sustained for a minimum of 4 weeks, coupled with a protein-restricted diet until liver function normalises, indicated by normal albumin levels. Serum bile acids may remain abnormal for over a year following shunt ligation (Tobias, 2012). However, if abnormalities persist nine months post-surgery, the likelihood of normalisation decreases. In such cases, additional laboratory values, including albumin, urea, and liver enzymes, should be utilised to monitor the patient's condition. If blood values fail to normalise six months post-surgery, a reevaluation is advised to explore potential issues such as incomplete shunt occlusion, multiple acquired shunts, or other liver diseases (Tobias, 2012). However, the primary goal of surgery is for the animal to be free of clinical signs and not require therapy. It is important to note that while laboratory findings often improve after surgery, they may still frequently remain abnormal, as observed in our study.

5. Conclusion

The absence of significant complications observed during the surgical procedure can be attributed to administering appropriate therapy to the dogs for a sufficient duration before the operation. This therapeutic approach was consistently maintained throughout the recovery period. Intensive patient monitoring was crucial in the initial 12-24 hours post-surgery. Furthermore, a thorough observation was kept for any neurological signs, ensuring prompt initiation of assistance if such signs manifested. No signs of portal hypertension were detected in any of the cases post-surgery.

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